Approach to Head Trauma
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Head trauma patients can look terrible on initial presentation but often do well with supportive care. For dogs experiencing severe blunt trauma, roughly 25% will have head trauma. Mortality rates for head trauma in dogs range from 18-24%. Motor vehicle accidents are the most common cause and account for approximately 50% of head trauma cases in dogs and cats. Other causes include falling, bite wounds, gunshot wounds, and other traumatic incidents. Traumatic brain injury (TBI) occurs when patients have neurologic deficits such as altered mentation, seizures, loss of consciousness, etc. Rapid recognition and response are required to give our patients the best chance. Thankfully, our patients compensate remarkably well to head injuries. Even those severely affected can recover with appropriate supportive care. The prognosis should not be assessed until the patient is fully stabilized. Improvement is often seen in the first 48-72 hours, but brain injuries can take weeks to recover.

Patient Evaluation

Like with all emergencies, triage (the ABCs) is key. Polytrauma is common, so a full evaluation is important. Neurologic assessment should be performed after resuscitation as shock muddies the water (e.g., alters the mentation). Assessment of the cardiovascular system should focus on the perfusion parameters (3 cranial: mentation, mucous membrane color, capillary refill time; 3 caudal: pulse rate, pulse quality, and relative distal extremity temperature). Respiratory rate, respiratory effort, and thoracic auscultation may indicate respiratory compromise. Point of care thoracic ultrasound (POCUS) is useful in triage looking for pneumothorax, pulmonary contusions, pleural effusion, and other parenchymal diseases (e.g., aspiration pneumonia).

After initial triage, our goal is to prevent and treat secondary injuries. While primary injury occurs at the time of trauma and cannot be changed, secondary injury occurs minutes to days following the injury and results from local and systemic derangements. Stabilization is important for limiting secondary brain injury. The focus is on adequate fluid resuscitation and appropriate oxygenation and ventilation to optimize cerebral perfusion. Secondary injury can be limited by monitoring and treating derangements such as hypotension, hypoxia, systemic inflammation, hyper- or hypoglycemia, hyper- or hypocapnia, hyperthermia, and electrolytes or acid-base balance abnormalities.

If possible, perform your neuro assessment before providing analgesics. However, trauma patients typically need analgesia and patient comfort should not be sacrificed. Interpret findings while noting the patient's stability and condition. Focus on mentation, brain stem reflexes, and motor activity/posture to assign a modified Glasgow coma scale (MGCS) score. The MGCS is useful for serial monitoring and is assessed as frequently as every 30 minutes in critical patients. An MGCS of 8 within the first 48 hours of hospitalization has been associated with a 50% probability of survival.

Imaging

Extensive imaging may be required in cases of polytrauma. Traditional imaging options include thoracic and abdominal radiographs and abdominal and thoracic ultrasound. More recently, full-

body computed tomography scan (i.e. trauma CT) has become our modality of choice because it typically does not require general anesthesia, is fast, and can be relatively cost-effective. CT also better identifies fractures and can reveal parenchymal damage, hemorrhage (intra- and extra-axial), and herniation. Patient positioning with CT is more forgiving and requires less manipulation compared to traditional radiography. MRI may be required for more subtle lesions, but disadvantages include the need for general anesthesia, longer procedure times, availability, and cost.

Treatments

Fluid Therapy

The goals of fluid therapy include rapid reversal of hypovolemia, prevention of hypotension, and maintenance of cerebral perfusion while avoiding intracranial hypertension. Treatments to avoid include diuretics like furosemide. Previously, diuretics were thought to reduce cerebral edema via dehydration, but the risk of hypovolemia/hypotension far outweighs any perceived benefits. Permissive hypotension or hypotensive resuscitation is a common strategy in trauma but is NOT appropriate for head trauma. Single episodes of hypotension have been shown to increase mortality in human TBI.

Head trauma can disrupt the blood-brain barrier making it less fluid-tolerant. Cerebral blood pressure autoregulation may be impaired, making the brain more sensitive to changes in volume and reliant on blood pressure. Balancing cardiac output and limiting cerebral edema remains challenging. Isotonic crystalloids should be titrated to effect. Although 0.9% NaCl contains the least amount of free water (i.e., the highest sodium content), it is also an acidifying solution, which may worsen the acid-base balance. Large volumes of isotonic crystalloids can exacerbate tissue edema. The volume of fluids given is likely more impactful than the type of isotonic crystalloid. Hypertonic saline (HTS) rapidly expands intravascular volume expansion. The dose for 7% HTS is 4 mL/kg IV over ~10 min. The response to HTS is rapid, but redistribution limits the duration of action to <75 min. HTS should be avoided if hypernatremia is present. Dehydration is often considered a contraindication for HTS, but experimental studies have demonstrated the efficacy and safety of HTS in dehydrated patients.

Colloids are controversial. Colloids may have better IV fluid retention and decreased risk of edema. However, these benefits are unproven, and some studies have shown negative effects. Leakage of colloids through a disrupted BBB may create oncotic shifts promoting edema formation, leading to increased ICP and mortality. Synthetic colloids such as hydroxyethyl starches may cause kidney injury although unproven in dogs and cats. Some studies favor synthetic colloids.

Hyperosmolar Therapy

Hyperosmolar agents decrease ICP by creating an osmotic gradient moving water to the IV space. Mannitol and HTS are both effective, but some recent meta-analyses favor HTS. Mannitol has an immediate plasma volume-expanding effect with a reduction in viscosity and improved microcirculatory flow. This causes vasoconstriction and decreases ICP. Mannitol is mostly known for its osmotic effect which takes 15-30 minutes to form and persists for 2-5 hours. Mannitol may also act as a free radical scavenger. Mannitol's diuretic effect may lead to hypovolemia and must be monitored closely. Extravasation of mannitol due to cerebral hemorrhage is a potential concern but remains unproven. Recommended dosing is 0.5 – 1.0 g/kg

IV over 15-20 minutes. Concurrent furosemide administration is not recommended as it increases the risk of dehydration and hypovolemia.

HTS shares similar mechanisms with mannitol. Potential advantages of HTS include volume expansion leading to improved cardiac output and blood pressure, a reduced likelihood of HTS crossing the BBB, improved regional cerebral blood flow by reducing endothelial swelling, and modulation of neuroinflammatory pathways. HTS should be avoided in dysnatremic patients. Recommended dosing is 4 mL/kg for 7.5% NaCl. A somewhat arbitrary cutoff is to avoid HTS if Na+>160 mEq/mL. HTS seems ideal for the hypovolemic patient, while both are reasonable for the euvolemic patient. Both can be used in the same patient.

Anesthetics, analgesics, and sedatives

Anesthesia may be required for surgery, imaging, and mechanical ventilation. Hypotension and alterations in CO2 may promote secondary injury. A balanced anesthetic technique is especially important. Inhalants should be limited, and intravenous anesthesia is recommended.

Analgesia is essential as pain itself can cause increases in ICP. Opioids are ideal as they are cardiovascular sparing and reversible. Full mu agonists such as fentanyl provide consistent titratable analgesia. Recommended dosing for fentanyl is 2-6 mcg/kg/h. Opioid agonist/antagonists such as buprenorphine cause less cardiovascular, respiratory, and CNS depression, but provides only moderate analgesia and are more difficult to reverse. However, having less sedation allows for easier patient assessment.

Benzodiazepines provide anxiolysis and sedation with minimal intracranial, cardiovascular, and respiratory effects. They also enable dose reduction of other agents, such as propofol, minimizing adverse effects.

Ketamine provides analgesia and is a good anesthetic adjunct. Ketamine may have neuroprotective effects. Ketamine also stimulates the cardiovascular system and has minimal respiratory depression. The benefits of ketamine have led to the reexamination of its role in neurotrauma. Historically, ketamine was thought to increase ICP. However, recent studies of ketamine in human TBI do not support the increase in ICP. In fact, some studies show improved cerebral perfusion and lower vasopressor requirements with ketamine.

Dexmedetomidine provides sedation, analgesia, and anxiolysis. It is also easily reversible. However, the use of alpha-2 agonists in head trauma is controversial and caution should be exercised.

Anticonvulsants

A recent study found a higher epilepsy rate in dogs (3.5-6.8%) with head trauma as compared to a standard population epilepsy rate of 1.4%. Seizure activity can increase ICP and metabolic demands leading to secondary injury. As such, seizures must be treated aggressively. Seizure prophylaxis is considered in human TBI for the first week, but there are no studies in veterinary medicine. Benzodiazepines such as midazolam are effective in the emergent setting. Maintenance anticonvulsants such as phenobarbital can be started. Levetiracetam may also be considered with a quick onset and minimal sedation. The duration of treatment is unknown, but tapering can be considered after 3-6 months.

Corticosteroids
Corticosteroids are not recommended for traumatic brain injury patients due to increased mortality and other common adverse effects. In other words, people who received steroids for head trauma died more. Death should be avoided.

Oxygen & Ventilation
Target normal oxygenation and ventilation. Hyperoxygenation/hyperoxemia may worsen reperfusion injury. Aim for a PaO2 >80 mmHg and SpO2 >94%. Flow-by oxygen is well tolerated by most patients. Nasal cannulas can be considered, but sneezing/coughing can increase ICP. Oxygen cages may be inadequate for patients requiring frequent care. Modified E-collars to create an "oxygen hood" are underrated and work well, especially in larger dogs. Aim for normoventilation (PaCO2 35-40 mmHg). Decreases in PaCO2 lead to vasoconstriction, with a PaCO2 less than 30 mmHg causing excessive vasoconstriction, low CBF, and cerebral ischemia. High PaCO2 leads to excessive CBF, increased CBF, and worsened ICP. Back in the day, hyperventilation was recommended to decrease ICP. However, hyperventilation worsens ischemia and is associated with poor outcomes in human studies.

Nutrition
Early nutritional support is essential. Enteral feeding is preferred. Esophagostomy tubes are well tolerated and allow feeding of most commercial diets. The MILA retrograde esophagostomy tubes allow easy placement with minimal manipulation compared to traditional placement with a Carmalt. Owners can use E-tubes at home which may decrease hospitalization times. Parenteral nutrition is also available for patients who cannot tolerate enteral feedings, are at high risk for aspiration pneumonia, or are poor candidates for tube placement.

References are available from the author upon request.