

Managing head-trauma patients

An overview of the evaluation and treatment of head trauma in small animal patients is provided by Søren Boysen DVM DACVECC, Faculty of Veterinary Medicine, University of Calgary, Canada

Traumatic brain injury (TBI) following head trauma may result in death from progressive inflammation and increased intracranial pressure (ICP). Through rapid identification of TBI and early initiation of tailored therapies, many dogs and cats can recover to lead functional lives, even in the face of severe brain injury. Initial assessment and serial monitoring of neurological status can be helpful in determining prognosis and evaluating response to therapy. Therapy is aimed at minimising further secondary brain injury by treating and preventing systemic conditions which can exacerbate inflammation in the brain or contribute to increased ICP. Correction and prevention of hypovolaemia is important to prevent ischaemic brain injury, but requires cautious use of intravenous (IV) fluids to avoid increases in ICP. Hypertonic saline is often used to combat hypovolaemia while simultaneously decreasing ICP. Mannitol can be used to decrease ICP along with other management strategies (such as head elevation, adequate ventilation, avoiding nasal lines and jugular vein occlusion). Pain control is crucial for head trauma patients, although analgesics with long-acting sedative effects may interfere with serial neurological assessments. Seizure control, glucose control, temperature regulation and supportive care are also very important to prevent further cerebral damage.

PATHOPHYSIOLOGY

Primary brain injury occurs at the time of trauma (including contusions, lacerations, haemorrhage and compression by skull fractures) and there is very little the veterinarian can do to treat the primary injury. Secondary injury occurs after the primary injury and is the result of ongoing inflammatory conditions, metabolic disturbances, ischaemia and hypoxaemia. These conditions are initiated by the primary injury and may be exacerbated by systemic changes in the animal (hypotension, hyperthermia, hypoglycaemia, hyperglycaemia, hypoxaemia and anaemia). Therapy is directed towards minimising further secondary injury.

TRIAGE AND INITIAL STABILISATION

The importance of simultaneously evaluating the cardiovascular, respiratory and neurological systems in trauma patients during the initial triage exam cannot be over-emphasised. Hypovolaemia and hypoxaemia are proven to contribute to ischaemic brain injury and increased ICP, and negatively impact outcome in patients suffering from TBI. The minimum emergency database (packed cell volume [PCV], total solids, blood urea nitrogen

[BUN], glucose and, if available, lactate, blood gas/pulse oximetry and arterial blood pressure measurement) should also be completed at the time of presentation.

NEUROLOGICAL EVALUATION

Following the initial triage exam, a concentrated neurological examination should focus on level of consciousness (response to toe pinch or loud noise), pupillary size and light reflexes, ability to elicit physiological nystagmus, and limb rigidity. A more thorough neurological examination, including evaluation of all cranial nerves, can be performed when the animal is stable. Remember that full recovery may take weeks to months and long-term deficits and seizures may occur. The Modified Glasgow Coma Scale (MGCS) may be helpful in determining a prognosis and in evaluating response to therapy (Table 1). Although the MGCS has been used to help determine the prognosis at the time of presentation it should not be used as a sole predictor of outcome. It is more valuable when used serially to detect deterioration or improvement in the patient. Serial assessments should be performed every 30 minutes until the animal is stable, and then every 60 minutes, to help detect changes that may require urgent intervention. The MGCS can help provide a more objective comparison of patient evolution when different clinicians in the hospital are performing serial neurological examinations.

Consciousness

There are four levels of consciousness. Prognosis decreases as the level of consciousness decreases and a state of coma warrants a guarded prognosis. Serial neurological assessment (every 30-60 minutes) will detect sudden changes in the level of consciousness, which warrants early intervention.

- Alert – responds normally to the environment. A state of delirium may also be present where the animal is conscious, but the response to normal stimuli is exaggerated;
- Obtunded/depressed – conscious and responsive to stimuli, but response is diminished;
- Stuporous/semi-comatose – unconscious state that requires strong stimuli (usually noxious toe pinch) to evoke a response (often reduced);
- Comatose – a state of unconsciousness unresponsive to all (even noxious) stimuli.

Pupil size and response to light

When evaluating pupils, the most important parameter is



Figure 1: Pupillary response to light is a key factor in determining prognosis in animals with head trauma. The cat in this photo has very dilated pupils with slow but present direct and consensual light reflexes.



Figure 2a: Decerebellate rigidity involves extension of the head and neck with rigid extension of the front limbs and flexed or intermittently flexed hind limbs.

response to light:

- Miotic pupils with response to light – fair-to-good prognosis;
- Unresponsive pupils (in the absence of ocular trauma) – grave prognosis.

A rapid change in pupillary constriction or dilation with a concurrent change in mentation often indicates an increase in ICP and the need for immediate therapy.

Physiological nystagmus

Physiological nystagmus is noted when the head is gently turned from side to side, which results in the rapid horizontal 'jerky' movement of the eyes. The loss of physiological nystagmus is associated with brain stem lesions and a poor prognosis. If the presence or absence of physiological nystagmus is uncertain, infusion of warm (37° Celsius) or cold (4° Celsius) water into the ear canal should result in the induction of physiological nystagmus.

Limb rigidity

- Decerebrate rigidity – extension of the head and neck (opisthotonus) with extension of the front and rear limbs suggests a grave prognosis. Animals with decerebrate rigidity have severely affected mentation and are stuporous to comatose.
- Decerebellate rigidity – opisthotonus with extension of front limbs. Hind limbs are often flexed, although



Figure 2b: Decerebrate rigidity involves extension of the head and neck with rigid extension of all four limbs. Most of these patients have severely affected levels of consciousness. Prognosis is generally worse than for decerebellate rigidity.

at times they may be extended (may be intermittent). Animals with decerebellate rigidity are conscious and have a better prognosis than patients with decerebrate rigidity. Progression of decerebellate to decerebrate rigidity is often a sign of progressive brain injury.

TREATMENT

Cardiovascular system

Hypovolaemia is common in trauma victims and is one of the most serious secondary complications associated with a worse outcome in humans with TBI. Hypovolaemia must be identified and corrected immediately as it causes further ischaemic brain injury. IV fluids must be given to correct hypotension and associated signs of hypovolaemia (tachycardia, pale mucous membranes, prolonged capillary refill times, hyperlactataemia). In addition, fluids must be continued following resuscitation to meet the patient's ongoing and maintenance requirements, which will help to ensure euvoalaemia and prevent hypovolaemia from recurring.

Isotonic crystalloids

Isotonic crystalloids are adequate fluids to treat shock in the head-trauma patient, but care must be taken to prevent further cerebral oedema. Large quantities of isotonic crystalloids are often required to reverse signs of shock and approximately 75% of isotonic crystalloid fluids enter the interstitial space within an hour, which can exacerbate cerebral oedema. The author is therefore conservatively aggressive with isotonic crystalloids in patients with TBI and will administer repetitive 10-15ml/kg boluses every 5-10 minutes until systolic blood pressure (BP) is >90mmHg. Depending on initial response to isotonic crystalloids, consider adding hypertonic saline or a colloid.



Figure 3: Oxygen therapy is often very important in patients with head trauma. An oxygen cage with sensor is ideal for small animal patients. Nasal lines that may result in sneezing should be avoided, if possible.

Hypertonic saline

Hypertonic saline (4-6ml/kg) may be a better fluid for the treatment of head-trauma patients. Small boluses of fluids are often sufficient to reverse the signs of shock and the increase in plasma osmolality associated with the hypertonic saline has been shown to act similarly to mannitol and decrease ICP. However, the duration of action for hypertonic saline is short-lived and patients must be closely monitored for recurrence of shock. Combining hypertonic saline with a colloid (one part 23% hypertonic saline mixed with 2.5 parts colloid given at 4-6ml/kg) may prolong the initial beneficial effects of hypertonic saline.

Colloids

Colloids (5ml/kg boluses until systolic blood pressure ≥ 90 mmHg) may also be used. The total fluid volume required tends to be less and end points of resuscitation tend to be reached faster with colloids than with isotonic crystalloids. However, it is not known how colloids are removed from the central nervous system (CNS) if they cross a disrupted blood-brain barrier and they may act to draw fluid into the CNS interstitium.

Blood products

Blood products should be given as needed to correct a falling hematocrit (ie. acute decrease in haematocrit to $<25\%$ in the animal with TBI), because haemoglobin contributes significantly to oxygen delivery.

Respiratory system

Oxygen should be supplied to all head-trauma patients during initial stabilisation (first 12-24 hours) to avoid hypoxaemia (avoid nasal lines that could cause sneezing and subsequently increased ICP). After 24 hours of oxygen therapy there is increased risk of oxygen toxicity if animals are maintained on too high a level of inspired oxygen ($>50\%$ inspired oxygen concentration). Therefore, supplemental oxygen should be discontinued if animals can maintain good oxygen saturation ($>96\%$ SaO₂) on room air or decreased to the lowest levels that will maintain good oxygen saturation. It is recommended that oxygen saturation be monitored to maintain levels at $>96\%$ (PaO₂ >80 mmHg) if pulse oximetry or blood gas analysis is available.

Decreasing intracranial pressure

Bradycardia in the face of hypertension (Cushing's reflex) is often reflective of increased ICP and warrants immediate therapy to reduce ICP. Note: the Cushing's reflex occurs with severe brain injury and these patients tend to be stuporous to comatose; look for another cause of bradycardia in the brain trauma patient that is fairly alert and responsive.

Mannitol

Mannitol should be given to patients with severe head trauma, neurologically deteriorating patients, and patients suspected to have elevated ICP. Mannitol dose is 0.25-1gm/kg over 10 minutes, repeated every two to six hours as needed, up to three times in 24 hours. Mannitol is an osmotic diuretic, so patients should be closely monitored for development of hypovolaemia. Avoid mannitol in dehydrated, hypovolaemic patients and in patients with underlying cardiac disease or hyperosmolar states (ie. significant hypernatraemia). Euvolaemia (through fluid administration) should be maintained during mannitol administration.



Figure 4: Note the entire surface on which the dog is lying in this image is slanted 30 degrees.

Head elevation

The cranial portion of the animal should be elevated 20-30 degrees above the pelvis in an effort to maximise cerebral venous drainage. Avoid occlusion or compression of the jugular veins, which could decrease cerebral venous drainage and increase ICP. The author prefers to place the animal on an inclined board to minimise the risk of jugular compression.

Carbon dioxide regulation

Intracranial vasodilation can cause increased ICP as blood pools within the vessels of the brain. Hypercapnia ($PCO_2 >45\text{mmHg}$) can cause excessive intracranial vasodilation. Efforts should be made to ensure adequate ventilation and maintain the carbon dioxide level between 25-45mmHg to prevent increased ICP. Hyperventilation of head trauma patients ($PCO_2 <25\text{mmHg}$) should be avoided as this can lead to vasoconstriction, decreased cerebral perfusion and deterioration of neurologic function. Referral to a 24-hour facility is recommended for comatose patients or patients requiring mechanical ventilation.

Avoid manoeuvres that increase intracranial pressure

Avoid nasal lines that might cause sneezing. Avoid medications that could cause vomiting (IV morphine and hydromorphone). Avoid compression of jugular veins. Administration of lidocaine during intubation (0.75mg/kg IV) may suppress gag and cough reflexes which increase ICP.

Analgesia/sedation

Pain control is crucial to decrease sympathetic activity, which may contribute to elevated ICP through increased cerebral blood flow. Avoid long-acting sedatives because they may prolong the periods between useful neurological examinations and interfere with the detection of deterioration. The author recommends fast and short-acting medications given at lower doses and titrated to effect. Opioids must be given slowly and at the lowest dose to effect as they may result in vomiting which can increase ICP. Consider local anesthesia when appropriate to decrease the need for systemic analgesia (ie. rib blocks for rib fractures).

Preference

Fentanyl (titrate to effect @ 5ug/kg IV boluses in cats and dogs to maximum dose of 20ug/kg and follow with a constant rate infusion (CRI) @ 10-20ug/kg/hr). Monitor respiratory rate and effort to be sure hypoventilation and hypercapnia is not induced, especially with higher doses.

Reasonable alternatives

Morphine (0.05mg/kg IV cats, and 0.2mg/kg IV dogs, given slowly every one to three minutes to effect, maximum total dose of 1mg/kg in dogs and 0.5mg/kg in cats). Total effective dose of morphine can then be divided by four and given as an IV hourly CRI. Hydromorphone (0.025mg/kg IV slowly q1-3 min to effect in dogs and

Table 1: Modified Glasgow Coma Scale (MGCS). For each of the three categories below, select the description that is most accurate for the patient.

Motor activity	
6	Normal gait; normal spinal reflexes
5	Hemiparesis, tetraparesis, or decerebrate activity
4	Recumbent; intermittent extensor rigidity
3	Recumbent; constant extensor rigidity
2	Recumbent; constant extensor rigidity with opisthotonus
1	Recumbent; hypotonia of muscles; depressed or absent spinal reflexes
Brain stem reflexes	
6	Normal pupillary light responses and oculocephalic reflexes (physiological nystagmus)
5	Slow pupillary light reflexes; normal to reduced oculocephalic reflexes
4	Bilateral unresponsive miosis; normal to reduced oculocephalic reflexes
3	Pinpoint pupils; reduced to absent oculocephalic reflexes
2	Unilateral, unresponsive mydriasis; reduced to absent oculocephalic reflexes
1	Bilateral, unresponsive mydriasis; reduced to absent oculocephalic reflexes
Level of consciousness	
6	Occasional periods of alertness and responsive to the environment
5	Depression or delirium; capable of responding to the environment
4	Semi-comatose; responsive to visual stimuli
3	Semi-comatose; responsive to auditory stimuli
2	Semi-comatose; responsive to noxious stimuli
1	Comatose; unresponsive to repeated noxious stimuli
Total the scores from the three sections	
3-8	Prognosis grave
9-14	Prognosis poor to guarded
15-18	Prognosis good

cats, maximum total dose of 0.2mg/kg). Oxymorphone (0.03mg/kg IV slowly q1-3 min to effect in dogs and cats, maximum total dose of 0.2mg/kg). Total effective dose of hydromorphone or oxymorphone can be divided by two to four and given as an hourly CRI.

Cats may become excitable with any pure opioid agonist, and butorphanol as a CRI may be better suited to some feline patients (CRI dosage of butorphanol in cats is 0.1-0.2mg/kg/hr). However, the author prefers to avoid the use of butorphanol in dogs with head trauma as the sedative effects can last up to between two and four hours and interfere with serial neurological examinations. Ketamine is not recommended in patients with head trauma as it has been associated with elevations in ICP. Diazepam (0.2mg/kg IV) is reversible and can be used for seizure management and to control excessive excitement and dysphoria seen in some head injury patients; however, prolonged sedation can make serial neurological examinations difficult.

The use of non-steroidal anti-inflammatory drugs (NSAIDs) in head-trauma patients is debatable, and NSAID administration should be delayed at least until it is certain the animal is neurologically stable, that there is no ongoing haemorrhage in the brain or elsewhere, and that the animal is not in shock.

PREVENT FURTHER CEREBRAL DAMAGE**Glucose control**

Both hypoglycaemia and hyperglycaemia have been associated with poorer outcomes in patients with TBI. Careful monitoring of glucose levels is recommended. In cases of documented and significant hypoglycaemia (glucose <3mmol/L), dextrose should be administered while blood glucose concentration is carefully monitored to minimise potential sustained hyperglycaemia. Evidence currently suggests that hyperglycaemia is detrimental to nervous tissue in patients with TBI so hyperglycaemia should be avoided (avoid dextrose unless hypoglycaemia is documented and reconsider the use of glucocorticoids).

Steroids

Although extremely controversial, steroids are not currently recommended for treatment of TBI in humans. The author cannot currently recommend the use of steroids in animals with TBI due to the potential to cause hyperglycaemia through counter-regulatory effects and the potential to cause immunosuppression.

Temperature regulation

Induced hypothermia cannot be recommended in veterinary patients at this time. However, efforts should be made to avoid hyperthermia and maintain body temperature between 37-38°C Celsius. A fan or cold, water-soaked towels applied to the neck and torso may be tried if the temperature increases to 39°C Celsius or higher. Shivering reflexes should

be avoided as they may result in increased ICP.

Seizure control

Rapid and aggressive therapy should be instituted to arrest seizures in head-trauma patients (diazepam 0.5-1mg/kg IV repeated up to three times; if diazepam is unsuccessful then follow with phenobarbital 4-16mg/kg IV [4mg/kg boluses every 5-10 minutes]). Propofol can also be used (1-4mg/kg IV followed by a CRI of 0.1-0.5mg/kg/min if needed). Be sure to monitor for hypoventilation (hypercapnia) and treat accordingly if phenobarbital or propofol are used.

Nutrition

Nutrition in patients with TBI can be challenging. Oral nutrition should be withheld until animals are responsive and possess a normal gag reflex to decrease the risk of aspiration. The effects of diet on hyperglycaemia must be considered when using parenteral nutrition and high-calorie diets. Although no clear answers are available to address nutrition in head-trauma patients, at this time efforts should still be directed towards early nutritional support.

Supportive care

Physical therapy, turning of the patient from side to side, lubrication of the eyes and moistening of the mouth every four hours, as well as placement of a urinary catheter to prevent urine scald, may be required in patients that are stuporous to comatose.

Reader Questions and Answers

1: WHAT SYSTEMIC CONDITION MAY SLOW THE PROGRESSION OF SECONDARY BRAIN INJURY?

- A: Hypovolaemia/hypotension
- B: Hypoglycaemia
- C: Hypothermia
- D: Hypoxaemia
- E: Hypercapnia

2: WHAT IS THE VALUE OF THE MODIFIED GLASGOW COMA SCALE IN THE MANAGEMENT OF HEAD TRAUMA PATIENTS?

- A: It allows the cost of therapy to be estimated based on the initial score calculated
- B: It is 100% sensitive and 100% specific for prognosis and can be used to rule out herniation
- C: It can accurately predict and correlates well with intracranial pressures allowing tailored therapy
- D: It will predict the likelihood of brain herniation at the level of the foramen magnum
- E: It can trend response to therapy and provides a more objective comparison between different doctors

3: WHAT NEUROLOGICAL PARAMETERS ARE ASSESSED ON THE MODIFIED GLASGOW COMA SCALE?

- A: Level of consciousness, pupillary size/light reflexes, physiological nystagmus, limb rigidity/motor activity
- B: Respiratory pattern, pupillary size/light reflexes, physiological nystagmus, gag/swallow reflex

- C: Palpebral reflex, menace response, limb rigidity/motor activity, pupillary size/light reflexes
- D: Menace response, level of consciousness, pupillary size/light reflexes, respiratory/breathing pattern
- E: Palpebral reflex, limb rigidity/motor activity and, pupillary size/light reflexes, gag/swallow reflex

4: STEROIDS NOT CURRENTLY RECOMMENDED FOR USE IN PATIENTS WITH HEAD TRAUMA BECAUSE THEY MAY CAUSE...

- A: Hyperglycaemia and immunosuppression
- B: Lipid peroxidation and free radical production
- C: Increased intracranial pressure and herniation
- D: Cerebral bleeding and coma
- E: Seizures and prolonged recovery

5: WHAT STRATEGIES CAN BE USED TO REDUCE INTRACRANIAL PRESSURE VIA INCREASED OSMOTIC PRESSURES?

- A: Furosemide administration and oxygen therapy
- B: Mannitol and hypertonic saline administration
- C: Head elevation of 20-30 degrees above the pelvis, ketamine administration
- D: Blood and plasma transfusions
- E: Potassium chloride and propofol administration

ANSWERS: 1: C, 2: E, 3: A, 4: A, 5: B