Many changes occur in the brain following a traumatic injury. Historically, the pathophisiology of TBI has been divided into primary and secondary injury.

Primary brain injury can result from a blow to the cranium or from rapid acceleration/deceleration, or rotation of the brain when it is slammed back and forth against the bony structures inside the skull. Primary brain injury can be further subdivided into focal and diffuse injury but most brain injuries have both of them.

Secondary brain injury. This lesion occurs in the postinjury phase and is due to associated physiological impairments resulting from the primary injury. Decreased cerebral perfusion, increased intracraneal pressure, brain edema, hypoxemia, and circulatory shock (hypotension) may increase brain injury. The main goal in the acute phase after a TBI is to stop the oxygen deprivation and increase the blood flow to maintain de cerebral perfusion pressure. Most secondary injury occurs during the first 12-24 hours after trauma, but it may occur at any time during the first 5-10 days in patients with very severe primary brain injury (Cooper, 1985). Focal ischemia, which occurs in approximately 90 percent of TBI cases, is implicated in initiating many of the toxin neurochemical processes. Increased levels of excitatory amino acids, endogenous opioid peptides, and acetylcholine are among the neurochemical agents that contribute to secondary brain damage.
Elevation of intracraneal pressure are related with poorer functional outcomes, especially if pressure is beyond 40 mmHg. Cerebral edema or swelling is attributed to disruption of the blood-brain barrier and impairment of vasomotor autoregulation with concomitant dilatation of cerebral blood vessels.
Physians can not treat primary brain injury but they can act decreasing secondary brain injury as a way to avoid further brain injury.