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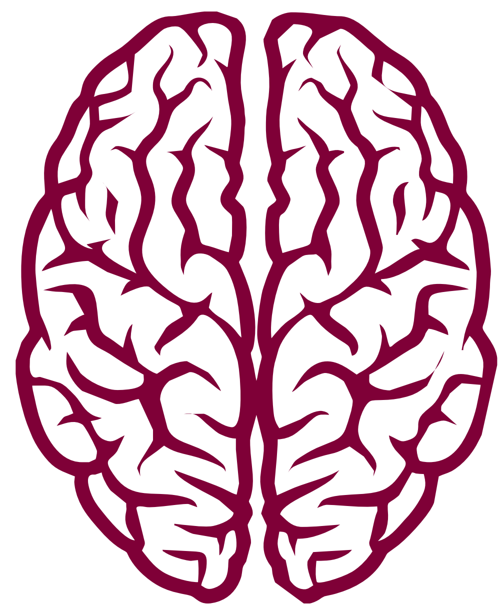
STANDARD TREATMENT GUIDELINES

NEURO SURGERY



DEPARTMENT OF HEALTH AND FAMILY WELFARE

GOVERNMENT OF KERALA

KERALA.HEALTH

STANDARD TREATMENT GUIDELINES
for
NEURO SURGERY

Foreword

At the outset, I appreciate the work done by the respective thematic teams and coordination done by the DME. The Standard Treatment Guidelines (STG) were prepared and published in 2021 in the thick of the Covid pandemic. On the last page of these volumes the road map was mentioned. The few points are mentioned here for the recall.

"The Department of Health has been taking a systematic approach of creating and enabling multiple initiatives with a focus on prevention along with improving health care services. Health care service delivery is one of the most important services and is always seen as a barometer to assess the Governance. While it is important to develop infrastructure, an essential prerequisite is to develop systems and processes to bring in standardization in management of patient care. ……………The foundation is laid and we take up the challenge to work on the unfinished agenda."

It was mentioned in the road map to have institutional mechanism to ensure updation of Standard Treatment Guidelines. The next step that was suggested was to do analysis of Karunya Arogya Suraksha Padhati (KASP) and standard treatment guidelines to work on developing a Balance Score Card to give information regarding compliance from the Hospitals and to build a "feedback loop" to improve. These initiatives remained at concept level on the last page! But following detailed discussions with Dr Vishwanathan, Director Medical Education, some of the foundational things were prioritized and given an impetus to take it to finality. In this journey, many committed doctors from various Medical Colleges of respective specialties participated. The previous coordination team members and experts were also consulted and they also participated in discussions and these Standard Treatment Guidelines are prepared.

The standard treatment guidelines will be made available in the Kerala Health portal (health.kerala.gov.in). This will enable the resource book availability not only to people within the state but to all in the country and outside our borders as well. I am confident that it will be used by students and practicing doctors. We request inputs based on the research from the Specialists and Experts. The teams shall continue to update and make any required changes in the STG by doing periodic updates.

The most important thing we all need to internalize is to have a shared vision and work as a team to reach to a state of 'excellence'. If we take a look at the preparation of the Directorate Medical Education Management Information System, documents of each Medical Colleges, it provides information regarding 'what we are, what we do and what we aspire to do', pandemic preparedness, AMR accreditation and many more such initiatives taken on scale, which are all outcomes of collective TEAM work. This has laid a foundation for involving all the stakeholders including undergraduate and postgraduate students. This should encourage the teams in Medical Colleges to believe in themselves and build future initiatives on such a sound platform.

I express my sincere thanks to Dr Vishwanathan for his patience and bearing with relentless follow ups! I also take this opportunity to thank each and every team and their members and everyone from Directorate Medical Education and Medical Colleges who supported these initiatives.

I would like to express my sincere gratitude to all those who have contributed to publish these Standard Treatment Guidelines.

I wish all the success to DME team to make Kerala MCH as a premier knowledge hub in Medical Science.

Dr Rajan Khobragade IAS

Additional Chief Secretary
Health & Family Welfare and
AYUSH Department
Govt of Kerala.

Message

Patient care today demands evidence-based, standardized, and contextually relevant clinical practice. In this regard, the publication of the **Second Edition of the Standard Treatment Guidelines** marks an important step forward in strengthening the quality, consistency, and accountability of healthcare delivery in Kerala.

The first edition laid a strong foundation for uniform clinical practice across specialties and super specialties. Since then, advances in medical knowledge, evolving treatment modalities, and the growing need for periodic updating have made it essential to revisit and refine these guidelines. The present edition reflects this commitment to continuous improvement and clinical excellence.

I am pleased to note that subject experts from various disciplines of Government Medical Colleges, private institutions and professional bodies have contributed as resource persons in the preparation of these guidelines. Their academic expertise, practical insight, and dedicated involvement have greatly enriched this edition. I deeply appreciate the sincere efforts of all the conveners, contributors, and coordinators whose collective commitment and teamwork made this publication possible.

These guidelines will serve as a valuable reference for clinicians, teachers, trainees, and healthcare institutions, helping to promote evidence-based decision-making and improve patient outcomes. I am confident that this edition will further support standardization of care and contribute to the advancement of medical education and clinical practice in the State.

I congratulate everyone involved in this commendable effort and commend this publication to all healthcare professionals.

Dr. K. V. Viswanathan
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Chapter 1 – Introduction & Background

Traumatic injuries involving the brain and spinal cord represent one of the most important and yet under-addressed public health challenges in India. Traumatic brain injury (TBI) and spinal cord injury (SCI) are associated with high mortality, prolonged hospitalisation, long-term disability, and profound socioeconomic consequences for patients, families, and society. A substantial proportion of those affected are young adults in their most productive years, resulting in loss of workforce, long-term dependency, and increased burden on healthcare systems.

India is among the countries with the highest incidence of road traffic accidents globally. Rapid urbanisation, exponential growth in vehicular density, mixed traffic patterns, inadequate road safety infrastructure, and inconsistent enforcement of safety regulations have all contributed to this burden. Kerala, while ranking high in the absolute number of reported road traffic accidents, demonstrates a comparatively lower severity and fatality rate, reflecting better access to emergency medical services and tertiary care facilities. Nevertheless, preventable deaths and disabilities due to neurotrauma remain unacceptably high, underscoring the need for standardised, system-based approaches to care.

Traumatic brain injury should not be viewed as a single static event. Instead, it represents a dynamic and evolving disease process. The initial mechanical insult produces primary brain injury, which occurs at the moment of impact and is largely irreversible. This includes contusions, lacerations, diffuse axonal injury, and vascular disruption. In contrast, secondary brain injury develops over minutes to days following the primary insult and is mediated by a complex cascade of pathophysiological processes including hypoxia, hypotension, raised intracranial pressure (ICP), cerebral ischemia, excitotoxicity, metabolic failure, inflammation, and infection. Importantly, secondary injury is potentially preventable and forms the principal target of modern neurotrauma management.

Over the past several decades, substantial improvements in outcomes after severe TBI have been achieved in high-income countries. These gains have not resulted from the discovery of new neuroprotective drugs, but rather from the implementation of organised trauma systems, early airway protection, strict control of oxygenation and blood pressure, rapid access to neuroimaging, timely neurosurgical intervention, protocol-driven intensive care, and early rehabilitation. The consistent application of evidence-based guidelines has been shown to reduce mortality, shorten hospital stay, and improve functional outcomes.

In India, however, the absence of a uniform, nationwide trauma care system has resulted in significant variability in outcomes. Differences in prehospital care, referral pathways, availability of trained personnel, critical care resources, and neurosurgical expertise lead to wide disparities in survival and disability. Many aspects of neurotrauma care continue to rely heavily on individual experience rather than standardised protocols, particularly in resource-limited settings.

The present Standard Treatment Guidelines (STG – 2025) have been developed to address these gaps. The objective is not to simply replicate Western guidelines, but to provide India-centric, pragmatic recommendations that can be implemented across different levels of healthcare delivery, from district hospitals to tertiary referral centres. These guidelines emphasise achievable standards of care, prioritisation of interventions with the greatest impact on outcome, and rational use of available resources.

The scope of this document extends across the entire continuum of neurotrauma care. It addresses prevention and epidemiology, initial assessment and classification, use of clinical scoring systems, management of concussion and diffuse brain injury, evidence-based treatment of severe traumatic brain injury in adults and children, management of specific structural injuries such as depressed skull fractures, and evaluation and treatment of spinal and spinal cord injuries. Particular emphasis is placed on prevention of secondary injury, protocol-driven decision-making, and early initiation of rehabilitation.

These guidelines are intended for use by emergency physicians, anaesthesiologists, neurosurgeons, intensivists, orthopaedic surgeons, residents, and nursing staff involved in trauma care. They also serve as a reference framework for training, clinical audit, and quality improvement initiatives. The document is designed as a living guideline, to be updated periodically as new evidence emerges and trauma care infrastructure continues to evolve.

Chapter 2 – Epidemiology of Traumatic Brain Injury

Traumatic brain injury constitutes one of the most significant contributors to injury related mortality and disability in India. The epidemiology of TBI reflects the broader patterns of trauma in the country and is closely linked to rapid socioeconomic change, urbanisation, increasing motorisation, occupational hazards, and evolving demographic trends. Understanding these epidemiological characteristics is essential for planning preventive strategies, allocating healthcare resources, and designing effective trauma systems.

Public Health Importance

Injuries have emerged as a major public health problem in India, rivaling communicable and non-communicable diseases as a cause of death and long-term disability. Among injuries, TBI is associated with the highest rates of mortality and permanent neurological impairment. Survivors frequently experience cognitive, behavioural, and physical deficits that interfere with education, employment, and social reintegration. The cumulative impact of these disabilities places a substantial burden on families, caregivers, and the healthcare system.

Magnitude of the Problem

Available estimates suggest that India records nearly one million injury-related deaths annually. Of these, approximately 50–60% involve injury to the brain, translating to an estimated 500,000 TBI-related deaths each year. For every death due to TBI, it is estimated that 30–50 individuals require hospitalisation, and many more sustain injuries that do not reach hospital care. This results in an estimated 15 million moderate to severe TBIs annually, with an even larger number of mild TBIs going unreported.

It must be emphasised that these figures likely underestimate the true burden of disease. India lacks a comprehensive, population-based trauma registry, and available data are derived from heterogeneous sources including police records, hospital admission data, and regional epidemiological studies. Variability in case definitions, reporting practices, and data capture further limits the accuracy of national estimates.

Demographic Characteristics

TBI predominantly affects young adult males, particularly those between 15 and 45 years of age. This demographic group represents the most economically productive segment of the population, amplifying the socioeconomic consequences of injury. A male predominance is consistently observed across studies, reflecting greater exposure to high-risk activities such as driving, riding two-wheelers, and certain occupations.

In recent years, a rising incidence of TBI among the elderly has been noted. This trend is largely attributable to an increased frequency of falls, age-related balance impairment, visual deficits, and widespread use of antiplatelet and anticoagulant medications. TBIs in the elderly are often associated with higher rates of intracranial hemorrhage, prolonged hospital stay, and poorer outcomes.

Alcohol consumption is a major contributory factor in traumatic brain injury, particularly in road traffic accidents and assaults. Alcohol intoxication not only increases the risk of injury but also complicates initial neurological assessment and is associated with more severe injury patterns.

External Causes of Injury

Road traffic injuries constitute the leading cause of TBI in India, accounting for approximately 60% of cases. Mixed traffic conditions, inadequate separation of vulnerable road users, poor road infrastructure, and inconsistent use of protective devices such as helmets and seat belts contribute to this high burden.

Falls are the second most common cause, responsible for approximately 25% of TBIs. These include falls from height in occupational settings, domestic falls, and falls among the elderly. Assaults and interpersonal violence account for roughly 10% of cases, with higher prevalence in certain urban and socioeconomically disadvantaged populations.

Pedestrians, two-wheeler riders, and bicyclists represent particularly vulnerable groups, with a disproportionately high risk of severe head injury. Without effective preventive strategies, road traffic injuries are projected to remain among the leading causes of death in India in the coming decades.

Patterns and Severity of Injury

The spectrum of TBI includes concussion, cerebral contusions, skull fractures, and intracranial hemorrhages such as epidural and subdural hematomas. Based on clinical severity, approximately 60% of TBIs are classified as mild, 20% as moderate, and 15–20% as severe. Polytrauma is present in nearly one-quarter of cases, further complicating management and adversely affecting outcomes.

Disability and Rehabilitation Needs

The burden of disability following TBI is substantial. Almost all survivors of severe TBI, approximately half of those with moderate TBI, and a significant minority of patients with mild TBI require some form of rehabilitation. Cognitive and behavioural impairments often persist even in patients who recover good motor function. It is estimated that more than five million individuals in India currently require rehabilitation services related to TBI.

Access to organised rehabilitation services remains limited, particularly in rural and resource-constrained settings. Early initiation of rehabilitation and communitybased support are critical determinants of long-term functional outcome.

Future Perspective

Traumatic brain injury is largely predictable and preventable. Strengthening road safety legislation and enforcement, improving helmet and seat belt use, reducing alcohol-impaired driving, and enhancing workplace safety are essential preventive strategies. In parallel, development of organised trauma systems, improvement in prehospital care, establishment of trauma registries, and investment in rehabilitation services are necessary to reduce the burden of death and disability from TBI in India.

Chapter 3 – Classification of Head Injury

Classification of head injury provides a structured framework for assessment, communication, clinical decision-making, and prognostication. Given the heterogeneity of traumatic brain injury, no single classification system is sufficient in isolation. A comprehensive approach incorporating clinical severity, mechanism of injury, anatomical pattern, and temporal evolution is therefore essential.

Severity-Based Classification

The most widely accepted method of classifying head injury severity is the Glasgow Coma Scale (GCS), which evaluates eye opening, verbal response, and motor response. Based on the initial GCS score, head injury is classified as:

- Mild head injury: GCS 13–15
- Moderate head injury:
- GCS 9–12 Severe head injury: GCS ≤8

This classification has important practical implications. Patients with mild head injury constitute the majority of presentations but require careful assessment to identify those at risk of intracranial pathology. Moderate head injury carries a significant risk of deterioration and frequently necessitates hospital admission and close monitoring. Severe head injury is a neurological emergency and mandates aggressive resuscitation, early neuroimaging, and intensive care.

It is important to recognise that the initial GCS score may be influenced by confounding factors such as alcohol intoxication, sedative medications, endotracheal intubation, facial injuries, and systemic shock. Serial neurological examinations and emphasis on the motor response are therefore recommended.

Mechanism-Based Classification

Head injuries may be broadly categorised based on the mechanism of injury into:

- Closed head injury, in which the skull and dura remain intact
- Penetrating head injury, in which there is breach of the skull and dura by an external object or bone fragment

Penetrating head injuries are associated with a higher incidence of intracranial infection, vascular injury, cerebrospinal fluid leak, and post-traumatic epilepsy. Management strategies differ substantially from those for closed head injuries, and early neurosurgical involvement is essential.

Anatomical and Pathological Classification

From an anatomical perspective, head injuries may be divided into focal and diffuse injuries.

Focal injuries include epidural hematoma, subdural hematoma, intracerebral hemorrhage, and cerebral contusions. These lesions are often visible on computed tomography and may be amenable to surgical intervention.

Diffuse injuries include diffuse axonal injury and diffuse cerebral edema. These injuries result from acceleration–deceleration and rotational forces and are typically associated with impaired consciousness disproportionate to imaging findings. Management is primarily medical and focuses on prevention of secondary injury.

Temporal Classification

Head injuries may also be classified according to the timing of clinical deterioration:

- Immediate deterioration, occurring at the time of injury
- Early deterioration, occurring within the first 24 hours

Delayed deterioration, occurring days to weeks after injury

This temporal framework underscores the importance of observation, repeat neurological assessment, and repeat imaging in selected patients, particularly those with moderate head injury or evolving symptoms.

Clinical Relevance of Classification

Classification of head injury directly influences clinical management. Severity classification guides decisions regarding airway protection, need for neuroimaging, level of monitoring, and ICU admission. Anatomical classification determines the need for surgical intervention, while temporal considerations inform observation protocols and follow-up imaging.

Importantly, classification systems should not be used in isolation to predict outcome or determine withdrawal of care. Prognostication in head injury requires integration of clinical examination, imaging findings, physiological parameters, and response to treatment over time.

Chapter 4 – Clinical Scoring Systems in Head Injury

Clinical scoring systems play a central role in the assessment, monitoring, communication, and prognostication of patients with traumatic brain injury. They provide a common language for healthcare providers, allow stratification of injury severity, guide triage and management decisions, and facilitate comparison of outcomes across institutions. However, no scoring system should be interpreted in isolation; scores must always be integrated with clinical judgment, imaging findings, and physiological parameters.

Glasgow Coma Scale (GCS)

The Glasgow Coma Scale remains the cornerstone of neurological assessment in head injury. It evaluates three components of consciousness: eye opening (E), verbal response (V), and motor response (M), with a total score ranging from 3 to 15.

The GCS is used extensively to:

- Classify head injury severity into mild, moderate, and severe categories
- Guide decisions regarding airway protection and need for intubation
- Determine urgency of neuroimaging and neurosurgical consultation
- Monitor neurological trends through serial examinations

Among the three components, the motor response is the most reliable predictor of outcome and should be emphasized, particularly when verbal assessment is confounded by intubation or facial injury.

Despite its widespread use, the GCS has important limitations. Alcohol intoxication, sedative medications, hypoxia, hypotension, and metabolic disturbances can significantly alter the score. In addition, intubation precludes verbal assessment, reducing the utility of the total score. For these reasons, serial assessments and documentation of individual components are strongly recommended.

Four Score (Full Outline of UnResponsiveness)

The FOUR score was developed to address some of the limitations of the GCS, particularly in critically ill and intubated patients. It assesses four domains: eye response, motor response, brainstem reflexes, and respiratory pattern.

Key advantages of the FOUR score include:

- Applicability in intubated patients
- Inclusion of pupillary and corneal reflexes
- Ability to detect locked-in syndrome and brainstem dysfunction
- Improved prognostic value in deeply comatose patients

The FOUR score is particularly useful in intensive care settings and complements the GCS rather than replacing it. Use of both scales provides a more comprehensive neurological assessment.

Pupillary Assessment and Brainstem Reflexes

Independent of formal scoring systems, pupillary size, symmetry, and reactivity are critical components of neurological evaluation. Pupillary abnormalities may indicate raised intracranial pressure, transtentorial herniation, or direct brainstem injury. Serial pupillary examination is essential for early detection of neurological deterioration.

Computed Tomography–Based Scoring Systems

Radiological scoring systems provide objective measures of injury severity based on computed tomography findings. They are particularly useful for risk stratification and prognostication in moderate and severe TBI.

The Marshall CT classification categorizes head injury based on the status of basal cisterns, presence of midline shift, and mass lesions. It provides a simple framework for describing diffuse injury patterns and identifying surgically significant lesions.

The Rotterdam CT score refines prognostication by incorporating additional variables such as traumatic subarachnoid hemorrhage and intraventricular hemorrhage. Higher Rotterdam scores are associated with increased mortality and poorer functional outcomes.

Radiological scores should support, but not replace, clinical decision-making. Early withdrawal of care should never be based solely on CT scoring systems.

Prognostic Models and Outcome Prediction

Several multivariable prognostic models, such as the IMPACT and CRASH models, combine clinical, radiological, and laboratory parameters to estimate outcome after TBI. While these tools may aid in counseling families and stratifying patients for research, they have limited utility in guiding individual bedside decisions during the acute phase.

Outcome prediction in TBI is inherently uncertain, particularly in the early period after injury. Prognostic discussions should therefore be cautious, iterative, and based on trends over time rather than single-point assessments.

Clinical Application and Limitations

Scoring systems are indispensable tools in neurotrauma care, but their limitations must be clearly understood. Over-reliance on numerical scores can lead to inappropriate clinical decisions, particularly in the context of confounding factors or evolving injury. The optimal approach involves repeated assessment, correlation with imaging and physiological data, and integration of scores into a broader clinical framework.

Chapter 5 – Concussion: Classification & Management

Definition and Scope

Concussion is a form of mild traumatic brain injury (mTBI) characterized by a transient disturbance of brain function induced by biomechanical forces. It is primarily a functional injury rather than a structural one, and routine neuroimaging is frequently normal. Concussion may occur with or without loss of consciousness and can result from direct impact to the head, face, neck, or elsewhere on the body with an impulsive force transmitted to the brain.

Concussion represents a significant proportion of TBI presentations to emergency departments and outpatient clinics. Although traditionally considered benign, it is now well recognized that concussion can result in prolonged symptoms, functional impairment, and, in rare cases, catastrophic outcomes if mismanaged.

Pathophysiology

The pathophysiology of concussion involves complex neurometabolic processes rather than macroscopic structural damage. Rapid acceleration–deceleration and rotational forces lead to neuronal membrane disruption, ionic shifts (particularly potassium efflux and calcium influx), release of excitatory neurotransmitters, and increased energy demand in the setting of reduced cerebral blood flow. This mismatch between energy supply and demand results in a period of cerebral vulnerability.

During this phase, the brain is particularly susceptible to secondary insults, and repeat injury may result in disproportionate neurological deterioration. This underlines the importance of early recognition and appropriate activity modification following concussion.

Clinical Presentation

The clinical manifestations of concussion are heterogeneous and may involve one or more of the following domains:

- Somatic symptoms: headache, dizziness, nausea, photophobia,
- phonophobia Cognitive symptoms: slowed thinking, impaired concentration, memory difficulties
- Emotional and behavioral symptoms: irritability, anxiety, mood changes
- Sleep disturbances: insomnia, hypersomnia, altered sleep–wake cycle

Symptoms may evolve over hours to days following injury. Loss of consciousness and post-traumatic amnesia are not mandatory for diagnosis and their absence does not exclude concussion.

Subtype-Based Classification

Recent evidence supports a subtype-based approach to concussion, particularly within the first 72 hours after injury. Five core clinical subtypes have been described:

1. Cognitive subtype – characterized by impaired attention, slowed processing speed, and memory difficulties.
2. Vestibular subtype – dizziness, imbalance, motion sensitivity, and nausea.
3. Ocular-motor subtype – visual blurring, difficulty with reading, eye strain, abnormal saccades or convergence.
4. Headache/migraine subtype – throbbing headache, photophobia, phonophobia, nausea, often migraine-like.
5. Anxiety/mood subtype – anxiety, emotional lability, low mood, and heightened stress response.

Two commonly associated conditions include sleep disturbance and cervical spine strain. These subtypes are not mutually exclusive, and a single patient may exhibit features of multiple subtypes, with dominance changing over time.

Pediatric versus Adult Concussion

Pediatric patients often present with prominent vestibular and balance-related symptoms, whereas adults more commonly demonstrate cognitive impairment and headache-dominant presentations. Children may have difficulty articulating symptoms, and behavioral changes or school performance decline may be the earliest indicators.

Recovery trajectories also differ, with children and adolescents sometimes experiencing prolonged symptoms, particularly when return to school and sports is not appropriately graded.

Assessment and Diagnosis

Diagnosis of concussion is clinical and is based on a thorough history and neurological examination. Assessment should include:

- Mechanism of injury
- Presence and duration of loss of consciousness or amnesia
- Symptom profile across multiple domains
- Cognitive assessment
- Vestibulo-ocular evaluation
- Cervical spine examination

- Screening for mood and sleep disturbances

Neuroimaging is not routinely required in uncomplicated concussion and should be reserved for patients with red flags such as deteriorating consciousness, focal neurological deficits, repeated vomiting, or suspicion of structural injury.

Management Principles

The primary goals of concussion management are symptom resolution, prevention of secondary injury, and safe return to normal activities. Key principles include:

- Early education and reassurance
- Brief period of relative physical and cognitive rest (24–48 hours)
- Avoidance of prolonged strict rest
- Gradual, stepwise return to activity based on symptom tolerance

Symptom-Specific Management

Management should be individualized according to the predominant symptom subtype:

- Cognitive symptoms: graded cognitive activity, temporary academic or work accommodations
- Vestibular and ocular-motor symptoms: early referral for vestibular and vision therapy
- Headache/migraine: appropriate analgesia, avoidance of medication overuse, migraine-directed therapy when indicated
- Anxiety and mood symptoms: reassurance, structured routine, psychological support when needed
- Sleep disturbance: sleep hygiene counseling, avoidance of sedatives when possible

Return-to-Activity and Return-to-Work

A graded return-to-activity protocol is recommended, with progression through stages only if the patient remains symptom-free at the current level. Premature return to full activity increases the risk of prolonged symptoms and repeat injury.

Complications and Special Considerations

Post-concussion syndrome refers to persistence of symptoms beyond the expected recovery period. Risk factors include prior concussion, severe initial symptom burden, anxiety, depression, and poor sleep.

Second impact syndrome, though rare, is a catastrophic condition resulting from repeat head injury before recovery from the initial concussion and underscores the importance of strict adherence to return-to-activity guidelines.

Clinical Summary

Concussion is a common but potentially complex clinical entity. Early recognition, subtype-based assessment, patient education, and structured return-to-activity protocols are central to optimal outcomes. Misconceptions regarding prolonged rest and underestimation of symptom burden should be actively addressed to prevent long-term morbidity.

Chapter 6 – Diffuse Axonal Injury

Diffuse axonal injury (DAI) is a major pathological substrate of traumatic brain injury and represents one of the most important causes of prolonged unconsciousness and poor neurological outcome following trauma. Unlike focal mass lesions, DAI is a diffuse injury involving widespread disruption of axons within the white matter tracts of the brain. Recognition of DAI is essential, as clinical severity is often disproportionate to findings on initial neuroimaging.

Mechanism of Injury

DAI results primarily from rotational acceleration–deceleration forces rather than direct impact. Rapid angular movement of the head leads to shearing stresses at the interface of tissues with different densities, particularly at the grey–white matter junction, corpus callosum, and brainstem. These forces cause stretching, disruption, and eventual disconnection of axons.

At a cellular level, axonal injury is initiated by mechanical deformation of the axonal cytoskeleton, followed by dysregulation of ion channels, calcium influx, mitochondrial dysfunction, and activation of proteolytic enzymes. Axonal swelling and secondary disconnection may evolve over hours to days, contributing to delayed neurological deterioration.

Pathological Classification

Diffuse axonal injury is classically classified into three grades based on anatomical involvement:

- Grade I – Widespread axonal injury predominantly at the grey–white matter junction
- Grade II – Grade I findings with additional focal lesions in the corpus callosum
- Grade III – Grade I and II findings with additional involvement of the dorsolateral brainstem

Increasing grade is associated with greater severity of injury, prolonged coma, and poorer functional outcome.

Clinical Features

Patients with DAI typically present with impaired consciousness immediately following injury. Loss of consciousness is often prolonged and may persist despite the absence of a large mass lesion on initial CT imaging. Neurological examination may reveal abnormal posturing, pupillary abnormalities, or impaired brainstem reflexes in more severe cases.

DAI should be suspected in patients with:

- Disproportionately low GCS relative to CT findings
- Persistent coma without mass lesion
- High-velocity mechanisms such as road traffic accidents or falls from height

Neuroimaging

Computed tomography may be normal or show only subtle findings such as small hemorrhagic foci or diffuse cerebral edema. CT is useful for excluding surgically treatable mass lesions but is relatively insensitive for detecting axonal injury.

Magnetic resonance imaging is more sensitive for the diagnosis of DAI. Susceptibility-weighted imaging (SWI) can detect microhemorrhages, while diffusion-weighted and diffusion tensor imaging can demonstrate axonal disruption. MRI findings correlate better with clinical severity and prognosis but are not routinely required in the acute unstable patient.

Management Principles

There is no specific surgical or pharmacological treatment for diffuse axonal injury. Management is therefore supportive and focused on prevention of secondary brain injury. Key principles include:

- Early airway protection and avoidance of hypoxia
- Strict prevention of hypotension
- ICP monitoring and management in severe cases
- Maintenance of adequate cerebral perfusion pressure
- Normocapnia and normothermia
- Early enteral nutrition and general intensive care support

Sedation and analgesia are used to control agitation and reduce metabolic demand. Seizure prophylaxis is administered according to standard severe TBI protocols.

Prognosis

Prognosis in DAI is variable and closely related to injury grade, duration of coma, and extent of brainstem involvement. Patients with Grade I injury may recover with minimal deficits, whereas Grade III injury is associated with high mortality and severe long-term disability.

Early prognostication is challenging and should be approached with caution. Outcome assessment should be based on serial neurological examinations, imaging trends, and response to supportive care over time rather than early imaging findings alone.

Rehabilitation Considerations

Survivors of DAI often experience significant cognitive, behavioural, and executive dysfunction even in the absence of major motor deficits. Early initiation of multidisciplinary rehabilitation, including neuropsychological assessment, cognitive therapy, and structured behavioural interventions, is critical for optimal long-term recovery.

Chapter 7 – Management of Severe Traumatic Brain Injury (Adults)

Severe traumatic brain injury (TBI), defined as a Glasgow Coma Scale (GCS) score of 8 or less after initial resuscitation, constitutes a neurological emergency requiring immediate, structured, and protocol-driven management. Mortality and long-term disability in severe TBI are largely determined not only by the primary injury but by the effectiveness with which secondary brain injury is prevented. The cornerstone of management is therefore meticulous physiological control, timely neurosurgical intervention, and coordinated intensive care.

Goals of Management

The primary objectives in the management of severe TBI are:

- Prevention of secondary brain injury due to hypoxia, hypotension, raised intracranial pressure, and metabolic derangements
- Maintenance of adequate cerebral perfusion and oxygen delivery
- Early identification and treatment of surgically remediable lesions
- Optimisation of systemic physiology and prevention of complications

Initial Assessment and Resuscitation

Management begins at the point of first medical contact and follows standard trauma principles with strict cervical spine protection. The priorities are airway protection, adequate ventilation, and circulatory stabilisation.

Patients with GCS ≤8 should undergo early endotracheal intubation to secure the airway and prevent hypoxia and aspiration. Preoxygenation and avoidance of hypotension during induction are essential, as even a single episode of hypoxia or hypotension significantly worsens outcome. Rapid sequence induction with agents that minimise hemodynamic instability is recommended.

Ventilation and Oxygenation

Adequate oxygenation is critical in preventing secondary brain injury. Arterial oxygen saturation should be maintained at ≥90%, with a target PaO_2 >60 mm Hg. Routine prophylactic hyperventilation should be avoided, as reduction in $PaCO_2$ leads to cerebral vasoconstriction and may precipitate cerebral ischemia.

Normocapnia ($PaCO_2$ 35–40 mm Hg) should be maintained during routine care. Brief, controlled hyperventilation may be used only as a temporising measure in the setting of acute neurological deterioration or impending herniation while definitive therapy is instituted.

Hemodynamic Management

Systemic hypotension is a major independent predictor of mortality in severe TBI. Systolic blood pressure should be maintained at or above 100 mm Hg in patients aged 50–69 years and at or above 110 mm Hg in patients aged 15–49 years or over 70 years. Isotonic crystalloids are preferred for volume resuscitation, and hypotonic solutions should be avoided as they may exacerbate cerebral edema.

Vasopressors may be required to maintain adequate blood pressure once euvolemia is achieved. The choice of agent should be guided by the patient’s cardiovascular status and institutional protocols.

Intracranial Pressure and Cerebral Perfusion Pressure Monitoring

Intracranial pressure (ICP) monitoring is recommended in salvageable patients with severe TBI and abnormal CT findings. It may also be considered in selected patients with severe TBI and normal CT scans who have additional risk factors such as age over 40 years, hypotension, or abnormal motor posturing.

Treatment should be initiated when ICP exceeds 22 mm Hg. Cerebral perfusion pressure (CPP), calculated as mean arterial pressure minus ICP, should be maintained between 60 and 70 mm Hg. CPP values below 50 mm Hg are associated with cerebral ischemia, whereas excessively high CPP may increase the risk of acute lung injury.

General Measures for ICP Control

First-tier measures for ICP control include head elevation to 30 degrees, maintenance of neutral neck alignment, adequate sedation and analgesia, avoidance of fever, and correction of hypoxia and hypotension. Frequent neurological assessment and close monitoring of physiological parameters are essential.

Hyperosmolar Therapy

Hyperosmolar agents are effective in reducing ICP by creating an osmotic gradient across the blood–brain barrier. Both mannitol and hypertonic saline are acceptable options, and current evidence does not clearly favour one agent over the other.

Mannitol is typically administered as intermittent boluses and requires monitoring of serum osmolality, renal function, and volume status. Hypertonic saline may be administered as bolus doses or continuous infusion, with careful monitoring of serum sodium and osmolarity. Choice of agent should be individualised based on hemodynamic status and institutional experience.

Sedation, Analgesia, and Neuromuscular Blockade

Adequate sedation and analgesia reduce cerebral metabolic demand, facilitate ventilator synchrony, and assist in ICP control. Agents such as propofol and shortacting opioids are commonly used. Neuromuscular blockade may be considered in patients with refractory intracranial hypertension to prevent coughing, agitation, and ventilator dyssynchrony.

Seizure Prophylaxis

Early post-traumatic seizures increase metabolic demand and may worsen secondary brain injury. Antiepileptic prophylaxis is recommended for the first seven days following severe TBI to prevent early seizures. There is no evidence to support routine continuation of prophylaxis beyond this period in the absence of seizures.

Temperature and Metabolic Management

Fever should be aggressively treated, as hyperthermia exacerbates neuronal injury. Prophylactic hypothermia is not recommended due to lack of benefit and increased risk of complications. Glycemic control should aim to avoid both hypoglycemia and severe hyperglycemia; overly tight glucose control is discouraged.

Nutrition and Supportive Care

Early enteral nutrition, initiated within 24–48 hours of injury, is associated with reduced mortality and infectious complications. Additional supportive measures include stress ulcer prophylaxis, deep vein thrombosis prophylaxis when safe, pressure injury prevention, and meticulous nursing care.

Surgical Management

Surgical intervention is indicated for evacuation of mass lesions such as epidural and subdural hematomas, large contusions with mass effect, and for decompressive craniectomy in selected patients with refractory intracranial hypertension. Decompressive craniectomy has been shown to reduce ICP and improve survival, though functional outcomes depend on patient selection and timing.

Prognostication and Ongoing Care

Early prognostication in severe TBI is inherently uncertain and should not rely on a single clinical or radiological parameter. Decisions regarding limitation or withdrawal of care should be deferred until the effects of sedation, metabolic derangements, and acute injury have been adequately assessed over time. Multimodal assessment and repeated evaluation are essential.

Clinical Summary

Effective management of severe traumatic brain injury requires early airway protection, strict avoidance of hypoxia and hypotension, ICP- and CPP-guided therapy, judicious use of hyperosmolar agents and surgery, and comprehensive intensive care. Adherence to protocol-driven management significantly improves survival and functional outcome.

Chapter 8 – Step-by-Step ED to ICY Management Algorithm for Severe Traumatic Brain Injury

The management of severe traumatic brain injury requires a structured, stepwise approach that begins in the emergency department and continues seamlessly into the intensive care unit. This algorithmic approach ensures early stabilisation, timely identification of life-threatening conditions, prevention of secondary brain injury, and appropriate escalation of care. While individual patient factors and institutional resources may vary, adherence to a systematic pathway improves consistency and outcomes.

Step 1: Primary Survey and Immediate Stabilisation (ABCDE)

Management begins with a standard trauma primary survey, with strict attention to cervical spine protection throughout.

Airway (A): Patients with a GCS score of 8 or less should undergo early endotracheal intubation to secure the airway and prevent hypoxia and aspiration. Manual in-line cervical stabilisation should be maintained during airway management. Preoxygenation is essential, and induction agents should be chosen to minimise hypotension.

Breathing (B): Adequate ventilation and oxygenation must be ensured. Target oxygen saturation should be ≥90%, with PaO_2 maintained above 60 mm Hg. Routine hyperventilation should be avoided. Ventilator settings should aim for normocapnia.

Circulation (C): Systemic hypotension must be aggressively prevented. Hemorrhage control, rapid assessment of volume status, and isotonic fluid resuscitation are priorities. Age-appropriate systolic blood pressure targets should be maintained. Vasopressors may be initiated if hypotension persists despite adequate volume resuscitation.

Disability (D): A focused neurological assessment including Glasgow Coma Scale score, pupillary size and reactivity, and gross motor response should be documented. Baseline neurological findings are critical for subsequent comparison.

Exposure (E): The patient should be fully exposed to identify associated injuries while simultaneously preventing hypothermia using active warming measures.

Step 2: Early Neuroimaging and Baseline Investigations

Once the patient is haemodynamically stabilised, urgent non-contrast computed tomography of the head should be performed. CT imaging allows identification of surgically remediable lesions such as epidural hematoma, subdural hematoma, and contusions with mass effect.

Repeat CT imaging is indicated in the presence of neurological deterioration, persistently elevated intracranial pressure, or following neurosurgical intervention. Routine repeat imaging in clinically stable patients should be individualised.

Baseline laboratory investigations including arterial blood gases, electrolytes, glucose, complete blood count, and coagulation profile should be obtained and corrected as necessary.

Step 3: Indications for Intracranial Pressure Monitoring

Intracranial pressure monitoring should be instituted in salvageable patients with severe TBI and abnormal CT scans. It may also be considered in patients with severe TBI and normal CT findings who have additional risk factors such as age greater than 40 years, hypotension, or abnormal motor posturing.

ICP monitoring provides objective data to guide therapy and allows timely escalation when treatment thresholds are exceeded.

Step 4: Definition of Physiological Targets

Once in the ICU, management should be directed toward clearly defined physiological goals:

- ICP less than 22 mm Hg
- CPP between 60 and 70 mm Hg
- $PaCO_2$ 35–40 mm Hg
- Oxygen saturation ≥90%
- Normothermia
- Avoidance of hypoglycemia and severe hyperglycemia

These targets form the foundation of protocol-driven intensive care management.

Step 5: First-Tier ICP Management

If intracranial pressure exceeds treatment thresholds, first-tier interventions should be instituted promptly. These include head elevation to 30 degrees, neutral neck alignment, adequate sedation and analgesia, avoidance of fever, correction of hypoxia and hypotension, and initiation of hyperosmolar therapy with mannitol or hypertonic saline.

Ventilation should be adjusted to maintain normocapnia. Short-term hyperventilation may be used only as a temporising measure in acute neurological deterioration.

Step 6: Second-Tier and Escalation Therapies

In patients with refractory intracranial hypertension despite first-tier measures, escalation of therapy is required. Options include deeper sedation, neuromuscular blockade, cerebrospinal fluid drainage when an external ventricular drain is present, and barbiturate coma in selected, haemodynamically stable patients.

Decompressive craniectomy should be considered in patients with refractory intracranial hypertension who do not respond to maximal medical therapy. Decision-making should be individualised and involve multidisciplinary discussion.

Step 7: Supportive ICU Care and Complication Prevention

Comprehensive supportive care is essential and includes early enteral nutrition, deep vein thrombosis prophylaxis when safe, stress ulcer prophylaxis, glycemic control, pressure injury prevention, and infection surveillance. Nursing care and regular repositioning play a crucial role in preventing secondary complications.

Step 8: Ongoing Assessment, Prognostication, and Rehabilitation Planning

Neurological assessment and physiological parameters should be reviewed frequently, and management adjusted accordingly. Early withdrawal of care should be avoided. Prognostication should be based on serial clinical examinations, imaging trends, and overall physiological stability.

Once the patient is stabilised, early planning for rehabilitation and long-term care should be initiated to optimise functional recovery.

Chapter 9 – Pediatric Severe Traumatic Brain Injury

Severe traumatic brain injury in children represents a distinct clinical entity with important anatomical, physiological, and developmental differences compared with adults. These differences influence injury patterns, response to secondary insults, monitoring thresholds, and overall outcomes. Pediatric severe TBI is defined as a Glasgow Coma Scale score of less than 9 in infants, children, and adolescents up to 18 years of age and requires specialised, protocol-driven management.

Unique Pediatric Considerations

Children have proportionally larger heads, weaker neck musculature, and thinner cranial bones, predisposing them to diffuse injury patterns. Cerebral metabolic demand is higher in children, while autoregulatory mechanisms may be less robust, rendering the pediatric brain particularly vulnerable to hypoxia and hypotension. In addition, age-related variations in normal vital parameters complicate assessment and resuscitation.

Neurological assessment in young children may be challenging due to limited verbal communication, fear, or irritability. Modified pediatric Glasgow Coma Scale scoring should therefore be used in infants and preverbal children.

Goals of Management

The primary goals in pediatric severe TBI mirror those in adults but must be adapted to age-specific physiology:

- Prevention of secondary brain injury
- Control of intracranial pressure
- Maintenance of adequate cerebral perfusion
- Prevention of early post-traumatic seizures
- Optimisation of long-term neurodevelopmental outcome

Monitoring and Physiological Targets

Intracranial pressure monitoring is suggested in children with severe TBI, although high-quality evidence defining precise thresholds is limited. In clinical practice, ICP values greater than 20 mm Hg are commonly treated.

Cerebral perfusion pressure targets are age-dependent. Current evidence supports maintaining a minimum CPP of 40 mm Hg, with lower values acceptable in infants and higher targets (45–50 mm Hg) preferred in older children and adolescents. CPP measurement should take into account the level at which arterial pressure transducers are zeroed and head positioning.

Airway, Ventilation, and Oxygenation

Early airway protection is essential in pediatric severe TBI to prevent hypoxia and aspiration. Oxygen saturation should be maintained above 90%, and normocapnia should be targeted. Routine prophylactic hyperventilation is not recommended. Brief hyperventilation may be used only as a temporising measure in acute neurological deterioration.

Hemodynamic Management

Hypotension is poorly tolerated in children with TBI and is strongly associated with adverse outcome. Age-appropriate blood pressure targets should be maintained using isotonic fluids and vasoactive agents when required. Careful attention to volume status is essential, as both hypovolemia and fluid overload may be harmful.

Intracranial Pressure Management

First-tier ICP management includes head elevation, neutral neck alignment, adequate sedation and analgesia, avoidance of fever, and hyperosmolar therapy. Hypertonic saline is preferred over mannitol in many pediatric centers due to more predictable hemodynamic effects. Continuous infusions or intermittent boluses may be used, with careful monitoring of serum sodium.

Ventricular cerebrospinal fluid drainage is effective for ICP control when an external ventricular drain is available. Neuromuscular blockade may be considered in refractory cases.

Seizure Prophylaxis

Early post-traumatic seizures are more common in children than adults. Antiepileptic prophylaxis during the first seven days following injury is suggested to reduce the incidence of early seizures, although no clear benefit has been demonstrated for long-term neurological outcome.

Temperature and Metabolic Control

Fever should be aggressively treated, as hyperthermia exacerbates secondary brain injury. Prophylactic hypothermia is not recommended due to lack of proven benefit and increased risk of complications. Glucose levels should be maintained within a safe range, avoiding both hypoglycemia and severe hyperglycemia.

Surgical Management

Indications for surgical intervention in pediatric severe TBI include evacuation of mass lesions, decompressive craniectomy for refractory intracranial hypertension, and cerebrospinal fluid diversion when indicated. Evidence is insufficient to define optimal timing or patient selection for decompressive craniectomy, and decisions should be individualised.

Prognosis and Long-Term Outcomes

Outcome after pediatric severe TBI is influenced by injury severity, secondary insults, and access to specialised care. Children may demonstrate greater neuroplasticity than adults, but severe injuries are associated with long-term cognitive, behavioural, and educational difficulties.

Rehabilitation and Follow-Up

Early initiation of multidisciplinary rehabilitation is essential. Long-term follow-up should address cognitive development, school reintegration, behavioural issues, and family support. Pediatric TBI is a chronic condition with evolving needs, and ongoing assessment is crucial.

Chapter 10 – Depressed Skull Fractures

Depressed skull fractures are the result of high-energy blunt impact to the cranium, leading to inward displacement of one or more bone fragments. These injuries are clinically significant because they are frequently associated with underlying dural tears, intracranial hematomas, cerebral contusions, cerebrospinal fluid leakage, and infection. Management decisions must be individualised and are based on the depth of depression, integrity of the dura, degree of contamination, associated brain injury, and neurological status of the patient.

Classification

Depressed skull fractures may be classified as:

- Closed (simple) depressed fractures, in which the overlying scalp remains intact and there is no communication with the external environment.
- Open (compound) depressed fractures, where there is an associated scalp laceration and communication between the fracture site and the external environment.

Compound fractures carry a substantially higher risk of infection, including meningitis and intracranial abscess, and generally require surgical intervention.

Pathophysiology and Associated Injuries

The inward displacement of bone fragments can directly compress the underlying cortex, resulting in focal neurological deficits or seizures. Dural breach allows communication between intracranial and extracranial compartments, predisposing to cerebrospinal fluid leakage and infection. Depressed fractures are commonly associated with epidural or subdural hematomas and cortical contusions, particularly when located over venous sinuses or eloquent cortex.

Clinical Evaluation

Patients may present with scalp lacerations, palpable skull depression, focal neurological deficits, seizures, or signs of raised intracranial pressure. A thorough neurological examination is essential. Particular attention should be paid to the presence of cerebrospinal fluid leak, contamination of the wound, and signs of local or systemic infection.

Computed tomography of the head with bone windows is the imaging modality of choice. CT accurately delineates the depth of depression, number of fragments, associated intracranial injuries, and involvement of venous sinuses.

Indications for Surgical Management

Surgical elevation and repair are recommended in the presence of one or more of the following:

- Depression greater than the thickness of the adjacent skull, typically more than 5 mm
- Dural penetration or cerebrospinal fluid leak
- Underlying intracranial hematoma or contusion requiring evacuation
- Gross contamination of the wound
- Focal neurological deficit attributable to bone fragment compression
- Significant cosmetic deformity, particularly in adults

Early neurosurgical consultation is essential in all suspected depressed skull fractures.

Conservative Management

Selected patients with closed depressed fractures may be managed conservatively. Criteria include minimal depression, intact dura, absence of neurological deficit, no underlying intracranial hematoma, and absence of wound contamination. Conservative management involves head elevation, analgesia, close neurological observation, and serial imaging when indicated.

Surgical Technique

The goals of surgery are to elevate depressed bone fragments, remove devitalised tissue, achieve watertight dural closure, evacuate associated hematomas, and restore the contour of the skull.

The procedure typically involves a scalp incision incorporating any laceration, meticulous debridement of contaminated tissue, elevation or removal of depressed fragments, repair of dural tears using primary closure or duraplasty, and reconstruction of the skull using plates, screws, or bone substitutes when required. Care must be taken when fractures overlie venous sinuses.

Antibiotic Therapy and Seizure Prophylaxis

Broad-spectrum antibiotics are recommended for all open depressed skull fractures to reduce the risk of intracranial infection. The duration of therapy should be guided by the degree of contamination and intraoperative findings. Antiepileptic prophylaxis may be considered, particularly in patients with cortical involvement or early post-traumatic seizures.

Pediatric Considerations

In infants and young children, depressed skull fractures may present as "ping-pong" fractures due to the pliability of the skull. Surgical intervention is reserved for fractures associated with dural tear, neurological deficit, significant depression, or cosmetic concerns. Selected cases may remodel spontaneously.

Outcomes and Follow-Up

With appropriate management, outcomes following depressed skull fractures are generally favourable. Long-term follow-up should focus on detection of posttraumatic epilepsy, infection, cosmetic deformity, and neurocognitive sequelae.

Chapter 11 – Thoracolumbar Spine Trauma

Thoracolumbar spine injuries constitute a major proportion of spinal trauma encountered in clinical practice and are frequently associated with high-energy mechanisms such as road traffic accidents, falls from height, and industrial injuries. These injuries may occur in isolation or as part of polytrauma and carry a significant risk of spinal instability, neurological deficit, chronic pain, and long-term disability. A structured, evidence-informed approach to evaluation and management is essential to optimise neurological and functional outcomes.

Objectives of Management

The goals of management in thoracolumbar spine trauma are:

- Accurate identification and classification of spinal injury
- Early detection of neurological deficit
- Prevention of secondary spinal cord injury
- Restoration and maintenance of spinal stability
- Early mobilisation and rehabilitation where appropriate

Initial Assessment and Stabilisation

All patients with suspected thoracolumbar spine injury should be managed according to trauma principles with strict spinal immobilisation until injury is excluded. A focused neurological examination must be performed, documenting motor power, sensory level, reflexes, sacral sensation, and sphincter tone. Associated injuries are common and should be actively sought.

Hemodynamic stability is critical, particularly in patients with concomitant spinal cord injury. Hypotension should be corrected promptly, as reduced spinal cord perfusion may exacerbate neurological damage.

Classification Systems

Use of validated classification systems improves communication and guides management decisions. The two most commonly used systems are:

Thoracolumbar Injury Classification and Severity Score (TLICS): This system incorporates injury morphology, integrity of the posterior ligamentous complex (PLC), and neurological status. Higher scores generally favour operative management, while lower scores support nonoperative treatment.

AO Spine Thoracolumbar Classification: This system categorises injuries based on morphology (compression, distraction, translation), neurological status, and patientspecific modifiers. It provides a comprehensive framework for describing injury patterns.

While these systems aid decision-making, no single classification reliably predicts outcome in all cases, and clinical judgment remains paramount.

Imaging Evaluation

Computed tomography is the primary imaging modality for evaluation of thoracolumbar spine trauma and allows detailed assessment of bony injury. Magnetic resonance imaging plays a crucial role in evaluating the integrity of the posterior ligamentous complex, spinal cord, intervertebral discs, and soft tissues.

MRI is particularly valuable when surgical intervention is being considered, as it may alter management in a significant proportion of cases by identifying occult ligamentous injury or spinal cord compression.

Neurological Assessment and Prognostic Factors

Neurological status at presentation is the strongest predictor of outcome. Assessment should be performed using validated tools, and particular attention should be paid to sacral sensation, voluntary anal contraction, and motor function below the level of injury. Serial examinations are essential to detect early deterioration.

Nonoperative Management

Nonoperative treatment may be appropriate in neurologically intact patients with stable fracture patterns. Evidence suggests that in selected thoracolumbar burst fractures without neurological deficit, outcomes with bracing and without bracing are equivalent. Decisions regarding bracing should therefore be individualised based on patient comfort, compliance, and institutional practice.

Close clinical and radiological follow-up is required to detect progressive deformity or delayed neurological compromise.

Operative Management

Surgical intervention is indicated in patients with spinal instability, progressive neurological deficit, significant deformity, or failure of conservative treatment. The goals of surgery are decompression of neural elements, restoration of spinal alignment, and stabilisation.

Posterior, anterior, and combined approaches may be used depending on injury pattern, neurological status, and surgeon expertise. Current evidence does not demonstrate clear superiority of one approach over another in terms of neurological outcome.

Timing of Surgery

The optimal timing of surgery remains debated. While early surgery has not consistently been shown to improve neurological recovery, it may reduce hospital length of stay, facilitate early mobilisation, and decrease complications. When feasible, surgery within the first 24–72 hours may be considered, particularly in polytrauma patients.

Special Surgical Considerations

Minimally invasive fixation techniques have gained popularity and have been shown to achieve outcomes comparable to open surgery, with potential advantages of reduced blood loss and soft tissue disruption. Routine addition of fusion in thoracolumbar burst fractures has not been shown to improve outcomes and may increase operative morbidity.

Thromboprophylaxis and Supportive Care

Patients with thoracolumbar spine trauma are at increased risk of venous thromboembolism. Thromboprophylaxis should be instituted once bleeding risk is controlled, using mechanical and pharmacologic measures as appropriate.

Rehabilitation and Long-Term Care

Early mobilisation and rehabilitation are critical components of care. Multidisciplinary rehabilitation should address pain control, mobility, bladder and bowel function, and psychosocial support. Long-term follow-up is essential to monitor spinal alignment, neurological status, and functional recovery.

Clinical Summary

Management of thoracolumbar spine trauma requires accurate classification, careful neurological assessment, judicious use of imaging, and individualised decision-making regarding operative versus nonoperative care. Early stabilisation and rehabilitation play key roles in optimising outcomes.

Chapter 12 – Spinal Cord Injury and Asia Impairment Scale

Spinal cord injury (SCI) is a devastating consequence of trauma that results in varying degrees of motor, sensory, and autonomic dysfunction. The burden of SCI extends beyond acute neurological deficits to long-term disability, psychosocial impact, and economic hardship. Early recognition, meticulous acute management, and standardised neurological assessment are essential to optimise outcomes and facilitate communication across treating teams.

Mechanism and Pathophysiology

Spinal cord injury may result from primary mechanical insult due to compression, distraction, or transection of the spinal cord, often associated with vertebral fractures or dislocations. This primary injury is followed by secondary injury processes including ischemia, edema, excitotoxicity, inflammation, and apoptosis, which evolve over hours to days. Prevention of secondary spinal cord injury through timely stabilisation and physiological optimisation is therefore a critical therapeutic goal.

Clinical Presentation

Patients with SCI may present with complete or incomplete loss of motor and sensory function below the level of injury. Autonomic dysfunction, including hypotension, bradycardia, bowel and bladder dysfunction, and impaired thermoregulation, is common. A high index of suspicion is required in all trauma patients, particularly those with high-energy mechanisms or altered consciousness.

Initial Assessment and Stabilisation

Initial management follows standard trauma principles with strict spinal immobilisation. Airway protection, oxygenation, and hemodynamic stabilization are priorities. Hypotension should be corrected promptly, as spinal cord perfusion is highly dependent on systemic blood pressure.

Neurogenic shock should be recognised and differentiated from hypovolemic shock. Maintenance of adequate mean arterial pressure is recommended to optimise spinal cord perfusion, particularly in the acute phase following injury.

Neurological Examination and Documentation

A detailed neurological examination should be performed once the patient is stabilised. This includes assessment of motor strength, sensory function, reflexes, and sacral sparing. Accurate documentation is essential for prognostication and monitoring neurological recovery.

ASIA Impairment Scale

The American Spinal Injury Association (ASIA) Impairment Scale is the most widely accepted system for classification of spinal cord injury. It provides a standardised method for assessing and documenting neurological deficits.

- ASIA A (Complete): No motor or sensory function preserved in the sacral segments S4–S5
- ASIA B (Sensory Incomplete): Sensory but not motor function preserved below the neurological level, including S4–S5
- ASIA C (Motor Incomplete): Motor function preserved below the neurological level, with more than half of key muscles having a grade less than 3
- ASIA D (Motor Incomplete): Motor function preserved below the neurological
- level, with at least half of key muscles having a grade of 3 or more • ASIA E (Normal): Normal motor and sensory function

The presence of sacral sparing is a critical determinant in distinguishing complete from incomplete injuries and carries important prognostic implications.

Imaging Evaluation

Computed tomography is the initial imaging modality of choice for evaluating spinal fractures and alignment. Magnetic resonance imaging provides superior assessment of the spinal cord, intervertebral discs, ligaments, and epidural hematomas. MRI is particularly useful in patients with neurological deficits or when surgical intervention is being considered.

Medical Management

There is no high-quality evidence supporting the routine use of high-dose corticosteroids in acute spinal cord injury. The potential risks, including infection and gastrointestinal bleeding, outweigh uncertain benefits, and routine administration is not recommended.

Hemodynamic optimisation, oxygenation, prevention of hypoxia and hypotension, and early surgical consultation form the cornerstone of medical management.

Surgical Management

Surgical intervention may be indicated for spinal instability, progressive neurological deficit, or ongoing spinal cord compression. The goals of surgery are decompression of neural elements, restoration of alignment, and stabilisation of the spinal column. Timing of surgery should be individualised based on neurological status, associated injuries, and overall physiological stability.

Rehabilitation and Long-Term Care

Early involvement of rehabilitation services is essential in patients with SCI. Comprehensive rehabilitation addresses mobility, spasticity, bladder and bowel management, pressure sore prevention, and psychological support. Long-term outcomes depend on injury severity, early management, and access to specialised rehabilitation services.

Prognosis

Neurological recovery after spinal cord injury is influenced by the severity and completeness of injury, early physiological management, and timely decompression when indicated. Incomplete injuries, particularly those with sacral sparing, have a more favourable prognosis.

Clinical Summary

Spinal cord injury management requires rapid stabilisation, prevention of secondary injury, accurate neurological classification using the ASIA Impairment Scale, and early multidisciplinary rehabilitation. Standardised assessment and protocol-driven care are essential to improve outcomes and quality of life for affected patients.

Chapter 13 – Conclusion

Neurotrauma, encompassing traumatic injuries to the brain and spinal cord, remains one of the most important causes of mortality, long-term disability, and socioeconomic burden in India. Rapid urbanisation, increasing motorisation, occupational hazards, and variable enforcement of safety regulations have contributed to a persistently high incidence of traumatic brain injury and spinal trauma. A significant proportion of affected patients are young adults in their most productive years, amplifying the impact on families, healthcare systems, and society at large.

Outcomes following neurotrauma are determined not only by the severity of the primary injury, but more importantly by the quality, timeliness, and consistency of early medical and surgical care. Hypoxia, hypotension, raised intracranial pressure, delayed diagnosis of surgically remediable lesions, and lack of organised critical care contribute substantially to preventable secondary injury. Wide variations in infrastructure, manpower, and clinical practices across healthcare settings in India further contribute to inconsistent outcomes.

The development and implementation of standardised, evidence-informed treatment protocols is therefore essential. Standard Treatment Guidelines provide a common framework for assessment, decision-making, and management across different levels of care, reduce unwarranted practice variation, and promote delivery of interventions that have the greatest impact on survival and functional recovery. Protocol-driven care has been shown to improve outcomes in neurotrauma by ensuring early airway protection, strict avoidance of hypoxia and hypotension, timely neuroimaging, appropriate neurosurgical intervention, and meticulous intensive care management.

These Standard Treatment Guidelines (STG – 2025) have been formulated to address the specific needs of neurotrauma care in the Indian context. They integrate current international evidence with practical considerations of resource availability and healthcare delivery systems. The scope of the guidelines spans the entire continuum of care, including epidemiology and prevention, classification and scoring systems, management of concussion and diffuse brain injury, evidence-based treatment of severe traumatic brain injury in adults and children, management of specific cranial injuries such as depressed skull fractures, and evaluation and treatment of spinal and spinal cord injuries.

The guidelines are intended to support clinicians involved in emergency care, neurosurgery, anaesthesia, critical care, orthopaedics, and rehabilitation. They are designed to complement clinical judgment rather than replace it, recognising that individual patient factors and institutional resources may necessitate flexibility in application. Emphasis is placed on prioritising achievable standards of care, prevention of secondary injury, multidisciplinary coordination, and early initiation of rehabilitation.

Neurotrauma care is a dynamic field, and these guidelines should be regarded as a living document. Periodic revision, audit of outcomes, training of healthcare personnel, and strengthening of trauma systems are essential to ensure continued relevance and effectiveness. Systematic adoption of these Standard Treatment Guidelines across emergency services, trauma centres, and intensive care units has the potential to significantly reduce preventable deaths and disabilities due to neurotrauma and improve long-term outcomes and quality of life for patients and their families.