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Medical Neurology for Advanced Practitioners Mini Series

Session One: Neurological Trauma - Fixing the Broken in a Practical Manner

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TRAUMA TO THE NERVOUS SYSTEM

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HEAD TRAUMA - INTRODUCTION

Severe head trauma is associated with high mortality in human beings and animals. Although there is no standard of care for head trauma in human medicine, a series of guidelines have been developed centered around maintaining adequate cerebral perfusion. The appropriate therapy for head trauma patients remains controversial in veterinary medicine due to a lack of objective information on the treatment of dogs and cats with head injuries. Treatment of affected animals must be immediate if the animal is to recover to a level that is both functional and acceptable to the owner. Many dogs and cats can recover from severe brain injuries if systemic and neurological abnormalities that can be treated are identified early enough.

PRIMARY PATIENT ASSESSMENT

As with all types of acute injury, the “ABCs” (airway, breathing, cardiovascular status) of emergency care are extremely important. Initial physical assessment of the severely brain-injured patient focuses on imminently life threatening abnormalities. It is important not to focus initially on the patient’s neurological status as many patients will be in a state of hypovolemic shock following a head injury, which can exacerbate a depressed mentation. Hypovolemia and hypoxemia need to be recognized and addressed immediately. In addition, a minimum essential data-base includes a PCV, total protein level, a blood urea level, and electrolyte levels as well as a urine specific gravity. Specific attention should also be paid to the serum glucose levels as hyperglycemia has been demonstrated to be related to head trauma severity, although unlike in humans, a specific association with outcome has not yet been demonstrated. Respiratory system dysfunction can be common after head injury. The most dramatic respiratory abnormality seen following head injury can be neurogenic pulmonary edema. Neurogenic pulmonary edema is usually self-limiting if the patient survives, and will resolve in a matter of hours to days, but can cause severe dyspnea, tachypnea and hypoxemia. Hypoxemia exacerbates the development of secondary tissue damage. There is no proven clinical localizing value to specific breathing patterns in veterinary medicine and these patterns may vary over time.

SECONDARY PATIENT ASSESSMENT

Once normovolemia and appropriate oxygenation and ventilation are established (see below), the patient should be thoroughly assessed for traumatic injuries. These include skull, vertebral and long bone fractures as well as splenic torsions and ruptured bladder and ureters. The neurological examination, cranial imaging and ICP measurement can then be considered.

Neurological Assessment

Neurological assessment should be repeated every 30 to 60 minutes in severely head injured patients to assess the patient for deterioration or to monitor the efficacy of any therapies administered. This requires an objective mechanism to ‘score’ the patient so that treatment decisions could be made logically.

The Modified Glasgow Coma Scoring System

In humans, traumatic brain injury is graded as mild, moderate or severe on the basis of an objective scoring system, the Glasgow coma scale (GCS). A modification of the GCS has been proposed for use in veterinary medicine and evaluated in relation to 48 hour survival. The scoring system enables grading of the initial neurological status and serial monitoring of the patient. Such a system can facilitate assessment of prognosis, which is crucial information for both the veterinarian and owner. The modified scoring system incorporates 3 categories of the examination (i.e., level of consciousness, motor activity, brainstem reflexes), which are assigned a score from 1 to 6 providing a total score of 3 to 18, with the best prognosis being the higher score. Scores of less than 8 indicate a less than 50% probability of 48 hour survival.

MEDICAL THERAPY

1. Minimizing increases in ICP

Simple precautions can be taken in positioning the animal with its head elevated at a 30° angle from the horizontal to maximize arterial supply to and venous drainage from the brain. It is also important to ensure that there is no constrictive collar obstructing the jugular veins as this immediately elevates ICP.

2. Fluid therapy
The basic goal of fluid management of head trauma cases is to maintain a normovolemic to slightly hypervolemic state. There is no support for attempting to dehydrate the patient in an attempt to reduce cerebral edema and this is now recognized to be deleterious to cerebral metabolism. In contrast immediate restoration of blood volume is imperative to ensure normotension and adequate CPP. Initial resuscitation usually involves intravenous administration of hypertonic saline and or synthetic colloids. Use of these solutions allows rapid restoration of blood volume and pressure while limiting volume of fluid administered. In contrast, crystalloids will extravasate into the interstitium within an hour of administration and thus larger volumes are required for restoration of blood volume. As a result this could lead to exacerbation of edema in head trauma patient. Hypertonic saline administration (4-5 ml/kg over 3-5 minutes) draws fluid from the interstitial and intracellular spaces into the intravascular space which improves blood pressure and cerebral blood pressure and flow, with a subsequent decrease in intracranial pressure. However, this should be avoided in presence of systemic dehydration or hypernatremia and it should be noted that the effects of this fluid only last up to an hour. Colloid solutions, such as Dextran-70 or Hetastarch should be administered after hypertonic saline is used, to maintain the intravascular volume. Hypertonic solutions act to dehydrate the tissues, thus it is essential that crystalloid solutions are also administered after administration of HSS to ensure dehydration does not occur. The sole use of colloids will not prevent dehydration; in addition, the co-administration of hypertonic solutions and colloids are more effective at restoring blood volume than either alone.

3. Osmotic diuretics
Osmotic diuretics such as mannitol are very useful in the treatment of intracranial hypertension. Mannitol has an immediate plasma expanding effect that reduces blood viscosity, and increases cerebral blood flow and oxygen delivery. This results in vasoconstriction within a few minutes causing an almost immediate decrease in ICP. The better known osmotic effect of mannitol reverses the blood-brain osmotic gradient, thereby reducing extracellular fluid volume in both normal and damaged brain. Mannitol should be administered as a bolus over a 15 -minute period, rather than as an infusion in order to obtain the plasma expanding effect; its effect on decreasing brain edema takes approximately 15-30 minutes to establish and lasts between 2 and 5 hours. Administering doses of 0.25 g/kg appear equally effective in lowering ICP as doses as large as 1.0 g/kg, but may last a shorter time. Repeated administration of mannitol can cause an accompanying diuresis, which may result in volume contraction, intracellular dehydration and the concomitant risk of hypotension and ischemia. It is therefore recommended that mannitol use is reserved for the critical patient (Glasgow coma score of < 8) or the deteriorating patient. There has been no clinical evidence to prove the theory that mannitol is contraindicated in the presence of intracranial hemorrhage. There is contradictory evidence that the combination of mannitol with furosemide (0.7 mg/kg) may lower ICP in a synergistic fashion, especially if furosemide is given first.

4. Oxygenation and ventilation
Hyperoxygenation is recommended for most acutely brain-injured animals. Partial pressure of oxygen in the arterial blood (PaO_2) should be maintained as close to normal as possible (at or above 80 mm Hg). Supplemental oxygen should be administered initially via face-mask as oxygen cages are usually ineffective as constant monitoring of the patient does not allow for a closed system. As soon as possible, nasal oxygen catheters or transtracheal oxygen catheters should be used to supply a 40% inspired oxygen concentration with flow rates of 100 ml / kg / min and 50 ml / kg / min respectively. If the patient is in a coma, immediate intubation and ventilation may be needed if blood gas evaluations indicate. A tracheostomy tube may be warranted in some patients for assisted ventilation. Hyperventilation has traditionally been known as a means of lowering abnormally high ICP through a hypocapnic cerebral vasoconstrictive effect. However, hyperventilation is a double-edged sword. Besides reducing the ICP, it induces potentially detrimental reductions in the cerebral circulations if the pCO_2 level is less than 30-35 mmHG. The major difficulty with hyperventilation is our present inability to monitor the presence and effects of ischemia on the brain. It is important that animals do not hypoventilate, and such animals should be ventilated to maintain a $PaCO_2$ of 30-40mmHg. Aggressive hyperventilation can be used for short periods in deteriorating or critical animals.

SURGICAL THERAPY
A description of the surgical techniques for intracranial surgery can be found elsewhere. Although it is rare that surgery is indicated in head injury cases, there are several specific abnormalities that can be associated with an episode of head trauma that may warrant the consideration of surgical treatment:

SPINAL TRAUMA
Acute spinal cord injury

Acute onset of non-ambulatory para-, hemi- or tetraparesis should be considered an emergency.

Etiology
The most common causes of acute spinal cord injury include acute intervertebral disc herniation (both Hansen type I and acute noncompressive nucleus pulposus extrusions, vertebral fractures and luxations, vascular disease (e.g. fibrocartilaginous embolism (FCE) and haemorrhage), and congenital malformation causing instability (e.g. atlantoaxial subluxation). Many chronic diseases, such as cervical spondylomyelopathy (Wobbler syndrome), neoplasia, discospondylitis and inflammatory or infectious spinal cord diseases, can also present acutely as a result of clinical decompensation or of the sudden development of associated pathology (i.e. vertebral fractures due to vertebral neoplasia or discospondylitis, intraparenchymal hemorrhage due to hemangiosarcoma and vasculitis).

Pathophysiology
Acute onset of spinal cord dysfunction is most commonly caused by a combination of one or more events including contusion, compression, ischemia, or laceration of the spinal cord.

Contusion
Contusion of the spinal cord is commonly caused by intervertebral disc extrusion, as well as vertebral fractures and luxations. Repeated contusion may occur in some diseases due to vertebral instability. Acute contusion of the spinal cord initiates a series of biochemical and metabolic events that expand the primary zone of tissue necrosis. The majority of this secondary damage occurs within 24 hours of injury and, although cellular apoptosis continues for weeks to months it is not common for clinical signs of deterioration to be evident much beyond 72 hours after the injury. The detrimental events are initiated by the initial mechanical insult, which causes release of neurotransmitters, damage to glial and neuronal cell membranes and damage to local vasculature. This causes energy failure and increased cell membrane permeability, and leads to a cascade of events, including destruction of the microvascular bed, leading to a progressive reduction in perfusion of the injured area, an increase in intracellular calcium concentrations, and free-radical production. Many of these factors interact to produce a cycle of destructive events. The end result is an expanding zone of cellular necrosis and apoptosis.

Primary assessment
On admission, the first aim is to stabilize the patient by assessing the airway, breathing and circulation (‘ABC’) and treating abnormalities where necessary as for any other emergency. Complete routine blood work and urinalysis should be obtained if possible; otherwise a packed cell volume (PCV), total protein level, blood urea nitrogen (BUN) assessment and glucose and electrolyte levels should be ascertained. Cardiovascular stability should be investigated with the aid of an electrocardiogram (ECG) and blood pressure measurements. The important systemic parameters to monitor, their suggested reference values after trauma and management protocols are similar to those following head trauma and are detailed later.

Secondary assessment
A thorough physical and orthopedic examination can follow the initial patient evaluation and stabilization. Consideration should also be given to obtaining a coagulation panel, a buccal mucosal bleeding time and a platelet count if there has been associated hemorrhage. A patient that has experienced blood loss, or that is expected to do so during surgery, should be blood-typed or cross-matched and appropriate blood products should be obtained.

Neurological assessment
The neurological examination should be aimed at localizing the lesion and determining its severity. Thoracolumbar spinal cord injury severity is commonly graded as follows:

0 – Normal
1 – Painful
2 – Conscious proprioceptive deficits, ataxia and paraparesis
3 – Non-ambulatory paraparesis
4 – Paraplegia with nociception intact
5 – Paraplegia with loss of nociception.

Lack of nociception is not as relevant in tetraparesis, however, special attention should be paid to the respiratory rate and pattern in any non-ambulatory tetraparetic or tetraplegic patient with a view to detecting hypoventilation. The presence of hypoventilation indicates the need for ventilatory support and immediate diagnostic workup.

Diagnostic imaging

Survey radiography: Thoracic radiographs should be evaluated after a significant trauma, looking for pleural effusions, contusions, pneumo-mediastinum and pneumothorax as well as the possibility of pericardial effusion and diaphragmatic herniation. If a vertebral injury is suspected, it is recommended that survey lateral radiographs are taken of the entire spine prior to additional manipulation of the animal. Sites particularly predisposed to fracture and luxation include the atlantoaxial junction, the thoracolumbar junction and the lumbar and lumbosacral spine. As some fractures can be subtle, good quality and well positioned radiographs from orthogonal planes are necessary. This may be accomplished with the animal awake and immobilized; analgesia may be required. Poor radiographic technique resulting in rotation of the spine (especially in the cervical area) can make assessment for unstable and malaligned vertebral segments difficult. Extreme care should be taken in positioning the animal for ventrodorsal views: horizontal beam radiographs can be obtained if the equipment is available. Sedation may be necessary to achieve accurate positioning for radiography in some animals. This should not be performed if the examiner is unsure of the physical diagnosis, as sedation often influences the results of the neurological examination. Additionally, sedation or anesthesia results in the loss of voluntary paraspinal muscle contraction, and unstable vertebral segments may be more likely to subluxate. It is important to remember that radiographs provide a static record of the location of the vertebrae at the time of the study but they do not allow for assessment of how extensive the displacement of the vertebrae was at the time of the injury and prior to radiology. As a result of the strong paraspinal musculature, vertebrae can be significantly displaced acutely at the time of injury but then subsequently pulled back into a more normal position. A scheme has been devised for predicting spinal instability in human patients based upon the degree of vertebral damage, which has been modified for use in animals. In this model the vertebrae are divided into three compartments. Damage to more than one compartment indicates a need for internal or external stabilization.

Advanced imaging: Myelography or other advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) is needed to evaluate spinal cord compression and to ensure that additional lesions unidentifiable on survey radiographs are not present. There is a mounting body of evidence that MRI can provide prognostic information with acute intervertebral disc herniation and it can identify complete physical disruption of the spinal cord. CT is invaluable in identifying bone defects that may not be apparent on survey radiography. Three-dimensional reconstruction from CT images may provide additional anatomical information regarding bone contour for surgical planning.
MRI has the distinct advantage of showing spinal cord parenchyma and soft tissue injuries as well as multiplanar images but does not provide as good bone detail as CT studies, although proton density and STIR images can improve the image quality of bone.

Urinary tract assessment: In addition to blood urea and creatinine values and urine specific gravity levels, urethral catheterization may be required in patients that have undergone a severe concurrent abdominal or pelvic trauma in order that urine production may be assessed over the subsequent 72 hours. This will also be of value in those patients with systemic shock due to the traumatic event. Abdominal ultrasonography may be required to evaluate the bladder wall and kidneys, and to detect the presence of free abdominal fluid; contrast-enhanced imaging of the urinary tract may provide further information on the function and form of the individual structures.

Prognosis

Factors influencing the prognosis of the acutely paralyzed animal include the nature of the underlying disease, the severity of the neurological signs, the duration of the signs and the owner's resources.

Severity of neurological signs

Nociception in affected limbs is the single most important prognostic indicator of injury severity that can be obtained from the neurological examination.

Nociception is tested for by applying heavy pressure to the bones of the digits with large haemostatic forceps or heavy pliers such that pressure is applied to the underlying periosteum. Nociception is present if the animal displays a conscious awareness of the stimulus rather than simply a reflex withdrawal of the limb. Typically, both medial and lateral digits of each paralysed limb are tested and the tail is tested. Lack of nociception implies *functional* spinal cord or peripheral nerve transection at the time of testing and provides useful prognostic information. As a general rule, animals with intact nociception have the potential to recover motor function if the underlying disease process can be prevented from progressing.
The prognosis for paraplegic animals that lack nociception in their pelvic limbs varies with the cause and is generally worse with longer duration of the signs.

It is unusual to be presented with a tetraplegic animal that lacks nociception as functional cervical spinal cord transection causes paralysis of the respiratory muscles. In addition, the sympathetic nervous system is interrupted as it runs down the cervical spinal cord, resulting in pronounced bradycardia. As a result, animals with extremely severe cervical spinal cord injuries die rapidly of hypoventilation and cardiac arrest.

GUIDELINES FOR THE TREATMENT OF SPINAL CORD INJURY

Medical therapy

Medical treatment of acute spinal cord contusion and ischemia is aimed at limiting the final extent of secondary tissue damage. Minimizing secondary injury is generally achieved by ensuring adequate perfusion and oxygenation of the animal. The Holy Grail of CNS trauma is to identify an effective neuroprotective agent that could be given in the acute phase of injury, but this has proven elusive making it all the more important to focus on maintenance of perfusion and oxygenation. Treatment must be initiated as soon as possible after injury, as the majority of secondary tissue damage occurs within 24 hours of the primary injury.

1. Corticosteroids*:* MPSS is used for its free radical scavenging properties rather than its anti-inflammatory effect. However, many question the validity of the results of the NASCIS Trials and the use of MPSS remains controversial in human medicine. MPSS use has been reported in experimental models of canine spinal cord injury and a benefit has not been seen although both studies are underpowered. Suggested MPSS protocols have been taken from human medicine. A therapeutic window of only 8 hours has been identified as delaying initiation of treatment for more than 8 hours may have a detrimental effect on outcome in people. As MPSS has both glucocorticoid and free-radical scavenging effects, it is postulated that delaying treatment until after the majority of free-radical-induced damage has occurred is more likely to result in glucocorticoid-induced side-effects. Indeed, although there continues to be widespread use of glucocorticoids such as dexamethasone to treat acute spinal cord injuries in veterinary practice, neither experimental studies nor retrospective analyses of clinical cases have shown benefit and the side-effects have been well documented.

2. **Polyethylene glycol:** Polyethylene glycol (PEG) has been advocated as an effective therapy of canine spinal cord injury and has been evaluated in a pilot clinical trial in dogs with acute disc herniations that suffered the most severe grade of injury (paraplegic with loss of nociception) (Laverty et al, 2004). This polymer has surfactant properties and targets and fuses damaged membranes following intravenous administration, thus interrupting the injury cascade. The treatment protocol is as follows: 3500 Da PEG as a 30% w/w solution in sterile saline administered at a dose rate of 2ml/kg as soon as possible after injury and repeated once 4 – 6 hours later. The published study did not encounter any adverse effects and reported recovery of ambulation in 13 of 19 PEG treated dogs (68%). This is comparable to other reports of recovery of ambulation in this population of dogs when treated with surgical decompression alone and there is currently no approved medical grade preparation of this compound.

Non-surgical management of spinal fractures

Non-surgical treatment of spinal fractures and luxations is dependent on whether the injury is determined to be unstable based on the three-compartment model. If there is no instability, cage rest for 6 weeks is adequate. If the fracture/luxation is determined to be unstable and owners decline surgery, an external splint can be placed in such a manner that the damaged area of the spine is immobilized; if the animal has significant skin, abdominal or thoracic injuries, splint placement may be contraindicated, in which case the animal should be strictly cage rested for 6 weeks.