Management of Traumatic Brain Injury: An Update

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KEYWORDS

• TBI • ICP • Sedation • PSH • Monitors • Multimodality • Informatics

KEY POINTS

- The management of patients with acute traumatic brain injury remains a cornerstone of Neurocritical care.
- The foundation of this management remains excellent critical care with attention to the airway, oxygenation, and hemodynamic support to avoid secondary injury associated with hypoxia and hypotension.
- There are brain-specific areas of focus, such as identifying and treating intracranial hypertension, avoiding seizures, and fever, which can lead to secondary injury and worsen clinical outcomes.

INTRODUCTION

The management of patients with acute traumatic brain injury (TBI) remains a cornerstone of Neurocritical care. TBI is a leading cause of death and disability in people less than 40 years of age with more than 2.5 million cases of TBI occurring in the United States each year.[1] The foundation of this management remains excellent critical care with attention to the airway, oxygenation, and hemodynamic support to avoid secondary injury associated with hypoxia and hypotension. In addition, there are brain-specific areas of focus, such as identifying and treating intracranial hypertension, avoiding seizures, and fever, which can lead to secondary injury and worsen clinical outcomes. In this article, the authors discuss the components of Neurocritical care support for these patients and review common techniques and therapies used in their care.

SEDATION AND ANALGESIA

It is generally accepted that minimizing sedation for patients in the intensive care unit (ICU) leads to improved outcomes specifically by reducing length of hospitalization, decreasing time on the ventilator, decreasing the incidence of delirium, and facilitating mobilization.[2] However, patients who suffer severe TBI have traditionally been heavily sedated early in their management for a variety of reasons. The rationale for early sedation and analgesia in this population has been to diminish pain and anxiety and improve ventilator synchrony. In addition, processes specifically targeted to patients with acute brain injury include decreasing oxygen consumption and metabolic demand in a time of acute cellular distress, mitigating secondary injury, and preventing an increase in intracranial pressure (ICP). Therefore, specific sedative agents and analgesics are typically used in this patient population.

Most sedatives decrease metabolic stress on acutely injured brain tissue. They achieve this by decreasing the cerebral metabolic rate of oxygen in a dose-dependent fashion and thereby minimize the mismatch between substrate delivery and energy demands.[3] Because cerebral metabolism is

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usually coupled to cerebral blood flow (CBF), the total CBF will decease when patients are given sedation. Changes in cerebral blood flow are seen most consistently with propofol infusions but may occur with benzodiazepines and some analgesics as well. The net effect of deceasing metabolism and blood flow is a decrease in ICP. Importantly, fentanyl and remifentanil have been associated with acute drops in mean arterial pressure, which cause a reflex cerebrovasodilation resulting in elevated ICP. In addition, if cerebral autoregulation is altered, acute hypotension due to analgesic administration may lead to further brain ischemia and secondary injury. Synthetic opiates such as fentanyl are still used for analgesia in this population; however, care should be given to maintain an adequate mean arterial pressure throughout the administration. **Table 1** lists some commonly used sedatives and analgesics used after acute TBI and their main disadvantages and benefits.

INTRACRANIAL PRESSURE MONITORING AND MANAGEMENT

Monitoring

It has been proven that patients with elevated ICP have worse outcomes and higher risk of mortality.[4,5]

The indications for ICP monitoring in severe TBI from the fourth edition of Management of Severe Traumatic Brain Injury[6] are as follows:

- Management of severe TBI patients using information from ICP monitoring is recommended to reduce in-hospital and 2-week post-injury mortality.
- The guidelines no longer specifically state which patients should be chosen for monitoring due to insufficient high quality evidence to support the recommendation.
- Clinicians should use their clinical judgement and perform intracranial monitoring in patients who are high risk for intracranial hypertension and clinical deterioration.

Despite the conflicting evidence and the lack of large randomized controlled trials studying its effect on outcomes, ICP monitoring has been a cornerstone in the management of patients with severe TBI. The BEST:TRIP trial recently studied 324 patients with severe TBI in Bolivia and Ecuador and found no difference in clinical outcomes at 6 months between patients that were randomized to the ICP monitoring group and those managed using standard imaging, osmotic medications, and clinical examination. Limitations of this study include variability in prehospital care, lack of previous experience from the participating clinicians with the use of ICP monitoring, and inconsistent availability of post-ICU support and rehabilitation.[7]

A group from New York performed a retrospective review of patients entered into the "TBI-trac" database that was designed and implemented by

Table 1
Frequently used sedatives and analgesics after traumatic brain injury

Sedative/Analgesic	Mechanism	Risks	Advantages
Midazolam	GABA agonist	Tachyphylaxis, may increase delirium and length of MV	Amnesia, suppresses seizures, decreased $CMRO_2$ and ICP
Propofol	GABA agonist	Hypotension, propofol infusion syndrome, increased triglycerides	Decreases CMROs and ICP, rapid onset, suppresses seizures
Barbiturates	GABA agonist	Hypotension, immune suppression, ileus	Decreases $CMRO_2$ strongly decreases ICP
Fentnayl/sufntnayl/ remifentanil	Mu receptor agonist	Hypotension and increased ICP, prolonged MV	Provides analgesia
Morphine	Mu receptor agonist	Unpredictable effect on ICP	Low cost
Dexmedetomidine	Alpha 2 agonist	Hypotension, bradycardia, limited effect on ICP	Minimal respiratory depression, quick onset, sedative and analgesic
Ketamine	NMDA agonist	Hallucinations, dysphoria, variable effect on ICP	Fast onset, minimal hypotension, suppresses seizures, sedation, and analgesia

Abbreviations: $CMRO_2$, cerebral metabolic rate of oxygen; GABA, gamma-aminobutyric acid; MV, mechanical ventilation; NMDA, *N*-methyl- D-aspartate.