South Afr J Anaesth Analg. 2022;28(5 Suppl 1):S104-109
https://doi.org/10.36303/SAJAA.2022.28.5.2872

South Afr J Anaesth Analg
ISSN 2220-1181 EISSN 2220-1173

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Update on traumatic brain injury

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Traumatic brain injury (TBI) is an alteration in brain function, or other evidence of brain pathology, caused by an external force. Clinically, TBI is described by severity as per the well-recognised Glasgow Coma Scale (GCS). Traditionally, according to the GCS, a mild TBI is defined by a GCS of 13–15; a moderate TBI by a GCS of 9–12, and a severe TBI by a GCS of less than 8. **Primary** TBI occurs at the time of the traumatic incident. Regardless of the cause, external mechanical forces transfer energy to intracranial contents, resulting in the pathological pattern of injury. **Secondary** TBI occurs at a molecular level, is initiated at the time of initial trauma and can lead to nerve cell death and cerebral oedema, which culminate in an exacerbation of the initial injury. Secondary TBI presents a host of factors which the anaesthesiologist can mitigate to potentially improve patient outcome, but at the very least, to prevent deterioration. These are aimed at managing mean arterial pressure, ventilation and therefore $PaCO_2$, glucose, temperature, intracranial pressure, seizures and coagulation. The Brain Trauma Foundation publish evidence-based guidelines on this subject that are freely accessible in order to standardise the treatment of this condition. The most recent publication is the fourth edition from 2016. The outcomes from the RESCUEicp and DECRA trials affect the decompressive craniectomy recommendations, and have been added after this, as part of the 'living guidelines' initiative.

Keywords: traumatic brain injury, anaesthesia, TBI, neuroprotection

Traumatic brain injury (TBI) is defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force".[1] An external force, as per this definition, is due to one of six entities:[1]

1. The head being struck by an object
2. The head striking an object
3. Acceleration/deceleration of the brain without direct external impact
4. A foreign body penetrating the brain
5. The force from a blast or explosion
6. Other forces yet to be defined

The global incidence of TBI, as per the Global Burden of Disease study in 2016 was estimated at 27.98 million cases per year,[1] with falls and road traffic-related insults being the most common and second most common causes, respectively.[1]

Clinically, TBI is classified by severity as per the well-recognised Glasgow Coma Scale (GCS). The GCS is a composite scale with three main components: best verbal response (5), best motor response (6) and eye opening (4). The score ranges from a minimum of 3, to a maximum of 15 (or 10 in an intubated patient, where the best verbal component is removed). The components of the GCS score are described in Table I.

Traditionally, according to the GCS, a mild TBI is defined by a GCS of 13–15; a moderate TBI by a GCS of 9–12, and a severe TBI by a GCS of less than 8.[1] More recently, there has been a proposed adaptation of this classification as up to one-third of patients with a GCS of 13 have an intracranial haemorrhage present.[3] As a result, a GCS of 14–15 should be reclassified as a mild TBI, 9–13 a moderate TBI, and a GCS < 9 as a severe TBI.[3]

Table I: The Glasgow Coma Scale

	Score
Eye opening	
Spontaneous	4
Response to verbal commands	3
Response to pain	2
No eye opening	1
Best verbal response	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best motor response	
Obeys commands	6
Localising response to pain	5
Withdrawal response to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1
Total	/15

The main benefits of using the GCS lie in its simplicity, reproducibility and prognostic capability.[1] However, the score does

not account for intoxicating substances, the presence of an endotracheal tube, or medical sedation and paralysis.[1]

A perhaps lesser-known clinical scoring system, the Full Outline of UnResponsiveness (FOUR) score, has been developed to address these shortcomings of the GCS, but it is arguably more complicated and lacks the prognostication benefits of the GCS.[1]

TBI may also be classified according to the Marshall and Rotterdam neuroimaging scales, both of which are computed tomography (CT) based grading scales. The Marshall scale uses six categories to predict the risk of raised intracranial pressure (ICP) and, accordingly, outcome.[1] Its main pitfall lies in its lack of reproducibility in patients who possess multiple types of brain injury, and the Rotterdam scale was developed to address this, but still requires validation.[1]

There are several defined clinical insults that can be visualised on neuroimaging:

1. Skull fractures.
2. Cerebral contusions.
3. Focal and diffuse axonal injury with cerebral oedema.[1]
4. Epidural haematomata (EDH): torn vessels in the dura mater (typically middle meningeal artery) and are associated with skull fractures in the majority of cases. EDH are convex/lenticular (lemon) shaped and don't usually present with underlying brain damage, and thus may have a better prognosis.[1]
5. Subdural haematomata (SDH): due to torn bridging veins, are concave/crescent (banana) shaped and usually **do** present with underlying brain injury. They are associated with relatively low trauma in the ageing population due to cerebral atrophy contributing to stretching of bridging veins.[1]
6. Subarachnoid haemorrhage (SAH): due to disruption of small vessels in the pia mater.[1]
7. Intraparenchymal haemorrhage.
8. Intraventricular haemorrhage: due to tearing of subependymal veins, or extension from intraparenchymal or SAH.[1]

TBI can be defined as primary or secondary in nature.

Primary TBI occurs at the time of the traumatic incident. Regardless of the cause, external mechanical forces transfer energy to intracranial contents, resulting in the pathological pattern of injury. Patients with a severe TBI may develop a coagulopathy due to systemic release of tissue factor and brain phospholipids into circulation, causing intravascular coagulation and a consumptive coagulopathy, that may result in the expansion of an already present bleed in the brain, and, consequently, a poorer prognosis.[1]

Secondary TBI occurs at a molecular level, initiated at the time of initial trauma, and can lead to nerve cell death and cerebral oedema, which culminate in an exacerbation of the initial injury. Some of these mechanisms include electrolyte abnormalities, mitochondrial dysfunction, inflammation, programmed cell death, neurotransmitter-mediated excitotoxicity, vasospasm-induced ischaemia, focal microvascular occlusion and vascular injury.[1]

The key for the anaesthetist is to mitigate the factors that lead to secondary TBI and thereby limit or halt the progression of the initial injury. This includes optimising mean arterial pressure (MAP), oxygenation, managing the ICP and cerebral perfusion pressure (CPP), temperature, seizure activity and glucose.

The Brain Trauma Foundation leads the way in conducting clinical and field research about TBI.[2] They publish evidence-based guidelines that are freely accessible. The most recent of these guidelines was published in 2016. The core idea is to ensure that the treatment of this potentially debilitating condition should be more homogenous in order to improve prognosis. However, there is a lack of randomised control trials to validate each recommendation.[3] The most important point to note is that patients should be treated in centres that can provide neurosurgical and neuro-intensive care facilities that follow established protocols to ensure improved outcomes.[3]

New evidence from The Trial of Decompressive Craniectomy for Traumatic Intracranial Hypertension (RESCUEicp) and The Decompressive Craniectomy in Diffuse Traumatic Brain Injury (DECRA) studies have been incorporated into the guidelines in 2020.[4]

The DECRA trial, an international, multicentre trial, saw 155 patients with a severe, diffuse (non-penetrating) TBI with refractory, raised ICP > 20 mmHg for 15 minutes in an hour despite intervention, being randomised within 72 hours of injury, to receive early bifrontotemporoparietal decompressive craniectomy (in addition to medical therapy). Medical therapy included optimising sedation and P_aCO_2, use of mannitol/hypertonic saline, neuromuscular blockade, external ventricular drainage of cerebrospinal fluid, induced hypothermia of 35 °C and barbiturates.[5] ICP was reduced, as was the duration of intensive care unit stay, but prognosis at six months was not improved, as per the Glasgow Outcome Scale (GOS-E).[5]

The Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of ICP trial (RESCUEicp) is another international multicentre randomised control trial, where 408 patients 10–65 years of age, with TBI and refractory intracranial hypertension of > 25 mmHg for one to 12 hours (despite medical management) underwent decompressive craniectomy. Medical therapy in this trial included elevation of the head, sedation, analgesia, ventilation, and the option of muscle relaxation. Also included were ventriculostomy, inotropic support, use of hypertonic saline, mannitol, loop diuretics and temperature management.[6]

Mortality was lower in the surgical group than in the medical management group at six months.[6] However, the main caveat was that the survivors of the intervention had greater dependency requirements (vegetative state and disability); measured by the

Table II: Brain Trauma Foundation Guidelines summary[4]

Topic	Recommendation
Decompressive craniectomy	**Level IIA**
	• *Bifrontal DC is not recommended to improve outcomes as measured by the GOS-E score at 6 months post-injury in severe CI patients with diffuse injury (without mass lesions), and with ICP elevation to values > 20 mmHg for more than 15 minutes within a 1-hour period that are refractory to first-tier therapies. However, this procedure has been demonstrated to reduce IP and minimise days in the ICU.*
	• *A large frontotemporoparietal DC (not less than 12 x 15 cm or 15 CM diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurological outcomes in patients with severe TBI.*
	• The committee is aware that the results of the RESCUREicp trial[2] were released soon after the completion of these Guidelines. The results of this trial may affect these recommendations and may need to be considered by treating physicians and other users of these Guidelines. We intend to update these recommendations if needed. Updates will be available at https://braintrauma.org/coma/guidelines.
Prophylactic hypothermia	**Level IIB**
	• *Early (within 2.5 hours), short-term (48 hours post-injury), prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse injury.*
Hyperosmolar therapy	Recommendations from the prior (Third) edition are not supported by evidence meeting current standards.
	Mannitol is effective for control of raised ICP at doses of 0.25 to 1 g/kg body weight. Arterial hypotension (systolic blood pressure < 90 mmHg) should be avoided.
	Restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurological deterioration not attributable to extracranial causes.
Cerebrospinal fluid drainage	**Level III**
	• *An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use.*
	• *Use of CSF drainage to lower ICP in patients with an initial GCS < 6 during the first 12 hours after injury may be considered.*
Ventilation therapies	**Level IIB**
	• Prolonged prophylactic hyperventilation with $PaCO_2$ of ≤ 25 mmHg is not recommended.
	Recommendations from the prior (Third) edition are not supported by evidence meeting current standards.
	Hyperventilation is recommended as a temporising measure for the reduction of elevated ICP.
	Hyperventilation should be avoided during the first 24 hours after injury when CBF often is reduced critically.
	If hyperventilation is used, SjO_2 or $BtpO_2$ measurements are recommended to monitor oxygen delivery.
Anaesthetics, analgesics and sedatives	**Level IIB**
	• Administration of barbiturates to induce burst suppression measured by EEG as prophylaxis against the development of intracranial hypertension is not recommended.
	• High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Haemodynamic stability is essential before and during barbiturate therapy.
	• Although propofol is recommended for the control of ICP, it is not recommended for improvement in mortality or 6-month outcomes. Caution is required as high-dose propofol can produce significant morbidity.[3]
Steroids	**Level I**
	• The use of steroids is not recommended for improving outcome or reducing ICP. In patients with severe TBI, high-dose methylprednisolone was associated with increased mortality and is contraindicated.
Nutrition	**Level IIA**
	• *Feeding patients to attain basal caloric replacement at least by the fifth day and at most by the seventh day post-injury is recommended to decrease mortality.*
	Level IIB
	• *Transgastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.*
Infection prophylaxis	**Level IIA**
	• Early tracheotomy is recommended to reduce mechanical ventilation days when the overall benefit is thought to outweigh the complications associated with such a procedure. However, there is no evidence that early tracheostomy reduces mortality or the rate of nosocomial pneumonia.
	• *The use of PI oral care is not recommended to reduce ventilator-associated pneumonia and may cause an increased risk of acute respiratory distress syndrome.*
	Level III
	• *Antimicrobial-impregnated catheters may be considered to prevent catheter-related infections during external ventricular drainage.*

Deep vein thrombosis prophylaxis	**Level III**
	• LMWH or low-dose unfractioned heparin may be used in combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial haemorrhage. • In addition to compression stockings, pharmacological prophylaxis may be considered if the brain injury is stable and the benefit is considered to outweigh the risk of increased intracranial haemorrhage. • There is insufficient evidence to support recommendations regarding the preferred agent, dose or timing of pharmacological prophylaxis for deep vein thrombosis.
Seizure prophylaxis	**Level IIA**
	• Prophylactic use of phenytoin or valproate is not recommended for preventing late PTS. • Phenytoin is recommended to decrease the incidence of early PTS (within 7 days of injury), when the overall benefit is thought to outweigh the complications associated with such treatment. However, early PTS have not been associated with worse outcomes. • *At present, there is sufficient evidence to recommend levetiracetam compared with phenytoin regarding efficacy in preventing early post-traumatic seizures and toxicity.*

$BtpO_2$ – brain tissue O_2 partial pressure, CBF – cerebral blood flow, CSF – cerebrospinal fluid drainage, DC – decompressive craniectomy, EEG – electroencephalogram, EVD – external ventricular drainage, GCS – Glasgow Coma Scale, GOS-E – Glowgow Outcomes Scale–Extended, ICP – intracranial pressure, ICU – intensive care unit, LMWH – low molecular weight heparin, $PaCO_2$ – partial pressure of arterial carbon dioxide, PI – povidone-iodine, PTS – post-traumatic seizures, RESCUEicp trial – Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of ICP trial, SjO_2 – jugular venous oxygen saturation, TBI – traumatic brain injury
Italic: New or revised recommendations

GOS-E. Surgical intervention-related survival may not result in improved quality of life.[6]

The intensive care management of TBI patients is pertinent to the anaesthetist as it is an extension of their perioperative management. As discussed earlier, mitigating secondary brain injury is the key goal. The sections of the executive summary of the Brain Trauma Foundation guidelines, which are pertinent to the anaesthetist, are reproduced in Table II.[4]

There are 10 main areas of interest to the anaesthetist when managing TBI in a critical care environment.

1. The most important variable that needs to be controlled is the **ICP**. This can be done using a three-pronged approach. *Physical* methods include elevating the head to 30° to allow for passive venous drainage from the brain without compromising cerebral perfusion, maintaining the neck in a neutral position, and not employing the use of tight neckties. *Physiological* management of raised ICP includes managing glucose, temperature, MAP and ventilation (by employing neuromuscular blockade, analgesia and sedation to prevent patient-ventilator dyssynchrony). *Pharmacological* management involves the use of agents such as hypertonic saline, mannitol and loop diuretics.[3]

Regarding the monitoring of ICP in TBI, an external ventricular drain (EVD) connected to a strain gauge is the most accurate and cost-effective intermittent monitor, and has the benefit of being diagnostic and therapeutic (allowing for cerebrospinal fluid drainage). Intraparenchymal monitors are simpler to site and have less risk of bleeding and infection, and offer the advantage of continuous monitoring.[3]

Table III: Updated monitoring recommendations[4]

Topic	Recommendations
Intracranial pressure monitoring	**Level IIB**
	• *Management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality.* Recommendations from the prior (Third) edition are not supported by evidence meeting current standards. ICP should be monitored in all salvageable patients with a TBI (GCS 3–8 after resuscitation) and an abnormal CT scan. An abnormal CT scan of the head is one that reveals haematomas, contusions, swelling, herniation or compressed basal cisterns. ICP monitoring is indicated in patients with severe TBI with a normal CT scan if ≥ 2 of the following features are noted at admission: age > 40 years, unilateral or bilateral motor posturing, or SBP < 90 mmHg.
Cerebral perfusion pressure monitoring	**Level IIB**
	• *Management of severe TBI patients using guidelines-based recommendations for CPP monitoring is recommended to decrease 2-week mortality.*
Advanced cerebral monitoring	**Level IIB**
	• Jugular bulb monitoring of $AVDO_2$, as a source of information for management decisions, may be considered to reduce mortality and improve outcomes at 3 and 6 months post-injury.

$AVDO_2$ – arteriovenous oxygen content difference, CPP – cerebral perfusion pressure, CT – computed tomography, GCS – Glasgow Coma Scale, ICP – intracranial pressure, SBP – systolic blood pressure, TBI – traumatic brain injury
Italic: New or revised recommendations

Table IV: Updated recommendations: thresholds[4]

Topic	Recommendations
Blood pressure thresholds	**Level III**
	Maintaining SBP at ≥ 100 mmHg for patients 50–69 years old or at ≥ 110 mmHg or above for patients 15–49 or > 70 years old may be considered to decrease mortality and improve outcomes.
Intracranial pressure thresholds	**Level IIB**
	Treating ICP > 22 mmHg is recommended because values above this level are associated with increased mortality.
	Level III
	A combination of ICP values and clinical and brain CT findings may be used to make management decisions.
	The committee is aware that the results of the RESCUEicp trial2 were released after the completion of these Guidelines. The results of this trial may affect these recommendations and may need to be considered by treating physicians and other users of these Guidelines. We intend to update these recommendations if needed. Updates will be available at https://braintrauma.org/coma/guidelines.
Cerebral perfusion pressure thresholds	**Level IIB**
	The recommended target CPP value for survival and favourable outcomes is between 60 and 70 mmHg. Whether 60 or 70 mmHg is the minimum optimal CPP threshold is unclear and may depend upon the autoregulatory status of the patient.
	Level III
	Avoiding aggressive attempts to maintain CPP > 70 mmHg with fluids and pressors may be considered because of the risk of adult respiratory failure.
Advanced cerebral monitoring thresholds	**Level III**
	Jugular venous saturation of < 50% may be a threshold to avoid in order to reduce mortality and improve outcomes.

CPP – cerebral perfusion pressure, CT – computed tomography, ICP – intracranial pressure, RESCUEicp trial – Randomised Evaluation of Surgery with Craniectomy for Uncontrolled Elevation of ICP, SBP – systolic blood pressure
Italic: New or revised recommendations

If there are signs of impending cerebral herniation, such as asymmetric pupil size, abnormal posturing, or Cushing's triad of hypertension, bradycardia and irregular respiration, then patients should be intubated and hyperventilated to a $PaCO_2$ of 25–30 mmHg. A bolus of mannitol or hypertonic saline can be employed.[3]

The longstanding debate of whether mannitol or hypertonic saline is preferred still has no consensus. Rather, the risks and benefits of each should be evaluated for the individual patient. Both agents create an osmolar gradient, and draw water across the blood–brain barrier (BBB) to reduce cerebral blood volume and ICP. They both have clinical effects that diminish with time, as compensatory osmoles in the brain are formed in 24 hours. They must both be weaned slowly to prevent reversal of the osmotic gradient and rebound cerebral oedema.[3]

Mannitol is given at a dose of 0.25–1 g/kg four to six hourly. It can result in hypovolaemia, and a higher MAP must be maintained, due to the raised ICP. This is accomplished using fluid resuscitation and vasopressor/inotropic support. Mannitol can leak through the damaged BBB, reverse the osmolar gradient and cause rebound cerebral oedema. Serum osmolality and renal function testing is vitally important when mannitol is used. [3]

Hypertonic saline is administered via central venous line as a 3% infusion or 23.4% bolus (30 ml over 10 minutes) for the treatment of acute elevations in ICP. This is titrated to serum sodium level. Volume depletion and hypovolaemia are less likely to occur, and it may be preferentially used in the setting of blood loss, hypovolaemia or hypotension. The reflection coefficient of hypertonic saline is 1.0 compared to mannitol, where it is 0.9, and the former is less likely to leak into brain tissue as a result. The main risks with its usage are circulatory overload, pulmonary oedema and normal anion gap metabolic acidosis due to the chloride load. Electrolytes and fluid balance must be meticulously managed as well.[3]

In patients with refractory intracranial hypertension, decompressive craniectomy, barbiturate coma and induced hypothermia are the modalities used.[3]

2. **Neurological** monitoring is vital. Clinical deterioration is associated with an expanding haematoma or increasing cerebral oedema, detected by serial neurological examinations that should be performed one to two hourly for the first 24 to 48 hours, and guides the need for repeat CT scanning. Patients with a haemorrhage-related severe TBI should have a repeat CT scan in six hours.[3]
3. Managing the **haemodynamics** of TBI patients is crucial. Isotonic saline is the fluid of choice to maintain euvolaemia. Hypotension should be avoided. Cerebral autoregulation is impaired in one-third of TBI patients, a phenomenon called "pressure passive". An elevated MAP can cause a raised ICP on the basis of an increased cerebral blood volume, while a

reduction in the MAP can cause hypoperfusion and cerebral ischaemia. CPP is defined by the difference between the MAP and ICP. The CPP goal is 60–70 mmHg, achieved by treating raised ICP, rather than elevating the MAP, as elevating the blood pressure increases cerebral blood flow (CBF), and can worsen cerebral oedema in the setting of impaired cerebral autoregulation.[3]

4. Artificial **ventilation**, on the basis of the patient being sedated and/or paralysed, allows for control of the $PaCO_2$. Acute hypercarbia can result in an elevated ICP, while hypocarbia may lead to cerebral ischaemia, both on the basis of altered CBF. Consequently, it is vital that end-tidal carbon dioxide concentrations are measured. Hyperventilation can reduce ICP, but should be avoided in the first 24–48 hours, when the brain is prone to cerebral vasoconstriction and therefore ischaemia; and a $PaCO_2 < 30$ mmHg should only be employed as a temporising manoeuvre in acute elevations in ICP for this reason. Hyperventilation can also raise lactate and glutamate levels, both of which can worsen cerebral injury. As a result, $PaCO_2$ should be maintained between 30–35 mmHg. Hypoxia should be avoided, and PaO_2 maintained above > 60 mmHg. Elevated intrathoracic pressures due to raised positive end-expiratory pressure (PEEP) may impede venous return from the brain and worsen ICP. PEEP improves oxygenation in patients with concomitant ARDS, where it should therefore be employed.[3]
5. To prevent and treat post-traumatic **seizures** (PTSs) in the setting of TBI, levetiracetam is used. Early PTSs can occur in a third of patients with severe TBI. Non-convulsive seizures are also a risk, and are only diagnosed on electroencephalography (EEG). The use of antiepileptics reduces PTSs but does not prevent the development of epilepsy. Seizures worsen TBI outcomes due to an increase in CBF, ICP and metabolic demand on the injured brain.[3]
6. **Venous thromboembolism (VTE)** is a real risk in patients with TBI. Intermittent pneumatic compression stockings should be used on every TBI patient from admission, and heparin should be started in all TBI patients 24 hours after admission, except if there is evidence of an expanding haemorrhage on repeat CT scan. The risk of haemorrhage expansion is always weighed against the risk of VTE.[3]
7. Patients with a moderate to severe TBI should have their **coagulation** status assessed on admission. Coagulopathy is common, and leads to a poorer prognosis. Warfarin should be reversed with prothrombin complex concentrate and vitamin K, and an INR of < 1.4 should be aimed for. In patients with thrombocytopenia, platelets should be kept above 75×10^9/L.[3]
8. **Glucose** should be maintained within normal range, 7.8–10 mmol/L. Abnormal glucose levels can lead to tissue acidosis on the basis of anaerobic metabolism, free radical damage and increased porosity of the BBB.[3]
9. **Hyperthermia** must be aggressively treated due to the risk of worsening an already raised ICP due to raised metabolic demand, cerebral blood volume and CBF. This is accomplished by employing the use of antipyretics and surface cooling. Hypothermia may result in shivering, which increases the cerebral metabolic demand and worsens brain tissue oxygenation. When cooling is necessary, it must only be done in a controlled (induced) manner (therapeutic hypothermia).[3]
10. **Nutritional** goals appropriate for the patient must be achieved within five to seven days from injury. Transpyloric enteral feeding reduces the risk of ventilator-associated pneumonia.[3]

In conclusion, our understanding of TBI is evolving. TBI is a broad spectrum of pathology that is potentially debilitating. It is vital that, as anaesthetists, we are equipped with up-to-date knowledge about mitigating the factors that can reduce the extent of the functional impact of the primary injury, so that patients may reap the benefits thereof in the long term.

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