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Brain injuries are common in dogs and cats, and can occur from many types of trauma, including motor vehicle accidents, falls, crush injuries, missile injuries, attacks from other animals, and inadvertent or purposeful attacks from humans. The prevalence and incidence of traumatic brain injuries (TBI) have not been studied in depth in veterinary medicine. However, in a recent study of brain injury, most dogs had suffered blunt vehicular trauma, while most cats were examined because of crush injuries. Management of brain trauma is complex and requires a basic knowledge of neuroanatomy, neurophysiology, and the indications and effects of the different medical and surgical therapeutic options. In addition, a global view of the patient is critical when treating head trauma patients and both, extracranial and intracranial priorities must be addressed. It is not uncommon in patients with TBI to find other life-threatening extracranial problems, such as penetrating thoracic and/or abdominal wounds, airway obstruction, or compromise of oxygenation, ventilation, or circulating volume problems that need to be identified and treated appropriately. Once the patient is systemically stable and the extracranial problems have been addressed, the clinician needs to focus on the intracranial priorities (maintenance of adequate cerebral perfusion, adequate brain oxygenation, and avoidance/treatment of increased intracranial pressure).

Primary injuries to the brain are a direct result of trauma and occur at the time of the traumatism. They include skull fractures, disruption of blood vessels causing hemorrhages, edema and parenchymal brain lesions directly caused by the traumatisms. The least severe brain injury is concussion, which is characterized by a brief loss of consciousness and it is not associated with any underlying histopathologic lesion. After regaining consciousness, some patients will exhibit mild disorientation for a short time. Contusion is the second lesion that can be seen, and it consists of parenchymal hemorrhage and edema. The clinical signs of contusions are variable and can range from mild to severe. Contusions can occur in the brain directly under the site of impact (“coup” lesions), and/or in the contralateral hemisphere (“contracoup” lesions) due to displacement of the brain within the skull causing impact on the opposite side. Mild contusion can be difficult to differentiate from concussion, but usually unconsciousness lasts for more than several minutes in the former. Lacerations are the most severe form of primary injury, and cause physical disruption of the brain parenchyma as well as significant hemorrhages. Hemorrhages can evolve into hematomas (intra- or extra-axial) that can cause compression of the brain, leading to severe localizing signs or diffuse neurological dysfunction. The current literature states that extra-axial hematomas are rare in dogs and cats suffering TBI. However, there is evidence that this type of injury occurs in 10% of animals with mild head injuries and up to 86% of dogs and 89% of cats with severe TBI. Although important, these injuries are rarely responsible for the death of the traumatized animal and are usually beyond the control of the clinician. Therefore, clinicians should focus on prevention, recognition and treatment of secondary brain injury.

Secondary brain injuries develop because of physiologic changes that occur as a result of the primary injury and lead to increased intracranial pressure (ICP). These changes develop over hours to days after the initial trauma and are induced by inflammatory mediators which are released after the initial hemorrhage and axonal injury. These secondary events occur simultaneously to perpetuate further and cause severe parenchymal damage, and subsequent increases in ICP, which can lead to permanent brain lesions. These events include enhanced activity of excitatory neurotransmitters, generation of reactive oxygen species (ROS), and production of proinflammatory cytokines, all of which contribute to neuronal cell damage and death. Cerebral edema formation, increased intracranial pressure (ICP), compromise to the blood-brain barrier (BBB), and alterations in cerebrovascular reactivity may ensue following secondary damage to brain tissue. These type of injuries are caused by a combination of...
intracranial and systemic insults that occur in both independent and interrelated ways. Treatment of head trauma is aimed at stopping or minimizing these secondary changes to protect the animal from the development and consequences of these secondary injuries.

Systemic disturbances that can contribute to secondary brain injury include hypotension, hypoxia, systemic inflammation, hypo- or hyperglycemia, hypo- or hypercapnia, hyperthermia, electrolyte imbalances and acid-base disturbances. Intracranial processes that participate and perpetuate secondary brain injury include: massive release of excitatory neurotransmitters (NT) causing cytotoxic edema, which leads to further release of excitatory NT, especially glutamate, causing massive intracellular calcium accumulation and cell death. In addition, local tissue acidosis and hypoperfusion triggers the production of ROS, which damage cell membranes. A third intracranial phenomenon associated with TBI is the release of inflammatory cytokines followed by the infiltration and accumulation of inflammatory cells, which incite nitric oxide (NO) production, leading to excessive vasodilatation and loss of pressure autoregulation in the damaged brain parenchyma. Lastly, increased ICP is a common and potentially deadly sequel of TBI. Severe increases in ICP trigger the cerebral ischemic response (Cushing reflex), which can be clinically recognized by systemic hypertension and reflex bradycardia. The central nervous system ischemic response in a patient with head trauma is a sign of potential life-threatening elevations in ICP, so it should be treated promptly.

The brain is contained in a non-distendible “box” (the skull), and is composed of three non-compressible elements: brain parenchyma, blood within blood vessels, and cerebrospinal fluid (CSF). Any increase in volume in any of these elements should be accompanied by a corresponding decrease in volume in one or both of the remaining in order to maintain a constant ICP. This is usually achieved through compensatory mechanisms (pressure autoregulation, chemical autoregulation) that allow redistribution of blood and CSF to allow more room within the skull for the swelling brain tissue. Pressure autoregulation is the ability to maintain a constant cerebral blood flow (CBF) during periods of hypotension or hypertension by means of altered vascular diameter and cerebrovascular reactivity. Chemical autoregulation is the response of the cerebral vasculature to changes in PaCO2, pH and O2 levels, calcium, potassium, and arachidonic acid metabolites. Cerebral vasodilation thus occurs in response to: decreases in mean arterial blood pressure (MABP), increases in PaCO2, and hypoxemia. Cerebral vasoconstriction occurs in response to increased MABP to decrease overall cerebral blood volume. In addition, cerebral ischemia triggers an increase in systemic blood pressure (Cushing’s reflex) in order to maintain normal cerebral perfusion pressure (CPP). When the compensatory mechanisms are exhausted or when there is a rapid increase in volume in one of the brain components, ICP increases (intracranial hypertension). The most detrimental effect of increased ICP is the reduction in cerebral blood flow (CBF), and thus cerebral ischemia. The final outcome of the ischemic insult is cerebral edema, which is a significant contributor to increased brain volume and ICP. Cytotoxic edema develops secondary to depletion of adenosine triphosphate synthesis, and the subsequent dysfunction of membrane transport mechanisms, which lead to accumulation of water and solutes within brain cells. Cytotoxic edema responds minimally to medical treatment. Vasogenic edema is clinically more significant and results from disruption of the blood-brain barrier, ischemia and release of vasoactive substances. Vasogenic edema is more responsive to medical therapy. Edema and ongoing hemorrhage can result in focally expanding masses, leading to further increases in ICP and development of brain herniations (displacement of brain tissue). Typical patterns include transtentorial, subfalcine, and foramen magnum herniations.

Assessment of patients with traumatic brain injuries should include a complete neurologic examination, with special attention to level of consciousness, posture and pupil size/response to light. The Modified Glasgow Coma Scale score is a quantitative measure that incorporates these three parameters and has been shown to be associated with 48 h survival in dogs with head trauma. In addition, and because of the likelihood of multi-systemic injury associated, patients with head trauma should be systemically stabilized and monitored. Emergency blood screening should consist of a packed cell volume and total solids to assess for hemorrhage, blood glucose to assess the severity of injury, and blood gases to assess ventilation, perfusion, and acid-base status. If possible, samples for complete blood cell count and blood biochemistry (including electrolytes and lactate) should be obtained prior to therapy in order to assess renal and hepatic function, as well as to screen for other systemic disturbances. In general, occlusion
of the jugular veins is contraindicated in patients with head trauma because of the likelihood of increasing ICP, so samples should be obtained peripherally. In general, patients with severe or progressing neurological signs despite aggressive systemic and intracranial stabilization therapy should undergo imaging of the head to try to localize potential surgically manageable lesions. In these cases, CT and MRI are the diagnostic imaging methods of choice.

Monitoring of patients with head trauma is essential for successful management. The overall duration and frequency of episodes of hypoperfusion and tissue oxygenation deficits have been associated with poorer outcomes in humans with TBI, so monitoring of these parameters is essential. Frequent qualitative assessment of tissue perfusion via mucous membrane color, capillary refill time, heart rate and pulse quality, as well as a quantitative assessment of blood pressure, oxygenation, and ventilation are crucial. MAP should be maintained at or above 80 mmHg in order to maintain adequate cerebral perfusion, and above 100 mmHg when measured with the Doppler technique (which measures systolic blood pressure). Continuous ECG monitoring should also be performed if possible to detect episodes of bradycardia, which would indicate increasing ICP (Cushing’s reflex), so special attention and measures should be taken when when systemic hypertension is present (MAP > 100 mmHg or systolic > 120 mmHg) and the ECG shows a low heart rate. The respiratory system should also be monitored, focusing on maintenance of oxygenation and ventilation. Oxygen saturation (measured via pulse oxymetry) should be maintained above 94%, while arterial oxygen tension (measured via arterial sample) should be above 80 mmHg. If oxygenation can not be monitored, oxygen supplementation is mandatory in head trauma patients. Ventilation can be assessed via end-tidal capnometry or by blood gas analysis, and carbon dioxide concentrations should be maintained within tight limits.

Management of patients with head trauma requires special attention to cerebral blood volume (CBV) and cerebral blood flow (CBF), since these are the ICP determinants that can be altered by the clinician. The main factors influencing CBV and CBF are:

**PaCO2.** Hypercapnia promotes vasodilation and hence increases CBF and ICP. Even mild hypercapnia can have a significant effect on intracranial pressure and therefore should not be tolerated. On the other hand, hyperventilation leading to hypocapnia induces vasoconstriction, which decreases CBF and leads to cerebral ischemia. Therefore manual or mechanical ventilation should be employed to maintain CO2 at the low end of the normal range in patients with head trauma (venous pCO2 40-45 mmHg, arterial pCO2 35-40 mmHg).

**MABP.** Pressure autoregulation may be lost in areas of brain trauma, ischemia, and increased ICP, causing CBF passively follow changes in MABP. The Cushing’s reflex occurs in response to increased ICP, causing increases in MABP to maintain adequate brain perfusion (CBF). In addition, patients with head trauma commonly present in hypovolemic shock, and volume resuscitation goals should be aggressive (MABP of 80-100 mmHg). Fluid therapy in the form of hypertonic and hyperosmotic solutions (colloids -hydroxyethylstarch, dextrans, gelatins-, isotonic crystalloids –0.9% saline solution-, hypertonic saline) which provide small-volume restoration of cardiac output and systemic arterial blood pressure should be used in these patients. In mild cases of head trauma, the administration of crystalloid fluids may be adequate.

**PaO2 and O2 content.** Due to chemical autoregulation phenomena, the cerebral vasculature dilates in response to decreases in PaO2 to ensure adequate brain oxygenation. In traumatized brain areas, this function may be lost and O2 content reduction (hemodilution, anemia) may lead to cerebral ischemia. Oxygen supplementation is always indicated in brain trauma patients. PaO2 should be maintained at a minimum of 80 mm Hg, since hypoxemia is one of the main causes of secondary brain injury.

**Cerebral metabolic rate (CMR).** Fever, pain, seizures and any condition that increases CMR cause increased CBF and increased ICP. Anticonvulsants (diazepam) should be administered to stop seizing activity, followed by phenobarbital to prevent development of more seizures in order to decrease CMR.

**Drugs.** Inhalant anesthetics cause cerebral vasodilation, thus increase ICP. Barbiturates decrease CMR, so they decrease ICP. On the other hand, barbiturates also cause systemic hypotension and hypoventilation, which can exacerbate increases in ICP.
Any brain trauma patient should be immediately evaluated for patency of airway, breathing and cardiovascular system. Resuscitation from shock should be performed and the patient evaluated for major body cavity injuries. Immobilization should be performed if vertebral fractures or instability are possible, and manipulations of the head and neck should be avoided, as well as jugular vein compression. Initial neurologic examination should include assessment of the state of consciousness, breathing pattern, brainstem reflexes such as size as responsiveness of pupils, ocular position and movements, cranial nerves, motor responses, responses to painful stimuli, posture, gait and postural reactions (in ambulatory animals). Serial neurologic examinations (every 2 hours) are essential to monitor progression and to be able to institute aggressive medical therapy when needed (increased ICP).

Based on the history and neurologic findings, diagnosis of brain trauma is straightforward. Advanced imaging of the brain (CT and MRI) is essential to determine the nature of the underlying lesion (skull fractures, intraparenchymal hemorrhage, extraparenchymal hematoma, edema, etc.), and if the lesion can be surgically treated. If a subdural hematoma is apparent on CT or MRI, craniotomy for decompression and evacuation of the hematoma is warranted. If available, intracranial pressure monitoring should be instituted. Continuous or serial monitoring of arterial blood gases and MABP (direct or indirect) is also essential to monitor progression and to institute adequate therapy if necessary.

The goal of treatment is to maintain brain tissue oxygen delivery, which requires maintenance of adequate circulating blood volume, systemic arterial blood pressure, and oxygen carrying capacity. Treatment of the traumatized brain patient includes supportive, special medical and surgical therapy measures.

In addition to the already described therapy, supportive therapy measures should include: Regular TPR, monitoring of arterial blood pressure (remember that hypotension is one of the main causes of secondary brain injury), blood gases, pulse oximetry, body temperature, heart rate and rhythm, and urine output. Head elevation, facilitates venous outflow from cerebral vasculature, and decreases ICP. Nutritional support (avoid hyperglycemia) is essential in the management of all patients with brain trauma, and has been shown to improve and hasten neurological recovery in human patients. In addition, jugular vein compression should be avoided in these patients. Recumbent patient care: physical therapy, frequent turning, eye lubrication, padded bedding, etc. Broad-spectrum antibiotics should be administered if there is concern about infection (open skull fractures, frontal sinus fractures, open wounds). The antibiotic of choice should have a good blood-brain barrier penetration.

Special medical therapy measures include: Hyperventilation (not always indicated). It is essential to maintain normoventilation (PaCO₂ 35 - 40mm Hg). Hyperosmolar treatment (mannitol): decreases blood viscosity, which improves cerebral perfusion and promotes cerebral vasoconstriction, thus decreasing ICP. Its hyperosmolar effect also draws water from the cerebral interstitium, which also lowers ICP. Its effect can be prolonged by the concomitant administration of colloidal fluids. Mannitol has an initial, transient effect which increases ICP. It should be administered as a bolus (1 g/kg iv over 15-20 minutes), thus continuous infusions and repeated doses can cause serum hyperosmolarity and promote fluid retention (serum osmolarity should be maintained ≤ 320 mOsm/l). If concomitant fluids are not administered to replace fluid loses, excessive diuresis and systemic hypovolemia may ensue mannitol administration. Glucocorticoids: there is a lot of controversy about the use of these drugs in head trauma patients. Their efficacy in these patients has not been proven, and administration of this type of drugs can have serious detrimental effects. Barbiturates: Pentobarbital-induced coma has been used in humans with refractory intracranial hypertension with positive results. By decreasing cerebral metabolic rate, they can also lower ICP. It is only recommended to use them as a last resort, since they also have negative effects (systemic hypotension, respiratory depression). In addition, adequate analgesia is essential in preventing further elevations in patients with head trauma. Opioids are commonly used analgesics due to their relative lack of adverse cardiovascular effects. However, opioids cause respiratory depression and hypotension, so they should be used with caution, specially in the presence of cardiovascular shock or BBB damage.
Lastly, surgical therapy for the head traumatized animal includes decompressive craniotomy, which is indicated when there are open skull fractures, depressed fractures, fractures involving blood vessels, subdural hematomas, or in patients with elevated ICP refractory to aggressive medical therapy.

References