TREATMENT OF HEAD AND SPINAL TRAUMA

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Introduction

Severe head and spinal injury are among the most challenging problems that present to the small animal practitioner. Often, neurologic injury is accompanied by other serious life-threatening problems including pneumothorax, pulmonary contusion, and traumatic hemoabdomen. Many of the animals have sustained multiple trauma and are in shock at the time of presentation. Head injury may be so severe that localization of head or spinal lesions and offering an accurate prognosis is difficult. The treatment of any patient with head and spinal injury involves making an accurate assessment to identify the extent of injury, prevention of further damage to the brain and spinal cord, and maintenance of cerebral perfusion pressure and end-organ function. Many cerebral injuries and spinal injuries with intact deep pain perception can carry an overall favorable prognosis with aggressive nursing care and tincture of time. Other types of injuries such as thoracolumbar spinal injury with loss of deep pain sensation are more clear-cut, but offer a less favorable prognosis.

Primary and Secondary Neuronal Injury

Injury to the brain and spinal cord involves primary injury that occurs at the time of impact, and secondary processes that occur as sequelae to the primary injury. The cascade of events and adverse sequelae that occur after the initial trauma are known collectively as secondary brain and spinal injury. At the time of impact, physical disruption and contusion of axons and blood vessels occur, and can lead to axonal laceration, the formation of subdural hematomas and subarachnoid hemorrhage. At the time of insult, vasogenic edema and hypovolemia can cause cerebral hypoxia and a depletion of ATP. Depletion of ATP results in inadequate cell membrane pump function, and an accumulation of intracellular calcium and sodium. Cerebral ischemia and disruption of axons ultimately results in the formation of oxygen-derived free radical species, nitric oxide, and elevated levels of the excitatory neurotransmitter glutamate that collectively perpetuate neuronal injury and the release of inflammatory cytokines. Cerebral edema can lead to massive increases in intracranial pressure if left untreated. In the spinal cord, secondary damage can result in ascending-descending myelomalacia and further loss of function with a much more grave overall prognosis. Rapid intervention to treat for shock, establishment and maintenance of cerebral blood flow and oxygen delivery, and recognition and treatment of cerebral edema are critical interventions to help increase the patient's chances for a favorable recovery.

Regulation of Cerebral Blood Flow and Oxygen Delivery

Cerebral blood flow is determined by cerebral perfusion pressure (CPP). The rate of blood flow is determined by the difference in pressure at the start and end of the blood vessel. Cerebral perfusion pressure is the difference between mean arterial pressure and intracranial pressure. Increases in intracranial pressure and decreases in mean arterial pressure during hypovolemic shock after trauma can both lead to a decrease in cerebral blood flow and cerebral oxygen delivery. Blood flow to the brain and maintenance of normal intracranial pressure are closely conserved when mean arterial blood pressure is between 50 – 150 mm Hg by a process known as "autoregulation". For example, if mean arterial blood pressure rises, vasoconstriction

of vascular beds will decrease cerebral blood flow and intracranial pressure remain normal. Conversely, if mean arterial blood pressure decreases, intracranial vasodilation occurs. The brain is also very sensitive to changes in carbon dioxide. As carbon dioxide levels rise (due to hypoventilation or pulmonary contusion, for example), cerebral blood vessels dilate. Therefore, hypercarbia can promote an increase in intracranial pressure in the severe multitrauma patient with concurrent pulmonary and cranial injury. Increases in cerebral blood flow, cerebral edema from secondary brain injury, intracranial hemorrhage, and decreased outflow of cerebrospinal fluid can all lead to elevations in intracranial pressure. The skull is a rigid box that is noncompliant, and cannot always accommodate the volume of its contents after severe head injury. Intracranial blood pressure autoregulation may not be conserved, leading to worsening cerebral hypoxia and the creation of a vicious cycle in which secondary brain injury and cerebral edema, left unchecked, can cause herniation of the brainstem through the foramen magnum. Systemic blood pressure will continue to rise in an attempt to compensate for the decrease in cerebral perfusion. Reflex bradycardia will occur. A sudden increase in systemic blood pressure and bradycardia in a traumatically head injured patient is known as Cushing's Reflex, and is strongly supportive of brainstem herniation.

Triage and Initial Stabilization of the Head/Spinal Trauma Patient

The assessment and treatment approach for any patient that has sustained severe trauma should be based on the ABC's, or "airway, breathing, and circulation". Examine the patient for a patent airway. Note the patient's respiratory status and listen carefully to thorax. The absence of lung sounds with rapid restrictive respiration may be associated with a pneumothorax or diaphragmatic hernia. Harsh crackles with cyanosis and respiratory difficulty can be associated with pulmonary contusions. Tachycardia can be associated with pain, anxiety, and hypovolemic shock. The patient's blood pressure should be measured, and intravenous fluids should be administered to restore circulating blood volume and cerebral perfusion pressure. In the past, permissive hypotension was used in head trauma patients, with the assumption that the administration of large volumes of crystalloid or colloid fluids can leak into the brain and contribute to cerebral edema and intracranial hemorrhage. More recently it has been documented that maintenance of cerebral perfusion pressure and cerebral oxygen delivery is of paramount importance in any head trauma patient. Intravenous crystalloid and colloid fluids should be titrated based on the patient's arterial blood pressure and response to treatment. Careful administration of intravenous crystalloid fluids at ¼ of the calculated shock volume of fluids (shock volume is 90 ml/kg in dogs and 44 ml/kg in cats) should be administered as rapidly as possible with careful and frequent reassessment of the patient's blood pressure. Alternatively, 5 ml/kg of hetastarch or dextran-70 can also be administered as a rapid IV bolus. The goal is to restore systolic blood pressure to 90 – 100 mm Hg, diastolic blood pressure to > 40 mm Hg, and mean arterial blood pressure to 60 – 80 mm Hg. This approach serves to restore cerebral blood flow and flow to vital organs such as the heart and kidneys, as well. Hypertonic saline (percent, volume to be administered) has also been used in combination with dextran-70 in the successful treatment of head trauma. The potent oncotic effect of IV hypertonic saline acts to pull fluid from the intracellular and interstitial fluid compartments into the intravascular space within 3 minutes of administration. The effect is very short-lived, and lasts approximately 20 – 30 minutes. When used in combination with a colloid such as Dextran-70, the fluid is retained in the intravascular space for a longer period of time. Hypertonic saline (7.5%, 3 - 5 ml/kg for dogs, and 2 – 4 ml/kg in cats hypertonic saline with 5 – 10 ml/kg colloid) has been shown to restore

extracellular sodium concentrations and decrease neutrophil chemotaxis to limit secondary brain injury in the head trauma patient. The patient's oxygenation status should be monitored closely with pulse oximetry or arterial blood gas analyses. Ideally, the patient's arterial partial pressure of oxygen (PaO2) should be maintained above 80 mm Hg, and oxygen saturation as measured by pulse oximetry above 90% SaO2. Supplemental oxygen should be administered by mask, hood, or flow-by in any patient with head trauma. The placement of nasal oxygen catheter(s) can result in patient discomfort and subsequent sneezing. Sneezing can increase intracranial pressure, so nasal oxygen catheters should be avoided. A minimum data base of PCV/TS, glucose, azostick, and lactate can be useful for baseline measurements and may help predict outcome.

Hyperglycemia has been shown to be a negative prognostic indicator in humans and dogs with severe head injury. Glucose acts as a substrate for anaerobic metabolism during periods of cerebral ischemia, and can lead to cerebral acidosis. For this reason, the administration of glucose-containing fluids (D5W, 0.45% NaCl + 2.5% dextrose) and ANY STEROID is contraindicated in the head trauma patient. Steroid use has not been documented to provide any benefit in traumatic brain injury, and can cause hyperglycemia that can potentially worsen secondary brain injury and cerebral edema. One exception to the use of steroids in head injury are those patients with severe facial, oropharygeal, and ocular trauma in which steroid use is necessary to decrease edema to maintain a patent airway.

Any trauma patient that exhibits abnormal neurologic postures should be confined to a backboard for stabilization and to prevent further injury during the initial triage period. Extensor rigidity of the forelimbs with flaccid paralysis of the hindlimbs is characteristic of Shiff-Sherrington posture, and is commonly associated with an injury between T3 and L3. The loss of deep pain perception in such patients carries a grave prognosis. Intact deep pain perception with Schiff-Sherrington has been associated with spinal shock and not necessarily due to transaction of the spinal cord, and can carry a slightly more favorable prognosis, depending on the exact location and extent of the injury. Flaccid paralysis of the forelimbs with the hindlimbs tucked up close to the body and opisthotonus is associated with severe injury to the cerebellum, and is known as decerebellate rigidity. Extensor rigidity of all four limbs and opisthotonus is known as decerebrate rigidity, and carries a very grave prognosis.

Neurologic Examination

Once the patient's ABC's have been accurately assessed and problems addressed. A complete neurologic examination can be performed. Pupil size and response to light, presence or absence of menace, physiologic nystagmus, mentation, ambulation, and reflexes should be evaluated in a step-by-step approach, starting from nose to tail. Mental status is often difficult to accurately assess until hypovolemic shock has been successfully treated. A patient's mentation can be categorized as normal, depressed, obtunded, stuporous, or comatose. A depressed patient may appear mentally dull and be slow to react to external stimuli including noise and touch. As mental status worsens, an obtunded patient will be depressed, and slower to respond to external stimuli. A stuporous patient appears unconscious, but will respond to painful or noxious stimuli. Coma is the most severe change in mentation, in which the patient is unconscious and completely unresponsive to a noxious stimulus. The presence of coma alone does not necessarily mean a poor long-term prognosis. The patient's mentation should be evaluated in combination with the patient's pupil size when gauging severity of condition in order to make a prognosis. Miotic pupils are associated with forebrain lesions. Mydriatic dilated pupils or loss of papillary light reflexes are associated with a rostral brainstem lesion, and carry a much more

guarded prognosis when in combination with stupor or coma. Anisocoria, or unequal pupil size, can be associated with either intracranial lesions, or extracranial lesions of the eye, brachial plexus, or vagosympathetic trunk. For example, a patient with normal mentation, miosis, and anisocoria can potentially have anterior uveitis secondary to a corneal abrasion, brachial plexus injury, or injury to the lateral neck affecting the vagosympathetic trunk on the side ipsilateral to the miotic pupil, with no intracranial lesion at all. Animals with Schiff-Scherrington posture should be placed on a flat backboard or other flat surface and strapped down to prevent movement and potential disruption of a partially displaced spinal fracture/luxation. Procedures to assess balance and muscle strength such as hopping and wheelbarrowing should not be performed until spinal trauma has been completely ruled out. Reflexes and deep pain perception should be evaluated. Withdrawal of the hind limbs in a patient with Schiff-Scherrington posture is a local reflex arc only, and should not be interpreted as perception of pain and functioning motor and sensory tracts through the spinal cord to the cerebral cortex. Additionally, if the patient attempts to move during the evaluation, motion at the fracture site can be perceived as painful without functioning sensory pathways to the rear limbs. Less than 5 – 8% of animals with loss of deep pain perception regain motor function and continence, and thus, prognosis for return to function must be considered poor, at best.

Radiographs should be performed if depressed skull or spinal fractures are suspected. Patients with suspected spinal fractures should never be moved from lateral position in order to take dorsoventral or ventrodorsal radiographs. A lateral radiograph of the suspected fracture site should be performed. In many cases, disruption of the articular facets, compression fractures, or obvious disruption of the spinal column can be visualized. Some injuries, however, may be difficult to accurately assess without an orthogonal view. Rather than move the patient and potentially cause further disruption and injury to the fracture, a ventrodorsal radiograph view can be obtained by turning the bucky at a 90 degree angle and placing an x-ray cassette behind the patient. Radiographs are a sensitive imaging modality for diagnosis of intracranial hemorrhage or edema. The use of computed tomography (CT) and magnetic resonance imaging (MRI) are more sensitive at detecting intracranial lesions. An MRI is considered to be the best imaging modality for detection of fibrocartilagenous emboli (FCE).

Treatment of Head and Spinal Trauma

The various forms of recommended treatment for head and spinal trauma remains a subject of wide debate and controversy. The treatment of severe head trauma in a patient that is obtunded, stuporous, or in a coma involves maintenance of cerebral perfusion and oxygen delivery and decreasing cerebral edema. Mannitol (0.5 – 1.0 g/kg IV over 10 - 15 minute) acts both as an osmotic diuretic and free radical scavenger in the patient with traumatic brain injury, and serves to decrease cerebral edema and secondary brain injury after the time of impact. The use of mannitol had fallen out of favor in the past because of the potential risk of worsening intracranial hemorrhage. The benefits of mannitol far outweigh the unsubstantiated risks, particularly in the traumatically head injured patient. Mannitol administration is not necessary in patients that are normal or depressed with an obvious skin abrasion or laceration on the face or head. This author reserves mannitol use to the patient with traumatic head injury that is obtunded, stuporous, or comatose, or exhibits a decline in mental status despite aggressive treatment to restore and maintain intravascular volume and normal blood pressure.

Steroid use has dramatically fallen out of favor for the treatment of traumatic brain injury. Glucocorticosteroids were thought to stabilize neuronal membranes. However, steroids

also decrease immune defense mechanisms, disrupt glucose homeostasis, contribute to negative nitrogen balance and insulin resistance, and can worsen intracranial acidosis. Additionally, steroid use has demonstrated equivocal results. The benefits of steroid use are unsubstantiated, and are far outweighed by their risks, and as such, are contraindicated at this time. The use of methylprednisolone sodium succinate (MPSS) is now considered the gold standard of care in spinal trauma (30 mg/kg IV once, then 15 mg/kg IV 2 and 4 - 6 hours later). The time frame for reported benefits in human patients is administering the MPSS within 8 hours of the traumatic event. After this time frame, there has been no documented benefit of steroid use in helping improve prognosis for return to function. In fact, a closer look at the literature has demonstrated that steroid use, in general, does not actually improve overall patient outcome with return to a relatively normal function. Steroid use is also associated with an increased risk of gastrointestinal ulceration. For these reasons, controversy exists whether steroids should be administered at all to patients with spinal trauma and intervertebral disk disease.

New and promising research has documented the use of intravenous polyethylene glycol (2 mg/kg IV at 0 and 6 hours) to spinal trauma patients repairs axonal membranes and decrease secondary oxidative injury to neuronal membranes. To date, no adverse consequences have been documented in clinical cases. Preliminary studies have demonstrated that the use of PEG dramatically hastens recovery in experimental dogs and clinical cases with spinal injury. Further research must be conducted to determine whether the compound shows promise for use in traumatic brain injured patients, as well.

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