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BETA ANTAGONIST IN TRAUMATIC BRAIN INJURY

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Abstract

Background: In developed countries, traumatic brain injury is the leading cause of death and disability among adults.

Keywords: traumatic brain injury, sympathetic storm, propranolol

Introduction:

Traumatic brain injury (TBI) is a serious public health problem that results in death and disability for thousands of people each year. The goal of this article is to provide a general review of the epidemiology, acute care, and chronic management of adult patients with traumatic brain injuries **(1).**

I **I. Definition:**

According to the Centers for Disease Control and Prevention (CDC), a TBI is disruption in the normal function of the brain that can be caused by a bump, blow, or jolt to the head. Traumatic impact injuries may be closed (non-penetrating) or open (penetrating) **(2)**.

I **II. Epidemiology:**

In 2014, the CDC documented 2.53 million TBI-related emergency department (ED) visits. There were approximately 288,000 TBI-related hospitalizations and 56,800 TBI-related deaths. These data include both adults and children **(3).**

Emergency Department Visits and Deaths

TBI-related ED visits and deaths have increased steadily. This increase may be partially attributed to improved brain injury awareness among providers and more accurate reporting and surveillance methods**.(4)**

Mechanisms of Injury:

The most common mechanisms of injury, in descending order of frequency, include unintentional falls, motor vechile accident (MVA), assault, other (no mechanism specified) **(5).**

Severity:

Understanding brain injury severity helps with both prognosis, recovery and anticipating patients' rehabilitation needs **(6)**.

	GCS[a] (First 24 h)	Loss of Consciousness	Alteration of Consciousness	Imaging	PTA
Mild	13–15	0–30 min	Up to 24 h	Normal	0–1 d
Moderate	9–12	>30 min and <24 h	>24 h	Normal or abnormal	>1 d and <7 d
Severe	3–8	>24 h	>24 h	Normal or abnormal	>7 d

Note: Some institutions use the term "mild complicated TBI" for patients who meet the mild classification by GCS, loss or alteration of consciousness, and posttraumatic amnesia but have abnormal imaging findings, such as a subdural hematoma or depressed skull fracture.[4]

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Glasgow coma scale:

Score	Eye Opening	Verbal	Motor[a]
1	None	None	None
2	To pain	Incomprehensible speech	Extension (decerebrate posturing) to pain
3	To speech	Inappropriate speech	Flexor (decorticate posturing) to pain
4	Spontaneous	Confused	Withdraws to pain
5	–	Oriented	Localizing response
6	–	–	Follows directions

[a] The motor score is the only part of the GCS with prognostic value.

Treatment options:

Before engaging in the treatment of PSS, it is critical to assess for and treat any potential underlying disorders, specifically hypovolemia, infection, or electrolyte disturbance. Following this, the goals of treatment are to :
(1) eliminate the cause;
(2) decrease the frequency of events; and
(3) decrease the intensity of events **(8).**

Beta-Adrenergic Antagonists:

One of the early considerations for the use of beta blockers for head injury came with the publication on a volume-targeted therapy principle (Lund Therapy) that aimed to achieve and maintain normovolemia within the brain. It was postulated that opening of the blood-brain barrier led to brain edema. Thus, effective treatment of brain edema would include reduction of hydrostatic capillary pressure and preservation of normal colloid osmotic pressure**. (9)**

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