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TREATMENT OPTIONS FOR HEAD TRAUMA PATIENTS

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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 centred 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 (intracranial pressure) measurement can then be considered.

Neurological assessment

Neurological assessment should be repeated every 30-60 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 72-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 72-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 (cerebral perfusion pressure). 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 solution (HSS) 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 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. Arterial blood pressure support

Presence of arterial hypotension despite fluid resuscitation (see above) may require administration of vaso-active agents such as dopamine (2-10 µg/kg/min). Conversely, arterial hypertensive episodes ("Cushing's response") may be managed with calcium channel blockers such as amlodipine (0.625 to 1.25 mg/cat every 24 hours; 0.5 to 1.0 mg/kg in dogs every 24 hours). However, the author recommends treating the increased ICP aggressively before using drugs to assist blood pressure regulation.

5. Oxygenation and ventilation

Hyperoxygenation is recommended for most acutely brain-injured animals. Partial pressure of oxygen in the arterial blood (PaO₂) 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 pCO₂ level is less than 30-35 mmHg. The major difficulty with hyperventilation is our present inability to monitor the presence and effects of ischaemia on the brain. It is important that animals do not hypoventilate, and such animals should be ventilated to maintain a PaCO₂ of 30-40 mmHg. Aggressive hyperventilation can be used for short periods in deteriorating or critical animals.

6. Seizure prophylaxis

Although the role of prophylactic anticonvulsants in preventing post-traumatic epileptic disorders remains unclear, seizure activity greatly exacerbates intracranial hypertension in the head injury patient. For this reason, it is recommended to treat all seizure activity in these patients aggressively but not prophylactically. As most cases need to be treated parenterally, phenobarbital (2 mg/kg IM q 6-8hrs) is recommended. This can be continued for 3-6 months after the trauma and can then be slowly tapered off if there have been no further seizures. Phenobarbital will have the additional benefit of reducing cerebral metabolic demands and therefore



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acts as a cerebral protectant.

7. Corticosteroids

Corticosteroids, known to be beneficial in brain edema attributed to a tumor, have been studied extensively in head injury. Clinical trials in people have not shown a beneficial effect of corticosteroids, including methylprednisolone sodium succinate, in the treatment of head injury. In fact, they are now contraindicated based on an increased incidence of mortality following their use. In addition, they have been associated with increased risks of infection, are immunosuppressive, cause hyperglycemia and other significant effects on metabolism.

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:

Acute extra-axial hematomas

Generous craniotomies are generally indicated once these abnormalities have been diagnosed with imaging. If the hematoma is due to a fracture across a venous sinus, there may be profuse bleeding associated with surgical intervention. The need for blood transfusions should be expected. Hematoma removal also risks the chance of bleeding from previously compressed vessels.

Calvarial fractures

A skull fracture *per se* may or may not have significant implications for patient management. Skull fractures are

typically differentiated based upon pattern (depressed, comminuted, linear), location and type (open, closed). A fracture is generally classed as depressed if the inner table of the bone is driven in, to a depth equivalent to the width of the skull. All but the most contaminated, comminuted and cosmetically deforming depressed fractures can be managed without operative intervention.

Acute intraparenchymal hematoma

In contrast to acute extra-axial hematomas, acute intraparenchymal clots may be conservatively managed, unless subacute enlargement of initially small intraparenchymal clots is identified with repeat MR scanning.

Hemorrhagic parenchymal contusions

Most hemorrhagic contusions do not require surgical management. The main indication for surgery with these types of lesions is limited to cerebellar contusions with compression of the 4th ventricle and brain stem; surgery aims to reduce the potential for further compression and herniation, which can develop over the initial 24-48 hours.

Intracranial hypertension (ICH)

Benefit can be found when decompressive procedures are carried out before irreversible bilateral papillary dilation has developed. Conversely, 'prophylactic' decompressive surgery seems inappropriate before non-surgical management of elevated ICH has been carefully maximized.