# Acute brain injury

- Aquaporins in Acute Brain Injury: Insights from Clinical and Experimental Studies
- Neurobiology of Chronic Pain, Posttraumatic Stress Disorder, and Mild Traumatic Brain Injury
- Assessment of White Matter Changes Using Quantitative T1rho Mapping in an Open-Field Low-Intensity Blast Mouse Model of Mild Traumatic Brain Injury (mTBI)
- The Many Faces of Child Abuse: How Clinical, Genetic and Epigenetic Correlates Help Us See the Full Picture
- Timing of Magnetic Resonance Imaging (MRI) in Moderate and Severe TBI: A Systematic Review
- Multiomics Analysis Reveals Role of ncRNA in Hypoxia of Mouse Brain Microvascular Endothelial Cells
- Electro-Acupuncture to Treat Disorder of Consciousness (AcuDoc): Study Protocol for a Randomized Sham-Controlled Trial
- Traumatic Brain Injury: Novel Experimental Approaches and Treatment Possibilities

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“Acute brain injury” and “early brain injury” are related terms but not synonymous. They are often used to describe different phases or aspects of brain injury, particularly in neuroscience and neurology.

## Definition

Acute brain injury refers to damage or trauma to the brain that occurs suddenly and often involves a rapid onset of symptoms. This can result from various causes, including traumatic events (such as head injuries or accidents), strokes, infections, or other medical conditions affecting the brain. Time Frame: The term “acute” generally implies the early stages of injury, but it does not specify a specific time frame. Acute brain injury can encompass the initial moments of injury and may extend into the early hours, days, or weeks following the event. Clinical Features: Acute brain injury is characterized by the immediate consequences of the injury, such as neurological symptoms, inflammation, and cellular damage. It may involve processes such as edema (swelling), bleeding, and changes in blood flow to the affected areas of the brain.

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Early Brain Injury:

Definition: Early brain injury refers to the initial damage to the brain that occurs shortly after the onset of a triggering event, such as trauma or a medical condition. It is a concept often used in the context of subarachnoid hemorrhage (bleeding in the space around the brain). Time Frame: The term “early” suggests focusing on the immediate aftermath of the triggering event, typically within the first 72 hours. However, the specific time frame may vary depending on the use context. Clinical Features: Early brain injury involves pathological processes that occur shortly after the insult to the brain. This can include mechanisms such as oxidative stress, inflammation, and changes in cerebral blood flow that contribute to secondary brain damage. In summary, while acute brain injury refers more broadly

to damage occurring in the early stages of an insult to the brain, early brain injury is a term often used in a more specific context, such as subarachnoid hemorrhage. Both terms highlight the importance of understanding and addressing the immediate consequences of brain injury to minimize secondary damage and optimize outcomes.

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Acute brain injuries, such as traumatic brain injury ischemic stroke and hemorrhagic stroke, are a leading cause of death and disability worldwide. While characterized by clearly distinct primary events damage in strokes and biomechanical damage in traumatic brain injuries-they share common secondary injury mechanisms influencing long-term outcomes. Growing evidence suggests that a more personalized approach to optimize energy substrate delivery to the injured brain and prognosticate towards families could be beneficial. In this context, continuous invasive and/or non-invasive neuromonitoring, together with clinical evaluation and neuroimaging to support strategies that optimize cerebral blood flow and metabolic delivery, as well as approaches to neuro prognostication are gaining interest [1)].

# Etiology

Acute brain injury can result from various etiologies, which can be broadly categorized into traumatic and non-traumatic causes. Here’s a detailed overview:

Traumatic Brain Injury (TBI) Blunt Trauma:

Falls: Common in older adults and children. Motor Vehicle Accidents: Collisions can cause significant head injuries. Sports Injuries: Contact sports can lead to concussions and other TBIs. Assaults: Physical violence can result in blunt force trauma. Penetrating Trauma:

Gunshot Wounds: High-energy projectiles can cause severe brain damage. Stabbings: Sharp objects can penetrate the skull and damage brain tissue. Non-Traumatic Brain Injury Vascular Causes:

Stroke: Ischemic Stroke: Caused by a blockage in blood vessels supplying the brain. Hemorrhagic Stroke: Resulting from a ruptured blood vessel, leading to bleeding in the brain. Transient Ischemic Attack (TIA): Temporary blockage causing symptoms similar to stroke. Hypoxic-Ischemic Injury:

Cardiac Arrest: Lack of blood flow to the brain can cause injury. Drowning: Leads to oxygen deprivation. Choking: Can obstruct airway and lead to hypoxia. Infectious Causes:

Meningitis: Infection of the protective membranes covering the brain and spinal cord. Encephalitis: Inflammation of the brain often due to viral infections. Metabolic Causes:

Hypoglycemia: Low blood sugar levels can damage brain cells. Hyperglycemia: High blood sugar levels can lead to complications, especially in diabetic patients. Electrolyte Imbalances: Abnormal levels of sodium, potassium, or calcium can lead to brain dysfunction. Toxic Causes:

Drug Overdose: Substances like opioids, stimulants, or alcohol can lead to acute brain injury. Poisoning: Exposure to toxic substances (e.g., carbon monoxide) can impair brain function. Other Causes:

Seizures: Status epilepticus can lead to acute brain injury due to prolonged seizure activity. Tumors:

Rapid growth of tumors can cause increased intracranial pressure and subsequent injury.

### Invasive cardiovascular procedures

Over the past 50 years, the number and invasiveness of percutaneous cardiovascular procedures globally have increased substantially. However, cardiovascular interventions are inherently associated with a risk of acute brain injury, both periprocedural and postprocedural, which impairs medical outcomes and increases healthcare costs. Current international clinical guidelines generally do not cover the area of acute brain injury related to cardiovascular invasive procedures. In this international Consensus Statement, we compile the available knowledge (including data on prevalence, pathophysiology, risk factors, clinical presentation, and management) to formulate consensus recommendations on preventing, diagnosing, and treating acute brain injury caused by cardiovascular interventions. We also identify knowledge gaps and possible future directions in clinical research into acute brain injury related to cardiovascular interventions [2].

## Protective Ventilation Strategies for acute brain injury

Protective Ventilation Strategies for acute brain injury.

## Systematic reviews and meta-analysis of randomized controlled trials

In a Systematic review and meta-analysis of randomized controlled trials Tsai et al. [3] determine whether a liberal (hemoglobin 9–10 g/dL) versus restrictive (hemoglobin 7–8 g/dL) red blood cell transfusion strategy leads to better neurological outcomes or survival in adult patients with acute brain injury, by conducting a meta-analysis and trial sequential analysis of randomized controlled trials (RCTs).

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The study aggregates data from patients with vastly heterogeneous pathologies—**traumatic brain injury**, **intracerebral hemorrhage**, **subarachnoid hemorrhage**, **ischemic stroke**, etc.—as if they constitute a uniform clinical entity. This epistemological shortcut undermines any attempt at nuanced interpretation.

> **Neurosurgical Reality Check**:

A transfusion strategy suitable for vasospasm-prone SAH may be harmful in ischemic stroke or TBI with raised ICP. Lumping them together is not simplification—it's distortion.

### Underpowered Despite Meta-Analysis

With only **6 RCTs** and **2,599 patients**, the meta-analysis lacks the power to make definitive clinical recommendations. The **trial sequential analysis (TSA)**, although methodologically sound, only

confirms what's obvious: the data are inconclusive.

> **Red Flag**:

A TSA on poorly matched, heterogeneous studies is like calibrating a broken clock—technically impressive, but ultimately misleading.

### Outcome Definition Failures

The primary endpoint—**"unfavorable neurological outcome"**—is ill-defined and inconsistently reported across included trials. There's **no harmonized scale** (e.g., GOS, mRS), which renders pooled effect sizes speculative at best.

> Translation: Garbage in, garbage out. Meta-analysis cannot rescue poor-quality endpoints.

### ⚠ Clinical Irrelevance of P-Values

The reported lack of statistical difference in mortality or outcomes (e.g., RR 0.97, p = 0.70) is **presented as equivalence**, but this is statistically and clinically incorrect. **Absence of evidence is not evidence of absence.**

> There's a disturbing trend in modern meta-research: turning null results into dogma.

## Neurosurgical Implications? Weak to Nonexistent

- No data on **transfusion timing**, **intracranial pressure dynamics**, or **neuromonitoring correlation** - No stratification by **surgical intervention**, **coagulopathy**, or **vasospasm management** - No insight into **neuroanemia** thresholds or thresholds for high-risk subgroups (e.g. delayed ischemia, decompressive craniectomy)

> This is a study about hemoglobin numbers, not neurosurgical patients.

## Final Verdict

Despite its polished statistical packaging, this meta-analysis exemplifies the **perils of academic overreach**. It reduces complex neurosurgical conditions to abstract hemoglobin thresholds, repurposes weak RCTs into a "definitive" conclusion, and then buries clinical nuance under statistical noise.

> **Recommendation**: Do not apply its conclusions blindly. Transfusion thresholds in neurocritical care must remain individualized until pathology-specific, neurophysiology-informed trials are conducted.

1)

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