Management of Traumatic Brain Injury

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KEYWORDS

• Traumatic brain injury • Neurotrauma • Secondary injury • Intracranial monitoring • Intracranial pressure • Craniotomy • Craniectomy • Decompression

KEY POINTS

- The diagnosis of severe traumatic brain injury is based on a variety of clinical and radiographic data and encompasses a wide heterogeneity of structural and physiologic insults.
- The concept of treatment thresholds is somewhat outdated; although there are specific physiologic ranges at which secondary injury clearly takes place, treatment of an individual patient's physiology at a given point in time must take into account numerous concurrent events, including the evolution of extracerebral injuries, structural brain lesions, cerebral edema, and cerebral hypoxia/ischemia.
- The options for treatment of severe traumatic brain injury are just as varied as the presenting pathologies and may include surgery for evacuation of mass lesions or decompression of herniating or compressed cerebral tissues, drainage of cerebrospinal fluid, pharmacologic sedation and paralysis, ventilator management, hyperosmolar euvolemic therapy, and prophylaxis against seizures, thromboses, and a variety of other complications.

INTRODUCTION

According to the Centers for Disease Control and Prevention, injury remains the leading cause of death in the United States for all persons aged 1 to 44 years, is the third leading cause of death for those aged 45 to 64 years, is the fifth most common cause of death for infants less than a year of age, and ranks seventh in those 65 years and older.[1] Traumatic brain injury (TBI) comprises the cause of death for approximately one-third of people with multitrauma.[2] The public health importance of TBI, therefore, cannot be overestimated.

RELEVANT ANATOMY AND PATHOPHYSIOLOGY

Severe TBI (sTBI) has traditionally been defined as those presenting with head trauma and brain injury with a postresuscitation Glasgow Coma Scale (GCS)[3] score of 3 to 8, although other classification schema exist. Patients with sTBI, and some with

Disclosure Statement: The authors have nothing to disclose.
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Surg Clin N Am 97 (2017) 1237–1253
http://dx.doi.org/10.1016/j.suc.2017.08.001
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so-called moderate TBI, that is, a GCS score of 9 to 12, require intensive care, sometimes for several days to a few weeks. The pathophysiology of TBI involves the initial blow (primary injury) that may result in numerous structural pathologies as well as initiation of the chemical, electrical, and inflammatory cascade of physiologic events that comprise the secondary injury of the brain. Furthermore, secondary insults, such as hypotension, hypoxia, seizure, and other physiologic events, have a profound impact on the degree of secondary injury sustained and ultimately the functional outcome of patients. Patients with polytrauma and sTBI represent a significant challenge because of the potential for ongoing secondary insults from other organ injuries and vascular and musculoskeletal trauma.

Thus, the treatment of sTBI must begin the moment that patients are assessed by first responders. Emergency personnel and physicians in multiple specialties must be conversant with the diagnosis and management of severe TBI so as to prevent secondary insults to the degree possible, to rapidly coordinate the surgical care of structural injuries requiring surgery, and to minimize secondary cerebral injury to improve long-term outcomes after sTBI.

Structural cerebral injury occurring as part of the primary injury cannot currently be repaired, but the effects of structural injury must be mitigated. Surgical repair of a variety of structural injuries is often undertaken early (in the case of compressive lesions causing pressure on the brain) or later in the course (as in the case of evolving cerebral edema, craniofacial repairs, and treatment of cerebrospinal fluid [CSF] leak or infection). Mass lesions may be classified as extra-axial (outside the brain tissue but inside the cranium) or intra-axial (within the brain tissue). Certain intracranial hematomas require immediate surgical intervention, generally those with sufficient volume to create outright cerebral herniation or cerebral compression that is symptomatic, that is, causing coma, neurologic deficit, or intracranial hypertension.

Management of intracranial pressure (ICP) in the face of hemorrhagic lesions and cerebral edema can be challenging, depending on the space occupied in the intracranial compartment by hematomas and edematous brain tissue. The Monroe-Kellie hypothesis states that the intracranial compartment has fixed volumes of the following components: cerebral tissue, cerebral blood, and CSF. As one compartment increases in volume or a mass lesion is added to the compartment, compensation must occur to maintain a normal ICP. This compensation initially involves displacement of CSF and venous blood into the spinal canal; but once a critical volume is reached in the intracranial compartment, cerebral compliance decreases and elastance increases, resulting in larger changes in ICP with smaller changes in volume. Therefore, small reductions in CSF can have a large impact on ICP control at this stage; likewise, removing mass lesions or increasing the size of the cranial compartment via craniectomy and duraplasty can very effectively control ICP.

CLINICAL PRESENTATION

Patients with sTBI by definition present in coma. They often arrive at the hospital having been intubated in the field because of suppression of respiratory function caused by the brain injury and/or inability to protect the airway because of the depressed level of consciousness. Trauma patients with TBI must be assessed for the presence of other injuries and should be presumed to have them until proven otherwise, given their inability to report history or symptoms.

Depending on the mechanism of injury, other injuries may be rather self-evident or occult. Typical high-speed motor vehicle crash patients or a pedestrian struck by a vehicle will often present with gross signs of trauma, including abrasions, contusions,