Updates in the Management of the Small Animal Patient with Neurologic Trauma

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KEYWORDS

• Neurotrauma • Traumatic brain injury • Head trauma • Acute spinal cord injury • Resuscitation • Dog • Cat • Cerebral ischemia

KEY POINTS

- Neurotrauma, including traumatic brain injury (TBI) and acute spinal cord injury (SCI), is a cause of significant morbidity and mortality in veterinary patients.
- Damage to neuronal cells can be divided into primary and secondary injury.
- Pharmacologic and nonpharmacologic therapies are directed at addressing primary injury in SCI as well as minimizing the effects of secondary injury in both TBI and SCI.
- Prognosis for neurotrauma patients depends on the severity of injury, the site of the lesion, and the timing and efficacy of the treatment of primary and secondary injury.

Neurologic trauma, encompassing traumatic brain injury (TBI) and acute spinal cord injury (SCI), is a cause of significant morbidity and mortality in veterinary patients. In one recent retrospective study evaluating blunt trauma in dogs, a diagnosis of TBI was made in 25% of cases and was associated with increased mortality.[1] Acute SCIs occurring secondary to trauma (including vertebral fracture or luxation [VFL], traumatic intervertebral disk herniation, spinal cord parenchymal contusions, and extra-axial hemorrhage) are also common, with an estimated incidence rate of 14% in cats and 9% in dogs based on the information from single-center retrospective studies.[2,3] The causes of neurologic trauma in dogs and cats include motor vehicular trauma, falls, crush injuries, bite wounds, missile injuries (eg, gunshot wounds), and either accidental or purposeful human-inflicted trauma.[4–7] Essential to the management of TBI and SCI is a thorough understanding of the pathophysiology of the primary and secondary injury that occurs following trauma.[8] This article reviews the

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pathophysiology of this primary and secondary injury, as well as recommendations regarding clinical assessment, diagnostics, pharmacologic and nonpharmacologic therapy, and prognosis.

MANAGEMENT GOALS

Damage to nervous tissue can be divided into primary and secondary injury. Primary injury occurs immediately after trauma and is the direct result of traumatic impact.[9–12] Secondary injury is often referred to as delayed injury, but usually begins within minutes of injury and can last several days to weeks afterward.[4,6,7,11,13–15] These categories may seem artificial at first, but are important when considering management.

Most TBI therapies are aimed at minimizing the effects of secondary injury. Because instability contributes to exacerbation of primary injury, depending on the type, management of acute SCI may include surgical therapy directed at stabilization to prevent further primary injury in addition to therapies directed at minimizing the effects of secondary injury.[15]

Primary Injury

Primary injury associated with TBI and SCI involves the physical disruption of intracranial structures (eg, TBI) and the spinal cord, vertebrae, and supporting structures (eg, SCI) that occurs at the time of impact. Primary injury is broadly classified as focal or diffuse depending on the extent of injury, and more specifically can be defined based on the location and type of injury.[11] The principal mechanical forces involved in neurologic trauma include concussion (eg, acceleration and deceleration), compression, shear, laceration, distraction, and contusion.[15–17] Primary injuries associated with TBI include epidural hematomas, subdural hematomas, subarachnoid hemorrhage, cortical contusions/hematomas, and traumatic axonal injury.[10] Primary SCI includes VFL, traumatic intervertebral disc herniation, intraparenchymal contusion, and extraaxial hemorrhage.

Secondary Injury

Box 1 summarizes the local factors contributing to secondary injury in neurologic trauma.[4,7,13,15–21] In addition, multiple systemic factors can potentiate secondary injury, most importantly hypoxia and hypotension but also hypercapnia, hypocapnia, hyperglycemia, hypoglycemia, acid-base disturbances, electrolyte abnormalities, hyperthermia, and systemic inflammation.[17,18] Other intracranial factors can also exacerbate secondary injury in TBI, including intracranial hypertension, edema, compromise of the blood-brain barrier (BBB), vasospasm, hemorrhage, infection, mass effects, and seizure activity.[17]

Secondary injury is potentiated by compromise of perfusion. Cerebral perfusion pressure (CPP) is defined as the net pressure facilitating blood flow to the brain, and is the difference between the mean arterial blood pressure (MAP) and intracranial pressure (ICP): CPP = MAP − ICP. Similarly, spinal cord perfusion pressure (SCPP) is the difference between MAP and cerebrospinal fluid pressure (CSFP): SCPP = MAP − CSFP.[22]

The Monroe-Kellie doctrine states that the cranial vault is a rigid, defined space that has a fixed volume with contributions from the brain parenchyma, cerebrospinal fluid (CSF), blood, and mass lesions (if present). An increase in the volume of any of these will result in a compensatory decrease in 1 or more of the others (defined as intracranial compliance; mainly reliant on changes in CSF or blood volumes), without which a pathologic increase in ICP will occur. With TBI the compensatory capacity of