Traumatic Brain Injury (TBI)

In animals with traumatic brain injury (TBI), two distinct aspects of injury are noted. The primary injury to the brain is caused by the actual traumatic event and is generally not treatable. The secondary injury occurs due to hypoperfusion as well as the series of biochemical and physiologic events initiated by the trauma. Secondary brain injury is the more interesting sequelae to TBI and results from the biochemical cascade initiated by excitatory neurotransmitter release, intracellular calcium influx, ischemia, hypoperfusion and inflammation. Some of these aspects are easier to control than others and systemic consequences of trauma (e.g. hypotension, hypoxia, acidosis) can worsen clinical signs and the secondary injury. There are also intracranial events that contribute to secondary injury, and these include intracranial hypertension, compromised blood-brain barrier, and cerebral edema. The aim of this lecture is to review the physiology of intracranial homeostasis, effects of trauma on intracranial pressure (ICP), assessment strategies for the head trauma patient and treatment strategies for elevated ICP.