Traumatic Brain Injury

Pathophysiology, Monitoring, and Mechanism-Based Care

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KEYWORDS

• Brain trauma • Primary and secondary injury • Intracranial hypertension
• Osmotherapy • Metabolic suppression • Neurological assessment
• Neurodiagnostics • Hypothermia

KEY POINTS

- Traumatic brain injury (TBI) is a significant personal and financial burden, with approximately 50,000 patients dying annually from TBI.
- Both blunt and penetrating brain trauma initiate the cascade of secondary brain injury.
- Secondary brain injury is often a process affecting relative volumes of intracranial blood, brain, and cerebrospinal fluid. Effective, mechanism-based therapies modulate relative volumes of intracranial content and control the evolution of intracranial hypertension.
- Techniques for monitoring intracranial pressure (ICP) and brain-tissue oxygen levels can yield actionable, real-time information and direct therapy. Goal-directed therapy to control ICP elevations and maintain brain-tissue oxygen levels can modulate the effect of secondary brain injury and improve outcomes.

INTRODUCTION: SCOPE OF ISSUES

Traumatic brain injury (TBI) is among the leading causes of death in patients younger than 45 years.[1] TBI is not solely a leading cause of death and disability[2] but also represents a significant financial and personal burden, both individually and on society. There are approximately 235,000 hospital admissions for patients surviving the initial traumatic event.[2] TBI has a mortality of approximately 50,000 patients; nearly 80% of patients who suffer mild to moderate TBI have neurologic sequelae 3 months after injury, many of whom require rehabilitation services.[1–3] In addition to the financial

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costs of caring for the patient after TBI, there are also human costs arising from loss of employment and productivity as well as altered family dynamics, low self-esteem, and grief associated with dramatic life changes and lost potential. Severe TBI is defined as a Glasgow Coma Scale (GCS) Score of 3 to 8 out of 15.[2,4,5] Mild TBI is defined by a GCS of 13 to 15, and moderate TBI is defined by a GCS of 9 to 12.[2,4,5] A summary of the GCS is shown in **Table 1**.[2,4–6] A comparison of mild, moderate, and severe TBI is shown in **Table 2**.[2,4,5] This article reviews the pathophysiology and mechanism-based management of TBI. The scope of the discussion includes epidemiology and pathophysiology, and appropriate nursing considerations related to assessment, diagnostics, and clinical management of persons with TBI. In addition, the reader is guided through a case study to explore the application dimensions of these considerations.

TBI PATHOPHYSIOLOGY AND TRAJECTORY

TBI may be classified in two ways. In closed head injury, the brain is injured from trauma to the skull or a sudden, severe motion causing the brain to make contact by force with the inner table of the skull.[2,5,7,8] This contact may lead to direct tissue injury and capillary hemorrhage such as cerebral contusion or vascular injury, causing epidural or subdural hematoma.[5,7] A second classification is penetrating brain trauma, an injury resulting from a projectile or sharp object (generally) penetrating the scalp, cranial vault, meninges, and brain tissue itself.[9,10] This injury also exposes the intracranial cavity and its contents to the external environment.[9,10] The small cross-sectional area allows maximal delivery of force at the point of contact and maximizes penetration.[9] A Comparison of blunt versus penetrating brain trauma is illustrated in **Fig. 1**.

TBI may be further differentiated into primary and secondary brain injury.[4,5,7–9,11–13] The first stage, primary brain trauma, begins at the moment of injury and may be the result of a depressed skull fracture, closed head injury, penetrating brain trauma, subdural/epidural hematoma, and/or traumatic intracerebral hemorrhage, as well as brain contusion or laceration.[1,5,7–9,11,13] Diffuse brain injury may occur from rapid acceleration/deceleration and cause diffuse axonal injury and/or brain edema.[1,5,7,11] The second stage, secondary brain injury, begins following the immediate trauma and includes brain ischemia, autoregulatory failure, anaerobic metabolism, increased tissue lactate,[7] cellular energy failure, release of excitatory amino acids, and loss of cell membrane integrity.[8,11–13] This loss of membrane integrity allows sodium and

Table 1
Glasgow Coma Scale score

Motor Response (M)	Verbal Response (V)	Eye Opening (E)
Follows commands: 6		
Localizing to stimulation: 5	Oriented: 5	
Withdrawal to painful stimulation: 4	Confused, appropriate: 4	Spontaneous: 4
Flexion (decorticate) posturing: 3	Disoriented, inappropriate: 3	Eye opening to voice: 3
Extensor (decerebrate) posturing: 2	Incomprehensible sounds: 2	Eye opening to stimulation: 2
No response: 1	No response: 1	No response: 1

Data from Refs.[2,4–6]