Pathophysiology and Management of Intracranial Hypertension and Tissular Brain Hypoxia After Severe Traumatic Brain Injury

An Integrative Approach

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KEYWORDS

• Intracranial pressure • Intracranial hypertension • Acute brain injury • Cerebral perfusion pressure • Cerebral oxygenation • Brain hypoxia • Multimodal monitoring • Traumatic brain injury

KEY POINTS

- Intracranial hypertension (IHT) is a major mechanism of damage after acute brain injury.
- IHT and brain hypoxia (BH) are potentially life-threatening secondary brain insults and they are closely interconnected.
- Invasive monitoring of intracranial pressure is the gold standard to obtain reliable values and is the cornerstone to guide treatment, whereas brain oxygenation may be monitored globally through saturation of jugular bulb (SjO_2) or locally through determination of oxygen tissular pressure ($ptiO_2$).
- There are multiple causes of IHT and BH, and a pathophysiological assessment is fundamental to adopt appropriate therapy.
- Achieving homeostasis of basic physiologic variables (physiologic neuroprotection) and avoiding secondary insults are crucial steps to control IHT and prevent BH.

INTRODUCTION

Increased intracranial pressure (ICP) can be a medical or surgical emergency.[1] Both intracranial and systemic events contribute to increased ICP after traumatic brain injury (TBI).[1,2] Intracranial hypertension (IHT) can be life-threatening by mechanical or vascular effects. Mechanical effects are mediated through different types and degrees of cerebral displacement and herniation. Vascular effects result from lowering cerebral perfusion pressure (CPP), which is defined as mean arterial pressure (MAP) minus ICP. The CPP is the driving force of cerebral blood flow (CBF). As the CPP decreases, CBF may become insufficient for adequate brain tissue perfusion and oxygenation.[3,4] The adequate levels of CBF and CPP vary among patients,

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and the optimal CPP value changes over time and is normally regulated by cerebral autoregulation. Increased ICP and low CPP are associated with mortality and poor long-term outcome.[5–9] Ischemic lesions are highly prevalent in autopsy specimens of individuals who die after severe TBI. Therefore, preventing brain hypoxia (BH) and ischemia by maintaining adequate supplies of oxygen and glucose represent major priorities in the management of any form of severe acute brain injury.[10–13] Recently, monitoring of cerebral tissue oxygenation has become increasingly used.[14–16] The causes of brain tissue hypoxia are multiple and may even occur in the absence of ICP increase or CPP decrease; thus, an exhaustive analysis of oxygen supply and utilization is necessary to optimize care and improve outcomes in TBI.[16–20]

WHEN TO START THERAPY FOR INTRACRANIAL HYPERTENSION?

Normal ICP varies with age, body position, and clinical condition.[1,21] In healthy individuals in supine position, it ranges between 7 and 15 mm Hg. Meanwhile, it becomes negative on standing (average −10 mm Hg).[1,2,21] In term infants, 1.5 to 6.0 mm Hg is considered normal, whereas in children, these values range between 3 and 7 mm Hg.[22] ICP can be increased transiently in physiologic situations, such as coughing or sneezing. In critically ill patients, occasional elevations can be observed with changes in position, aspiration of secretions, asynchrony with mechanical ventilation, and physical therapy.[1,2,5]

IHT is traditionally defined as ICP greater than 20 mm Hg for more than 5 to 10 minutes.[23,24] Treatment is generally considered indicated above this threshold. In other situations, such as after decompressive craniectomy or when there are contusions close to the midbrain (temporal lobes, basal region of frontal lobes), it may be advisable to use a threshold of 15 mm Hg to start therapy.[25] Yet, the ICP threshold and the optimal time to initiate or intensify ICP treatment are subjects of debate because available evidence has limitations.[5,6,24–28] The cutoff of 20 mm Hg was established from retrospective analysis of data from the Traumatic Coma Data Bank.[5] This threshold fails to take into account the age of the patient or the lesional type. Furthermore, the cutoff of 20 mm Hg is taken as a static parameter, when in truth is highly dynamic and dependent on the situation. The last version of the Brain Trauma Foundation guidelines suggests initiating treatment when ICP is greater than 22 mm Hg.[28] The pressure reactivity index evaluates the autoregulatory capacity of the cerebral vasculature by analyzing the changes of ICP in relation to changes in MAP, and this dynamic correlation may be used to individualize the CPP target and consequently the ICP threshold for treatment.[29] Measurements of cerebral perfusion also may be used to individualize ICP therapy.[30]

INTRACRANIAL PRESSURE MONITORING

Invasive ICP measurement (intraparenchymal, intraventricular) is the ''gold standard'' for monitoring.[1,2] ICP waves are the result of pulse transmission of arterial pressure via the choroid plexus to the cerebrospinal fluid (CSF) and brain parenchyma.[31] Intracranial compliance can be assessed by ICP waveform analysis and the correlation coefficient between the ICP waveform amplitude and the mean ICP.[31–34] The relationship between ICP and MAP fluctuations can provide vital information pertaining to the underlying status of the cerebrovascular reactivity and autoregulation.[35,36]

Attempts have been made to identify loss intracranial compliance through ICP waveform analysis. The normal ICP waveform has 3 components of progressively decreasing amplitude (P1 > P2 > P3), but as intracranial compliance gets exhausted, the P2 component becomes higher than P1, thus adopting a pyramidal form (**Fig. 1**).[32,33,37] Oftentimes the ICP waveform changes before any major rise in ICP can be detected. Thus, the analysis of the ICP wave is a useful tool to gauge the intracranial compliance.[31–33,37,38] When ICP becomes elevated, abnormal waves begin to appear. These pathologic waves, known as “Lundberg waves,” reflect poor compliance. A waves, also known as plateau waves, represent sustained, pronounced elevations of ICP and demand emergency treatment. B waves are briefer and less severe raises in ICP that uncover decreased intracranial compliance but do not always necessitate emergent interventions (see **Fig. 1**).[37–39]

PATHOPHYSIOLOGY OF INCREASED INTRACRANIAL PRESSURE

For a correct therapeutic approach, it is essential to take into account the following premises (**Table 1**):

1. Any increase in an intracranial component (brain tissue, arterial or venous blood, or CSF) comes at the expense of another component (Monroe-Kellie principle). The addition of