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HEAD TRAUMA IN GENERAL PRACTICE: A NEUROLOGIST'S PERSPECTIVE

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Head trauma is a relatively common presentation in general practice, with the most frequent causes being road traffic accidents, bites, kicks and falls. Fortunately, many cases will require only basic supportive care and management of superficial wounds, ocular, jaw or dental injuries. However, head trauma may also result in structural or physiologic disruption of the brain, termed traumatic brain injury (TBI), which can be fatal. Early recognition and management of TBI is vital to give the best chance of a successful outcome. Head trauma can result in two associated types of brain injury:

Primary injury - the direct physical disruption of the brain and its associated tissues that occurs at the time of trauma (e.g. parenchymal contusion or laceration, blood vessel rupture). Primary brain injury has already occurred by the time of presentation and as such cannot be prevented or directly treated.

Secondary injury - this highly complex cascade of events occurs over minutes to days following primary injury and includes release of excitatory neurotransmitters from damaged brain parenchyma, energy depletion, failure of ion homeostasis, cellular swelling, activation of destructive intracellular enzymes, local production of reactive oxygen species (ROS), local tissue acidosis, and release of inflammatory cytokines. These processes have the potential to result in a catastrophic, expanding focus of tissue injury and cell death. Therefore, the **focus of TBI management is the prevention and treatment of secondary injury.**

The systemic changes that frequently accompany trauma of any kind, such as hypotension, hypoxia, hypo- or hypercapnia, and derangements in electrolyte and glucose levels, can all exacerbate secondary injury. Therefore, systemic stabilization forms a vital role in the initial management of TBI.

INTRACRANIAL PRESSURE AND CEREBRAL PERFUSION

The cranial vault can be thought of as a closed box constrained by a rigid skull. There is a constant low pressure of 5-12 mmHg within this box that is exerted by the tissues and fluids contained within it. This is termed the intracranial pressure (ICP), against which the mean arterial blood pressure (MABP) must pump to provide the brain with an adequate supply of oxygen and nutrients:

Cerebral perfusion pressure (CPP) = MABP – ICP

According to the principle of the Monrie-Kellie doctrine, if there is an increase in fluid or tissue volume within this closed box (e.g. blood, oedema, skull fragment), then this must be met with an equal and opposite decrease in the volume of another compartment to keep the ICP constant (e.g. loss of CSF or blood). This process is known as 'volume buffering' and underlies the concept of intracranial compliance. If this compensatory capacity becomes overwhelmed, then any additional increase in cerebral volume will result in an exponentially large increase in ICP; an increase in ICP to >20mmHg can rapidly result in death, secondary to reduced cerebral perfusion, brain herniation, brainstem compression and cardiorespiratory failure.

In the normal animal, the cerebral blood flow (CBF) is kept at an optimal and constant level in spite of fluctuations in arterial blood pressure (MABP between 50-150mmHg) by either dilating or constricting the cerebral blood vessels to alter the resistance to blood flow. This process is known as pressure autoregulation:

CBF = CPP / Cerebral vascular resistance (CVR)

A process called chemical autoregulation also regulates the CVR in response to changes in cerebral oxygen and carbon dioxide levels to optimize the CBF, with cerebral arterial vasodilation to increase CBF in response to hypercapnia, hypoxaemia, or increases in cerebral metabolic rate, and cerebral arterial vasoconstriction to decrease CBF in response to hypocapnia.

Head trauma (and some anaesthetic agents) can result in failure of these regulatory mechanisms and loss of normal vascular responses. This can lead to potentially catastrophic alterations in CPP and ICP in response to both the intracranial changes and systemic derangements observed in trauma cases (e.g. hypoxia, hypotension, hypercapnia). This underlies the vital importance of systemic stabilization in the management of TBI, aiming to maintain 'normal' blood pressure, blood oxygen and carbon dioxide values as far as possible.

TRIAGE AND GENERAL ASSESSMENT

The initial assessment of any trauma patient should focus on evaluation of the respiratory and cardiovascular systems, together with identification of any life-threatening injuries. This should always occur before a neurological examination is performed. The initial triage can be divided according to the 'ABC' of emergency care (airway, breathing, circulation). Thoracic focused assessment with sonography for trauma (TFAST) can be performed to assess for pneumothorax, pleural effusion (e.g. haemothorax) or pulmonary contusion. Systemic stabilization should be prioritized over performing diagnostic imaging unless there is a clinical suspicion of pneumothorax, or significant pleural effusion that may require emergency drainage. An ECG should also be performed, as cardiac arrhythmias are not uncommon secondary to traumatic myocarditis. Bladder integrity and the presence of abdominal effusion (e.g. haemoabdomen) should also be considered.

SEIZURE MANAGEMENT

If an animal presents in a state of generalized seizure activity following head trauma, then this should be managed as a priority. The most common first line medications are diazepam (0.5-1.0 mg/kg *IV*) or midazolam (0.2 mg/kg *IV*). Levetiracetam, phenobarbitone, ketamine and propofol can also be used if seizure activity persists in spite of benzodiazepine administration. The prophylactic use of anti-epileptic drugs in all patients with head trauma, whether they have shown seizures or not, remains controversial. A recent meta-analysis with evidence quality assessment in humans looked at the association of early seizure prophylaxis with post-traumatic seizures and mortality. This supported The Brain Trauma Foundation's guidelines for acute, short-term antiseizure prophylaxis (e.g. phenytoin, levetiracetam) to minimise the risk of early seizures and consequent secondary brain injury. However, primary seizure prophylaxis within 7 days postinjury did not appear to be associated with a substantial difference in the 18- or 24-month epilepsy risk or all-cause mortality, but the evidence was of only low / moderate quality. Owners should be warned that an estimated 6-10% of dogs with head trauma may go on to develop 'post-traumatic seizures/epilepsy' in later life.[1]

SYSTEMIC STABILIZATION

Animals will commonly present in hypovolaemic shock following trauma and it is essential that this is recognized and addressed as soon as possible, with the aim of establishing normovolaemia, restoring cerebral perfusion, and minimizing the extent of secondary brain injury. **Systemic stabilization remains the most important factor in determining the outcome following TBI.**

Oxygen should be delivered by flow-by, with or without a mask. Nasal oxygen prongs or cannulas should be used with care, as they may stimulate sneezing which can further increase ICP. The use of an oxygen cage is not advised, as their use is likely to preclude the frequent monitoring required in these cases. Goals for oxygenation are: normal respiratory rate and pattern, normal mucous membrane colour, SpO_2 - >95% PaO_2 - >80-90mmHg.

Adequate ventilation is required for both oxygenation of the blood and for carbon dioxide exchange. This is important, as $PaCO_2$ is a potent regulator of CBF and cerebral blood volume (CBV). The goal of ventilation, whether spontaneous, manual or mechanical, is to maintain a normal $PaCO_2$ of 35-40mmHg. Deliberate

hyperventilation in an attempt to reduce $PaCO_2$ levels, thereby stimulating cerebral vasoconstriction to reduce the CBV and ICP, has been suggested in the management of TBI.[2] However, this is not currently recommended due to the risk of exacerbating cellular ischaemia in a brain that is already vulnerable to hypoxaemia. It is therefore advised that the $PaCO_2$ level is not allowed to fall below 35mmHg.

Intravenous access by placement of one or more wide-bore intravenous catheters is essential to allow fluid resuscitation and the administration of medications. Blood can be acquired for a minimum database at this time (packed cell volume (PCV), total protein (TP), blood glucose, urea, electrolyte levels). Head trauma may be associated with hyperglycaemia in dogs, cats and humans, with a concern that this may potentiate neurologic injury, and a previous study demonstrated that the degree of hyperglycaemia was associated with the severity of head trauma in dogs and cats. Whilst the degree of hyperglycaemia has so far not been correlated with survival or outcome in cats, it is still prudent to regularly monitor blood glucose and to keep this parameter within the normal range.

The goal of fluid therapy is the rapid restoration of circulating fluid volume to ensure an adequate CBF and oxygen delivery to vital organs. The choice of fluid remains controversial and will also depend on availability in general practice. Regardless of the fluid used, vital parameters should be assessed regularly, and the fluids titrated to effect to avoid overload.

Isotonic crystalloids - Boluses of 15-20ml/kg (dog) or 10-15ml/kg (cat) of 0.9% NaCl can be given until there is normalization of the heart rate, pulse quality, capillary refill time, mucous membrane colour and blood pressure (MABP 80-100mmHg).

Hypertonic saline - Smaller volumes of this hypertonic solution can be used to produce a rapid rise in blood osmolarity, drawing fluid into the circulation from the interstitial and intracellular compartments. This volume expanding effect occurs within minutes and lasts for 15-75 minutes, before redistribution to other fluid compartments. The recommended dose is 4ml/kg of 7.5% sodium chloride administered over 5 minutes. The administration of isotonic crystalloids following the use of hypertonic saline is essential to prevent tissue dehydration, provide maintenance fluid requirements and correct for on-going losses.

A mention should be made here of something called the ‘Cushing reflex’. This is a reflex response by the body that occurs when the ICP rises to a point that cerebral perfusion is compromised. This reduction in CPP results in a rise in brain CO_2 levels, followed by a reflex increase in MABP to maintain cerebral perfusion and a subsequent baroreceptor reflex that reduces the heart rate to protect end-organs. It is therefore characterized by systemic hypertension (>160mmHg) in the presence of marked bradycardia (<40-60 bpm). This reflex should always be accompanied by other indicators of increased ICP (e.g. reduced level of mentation), and care should be taken not to confuse it with other causes of bradycardia, such as medetomidine/dexmedetomidine or opiate administration. The focus of management should be on the cause of this reflex by reducing the increased ICP and not on directly treating the bradycardia (e.g. glycopyrrolate administration) as the bradycardia is protective in this instance.

To aid venous drainage from the head, and to minimize increases in intracranial venous blood volume, the animal’s head should be kept in the midline and elevated by 10-30 degrees. The jugular veins should be kept free of pressure or occlusion to assist with venous drainage.

NEUROLOGIC ASSESSMENT

Neurologic assessment should ideally be performed once normovolaemia, normotension, appropriate oxygenation and ventilation are achieved. Frequent reassessment and recording of trends are vital to guide the response to treatment and potential prognosis, with repeat assessment recommended every 30-60 minutes until the animal is stable. The use of a scoring system, such as the Modified Glasgow Coma Scale (MGCS) or Animal Trauma Triage (ATT) score, allows for more objective assessment of neurologic dysfunction. It may also be useful to guide prognosis, aid in the consistency of monitoring progression, and in case handover between staff responsible for the animal’s care.[3,4]

MANAGEMENT OF INCREASED INTRACRANIAL PRESSURE

The initial management of increased ICP should always focus on normalization of systemic parameters (MABP, PaO_2, $PaCO_2$). If the animal's clinical status is deteriorating in spite of systemic stabilization, then additional interventions include the use of hyperosmolar agents such as mannitol (0.5-1 g/kg *IV* over 15-20 minutes) or hypertonic saline (4ml/kg of 7.5% soln. *IV* over 5 minutes).[5] These agents create an osmotic gradient that shifts water from the interstitial space to the intravascular space. This increases the circulating fluid volume, reduces blood viscosity, improves cerebral perfusion and oxygen delivery, and decreases ICP. Hypertonic saline is a more appropriate fluid choice in a hypovolaemic animal, but both agents are reasonable choices in a euvolaemic animal. Previous concerns regarding the use of mannitol in animals with suspected cerebral haemorrhage have not been supported by clinical studies.

ANALGESIA, SUPPORTIVE CARE AND NUTRITION

Pain and anxiety may increase the ICP, therefore the use of analgesics and/or sedatives is vital. Opiates are the most common first-line analgesics (such as methadone, butorphanol or fentanyl). The use of NSAIDs should ideally be reserved for when the animal is haemodynamically stable.

Any recumbent animal should be frequently turned and provided with soft, clean, dry bedding. A urinary catheter can be placed to allow monitoring of fluid 'ins-and-outs' and also to relieve the anxiety or discomfort associated with a need to urinate. The blood glucose level should be monitored and kept within the normal range, and the body temperature should be maintained between 37-38.5°C. Consideration should also be made for the provision of nutrition during the recovery period.

DIAGNOSTIC IMAGING

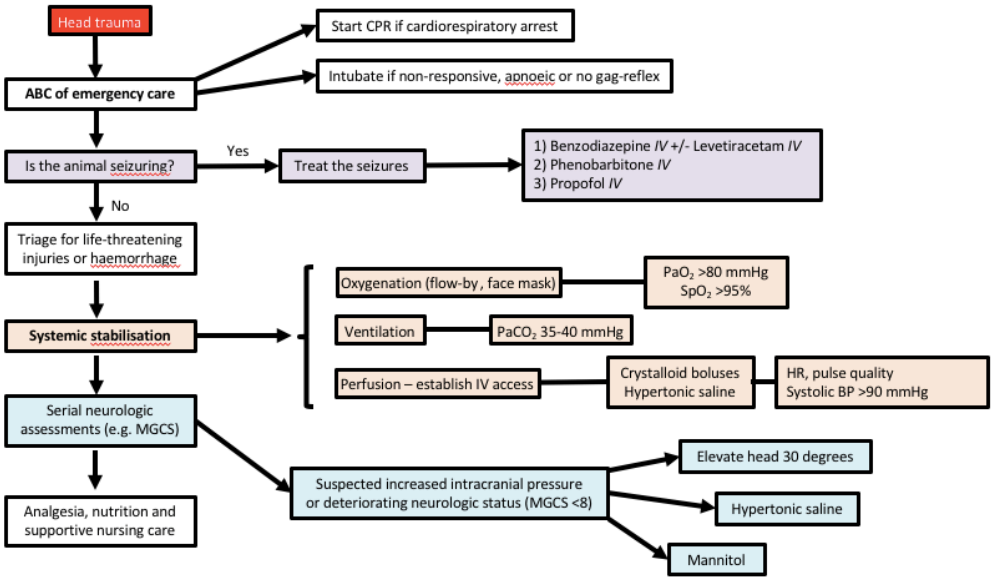
Once normovolaemia, normotension and adequate oxygenation/ventilation are established, basic imaging to assess the thorax (e.g. pleural effusions, pneumothorax, diaphragmatic rupture) and abdomen (e.g. haemoabdomen, uroabdomen) can be performed. Spinal radiographs could also be taken to assess for spinal injuries that may influence both the safe handling of the patient and interpretation of the neurological examination. Computed tomography (CT) offers a rapid technique for screening animals following trauma, either conscious or under light sedation, and allows identification of skull fractures or haemorrhage that may benefit from surgical treatment. The utility of brain MRI in head trauma cases has been reported and the findings on MRI appear to correlate with both the clinical status (MGCS) and outcome.[6]

THE ROLE OF CORTICOSTEROIDS

Following studies in human medicine, it is widely accepted that the use of high/'shock' doses of methylprednisolone sodium succinate (MPSS) in head trauma is contraindicated, with the theoretical benefit in terms of free radical scavenging being outweighed by the detrimental effects of immunosuppression (pneumonia, urinary tract infections), delayed healing, gastrointestinal adverse effects, hyperglycaemia and cerebral acidosis.[7] However, the role of corticosteroids in head trauma has recently been revisited in humans, with a suggestion that there may be a role for short-term use of lower doses of dexamethasone around 7 days post-trauma in some cases, but this has yet to be established in dogs and cats.

WHEN SHOULD REFERRAL BE CONSIDERED

- For continued stabilization, monitoring and assessment
- Management of other injuries:
  - Jaw fractures / dental problems
  - Ophthalmic injuries
  - Diaphragmatic / bladder rupture
  - Limb bone or vertebral fracture
- Advanced imaging (e.g. facial / jaw fractures, depressed skull fracture)
- Deteriorating neurological status in spite of systemic stabilisation



Taken from **A Practical Approach to Neurology for the Small Animal Practitioner**, Freeman and Ives, Wiley publications

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