**Clinical Commentary**

**Head trauma: A neurological emergency**

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Trauma to the nervous system may involve the brain, brainstem, spinal cord or peripheral nerves (Owen and Maxie 1978; Stick *et al.* 1980; Reed 1987, 1994; Stewart 1987; Collatos *et al.* 1991; Sinha *et al.* 1991; Scheffer *et al.* 2001). The frequency of trauma to the nervous system of horses has been reported to account for up to 22% of CNS disorders in one study (Feige *et al.* 2000). Head trauma is often one of the most difficult injuries to manage because of the direct effects of the injury as well as the secondary complications of bacterial meningitis, such as was seen in the horse in the report by Atherton *et al.* (2007). Traumatic brain injury may result in focal or diffuse injury to the brain and can lead to death due to damage of vital brain structures, haemorrhage or infection. Head trauma affects horses of all ages although foals and young horses appear to be most commonly affected. Clinical signs can be mild to severe depending on the extent of brain damage. Common head injuries that horses sustain are fractures of the basisphenoid, basiocipital and temporohyoid bones. These injuries occur as a result of falling very hard, flipping over backwards or running into an immovable object. Injury to the temporohyoid apparatus often has an acute onset, although the injury may result from a more long standing problem.

Traumatic brain injury may occur with or without a fracture, and some of the most severe injuries to the brain occur when the injury is contained within the closed calvarium. Common clinical features of head trauma in horses include an altered level of consciousness (stupor to coma), abnormal behaviour, cranial nerve deficits, and sometimes skull fractures. If the veterinarian is able to reduce the severity of the secondary damage to the nervous tissue it helps survival and recovery of as much nervous tissue as possible.

In hypovolaemic shock, autoregulation mechanisms will be activated to protect the ischaemic sensitive neurons of the brain and spinal cord by maintaining adequate blood flow. Cerebral perfusion above 12 ml/mg/min is adequate to prevent damage to neuronal cells (Leker *et al.* 2002; Mobbs *et al.* 2002). While changes in cerebral perfusion of 50–150 mmHg do not cause significant changes in cerebral blood flow, perfusion pressures below 50 mmHg often results in ischaemic necrosis (Hardman and Manoukian 2002; Bayir *et al.* 2003).

The majority of published information about diagnosis and management of head injury in horses and foals is derived from isolated case reports and textbooks. The information is extrapolated from articles about the problem in man or domestic animals other than horses (Turner 1979; Stick *et al.* 1980; Adams and Mayhew 1985; Koblik *et al.* 1985; Reed 1987, 1994; Stewart 1987; Mayhew 1989; Sinha *et al.* 1991; George 1996; Feige *et al.* 2000). Clinical signs following head trauma are dependent on location and severity of damage. Performing a neurological examination on a horse with head trauma can be quite challenging and sometimes requires the use of sedation, especially if the animal is attempting to stand but is not fully aware of its surroundings or lacks coordination due to either cerebellar or vestibular damage. Evaluation of the level of consciousness, along with cranial nerve signs, can help with neuroanatomic localisation of brain or brainstem injuries. When a horse with a head injury either lays or falls down, it may become anxious and attempt to rise, which can lead to further damage to the nervous system. Xylazine is this author’s usual drug of choice, although chloral hydrate, diazepam, midazolam, phenobarbital and acepromazine may also be considered.

Brain injuries include concussion, contusion, laceration and haemorrhage. These injuries describe symptoms that progress from least to most severe (Stick *et al.* 1980; Reed 1987, 1994; Stewart 1987). Concussion is associated with a brief loss of consciousness but does not result in permanent brain damage and has a favourable prognosis. A contusion indicates a distinct area of swollen brain tissue along with vascular damage. Cerebral lacerations are often caused by sudden acceleration or deceleration injuries or by a projectile object penetrating the skull and indicate structural damage to the nervous tissue (Stick *et al.* 1980; Reed 1987, 1994; Stewart 1987). Haemorrhage within the brain parenchyma or within a closed calvarium can lead to very serious and debilitating consequences, as a result of direct injury to the brain, secondary swelling or herniation.

Regardless of the cause, primary brain injury that initially appears modest may later appear more severe as a result of secondary injury due to haemorrhage, ischaemia or infection. The brain has a high metabolic rate and high oxygen demands, with very minimal energy stores, thus it is subject to very rapid changes when there is loss of oxygen and glucose, and even modest hypotension can convert a reversible brain injury into one in which ischaemic brain...
damage is irreversible (Mazzola and Adelson 2002; Bayir et al. 2003). Therefore, initial aggressive treatment of hypotension is important. Secondary changes result from the direct trauma or ischaemic damage following release of excitatory amino acid neurotransmitters (e.g. glutamate) from damaged brain cells. The excess quantity of neurotransmitters leads to instability of ion channel receptors on brain cells that may not have been damaged, resulting in massive influx of calcium ions into the cells.

A continuous supply of energy is required to maintain membrane potentials and electrochemical gradients to allow normal synaptic transmission. The supply of energy for the brain is produced by oxidation of glucose. When the supply of energy is disrupted, regardless of the reason, neuronal function will be altered within minutes (Biegon 1995; Hardman and Manoukian 2002; Gruen 2002). The brain also requires a nearly constant supply of oxygen. Lack of oxygen may be the result of hypoxaemia or ischaemia. Progressive hypoxaemia leads to increased glycolysis, decreased neurotransmitter production, increased lactate concentration, lowered ATP and energy production, and finally permanent damage to neurons. If cerebral blood flow can be quickly and adequately restored many of the clinical signs might be reversed.

The physical examination is important to help identify injuries to other body systems. Horses with head trauma may also suffer fractures of the axial or the appendicular skeleton and sometimes have a serious injury of the chest or abdominal cavity. Careful monitoring of heart and respiratory rates as well as measurement of blood pressure should be a routine part of the examination. Fluid therapy, usually with 0.9% saline, along with careful monitoring of clinical signs is important. In man the use of dextrose in water or hypotonic solutions may have a deleterious effect on the recovery from head trauma (Schwartz and Fehlings 2002).

The neurological examination should begin by restraining the horse in a safe protected area and should proceed without sedation if possible. The examination should include an assessment of the horse’s level of consciousness, cranial nerve function, head posture and coordination. The pattern of respiration should be monitored carefully, noting any changes over time or changes that occur in response to therapy, as this may indicate a serious life-threatening brainstem lesion. Focal damage to brainstem may be partially assessed by noting changes in the size and function of the pupils, as well as changes in the level of consciousness of the horse.

If the brainstem, especially in the region of the midbrain, is injured the horse will develop severe depression or even coma, loss of normal pupillary light responses and asymmetric pupil size. Damage to the region of the medulla oblongata could result in strabismus, a head tilt and other signs of vestibular disease. If a horse is standing one should attempt to evaluate its ability to walk. If the horse is recumbent and unable to rise the spinal reflexes should be examined. When a reflex is absent it indicates either damage to the motor horn cell in the spinal cord or peripheral nerve injury.

All parts of the neurological examination are important but in a recumbent horse that has suffered serious head trauma, the pupil size, symmetry and response to light directed into the eye are critical. A change in pupil size from bilateral constriction to bilateral dilation with no response to light shone in either eye is an indication of a worsened condition and indicates the need for prompt therapy.

**Treatment**

Regardless of the cause of the head trauma, prompt attention to alleviate further damage by careful assessment and treatment is important. Most head and spinal cord injuries occur away from a referral centre and therefore will need to be monitored, at least for a time, by the attending veterinarian and the owner. The most important diagnostic tool available to the attending veterinarian is the neurological examination. Careful monitoring implies frequent or continuous evaluation, as well as alterations in therapy when indicated.

In addition to the changes detected by neurological examination, the veterinarian may also monitor selected biochemical changes in the blood or cerebrospinal fluid. In blood the arterial blood gas tensions may be of help to determine the need for either controlled or assisted ventilation.

Initial management should include adequate sedation, intubation in order to provide ventilation, maintenance of fluid balance with 0.9% saline, muscle relaxants as needed, anticonvulsant therapy, mannitol at 0.25 mg/kg bwt i.v. or dimethylsulphoxide (DMSO), glucocorticoids, nonsteroidal anti-inflammatory agents and barbiturates. Use of supplemental oxygen combined with hyperventilation will provide transient reduction of brain swelling. Hypothermia has been shown to have positive sparing effects in damaged brain tissue.

Initial therapy is dependent upon the signs demonstrated by the horse at the time of your examination. In horses that are aware and appropriate, you might wait until performing further diagnostic testing, such as radiographs or a CAT scan before initiation of treatment. In horses that are presented recumbent with obvious signs of recent trauma and/or an altered level of consciousness, aggressive treatment and potentially even surgical intervention to evacuate cerebral haemorrhage may be indicated.

One possible treatment is DMSO at a dose of 0.9–1.0 g/kg bwt i.v. (Reed 1987, 1994; Stewart 1987; Shi 2001). The proposed mechanism of action of DMSO is to reduce the formation of arachadonic acid metabolites such as thromboxanes and prostaglandins, resulting in stabilisation of membrane phospholipids (de la Torre et al. 1975; Rucker et al. 1981; Shi et al. 2001). In addition, DMSO may act as a scavenger of free radicals generated by peroxidation of lipid membranes in the CNS.

Barbiturates may have a protective effect against ischaemia following brain injury by lowering cerebral metabolism and by retarding peroxidation of lipids within brain cell membranes. Although an exact dose and frequency are not worked out in horses, phenobarbital at a dose of 5–10 mg/kg bwt i.v. given to effect may be useful.

Surgical decompression is controversial, although with the ability to evaluate horses using CAT scan and in some
locations even magnetic resonance imaging, surgical intervention may become routine. Indications are most appropriate when deterioration occurs during medical therapy. This regression is due to either cerebral oedema or haemorrhage, and craniotomy should be considered to either evacuate a haematoma or to allow swelling of the brain to occur unimpeded.

The treatment plan should include the use of broad-spectrum antimicrobial agents, sedatives and a mechanism for long-term feeding. The horses’ intestinal tract should be functioning normally. Therefore, the use of an indwelling nasogastric tube may be quite helpful and is especially indicated for foals, which have very small energy reserves.

**Manufacturer’s address**

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**References**


Whenever there is trauma sustained to the face, skull, neck, or back, neurological trauma should always be considered. It is important to note that neurological trauma can also pertain to peripheral nerves. Peripheral nerve injury should be considered in patients with extremity trauma (see Extremity Trauma Guideline). Emergency management of the head injured patient focuses on minimizing secondary brain injury, and thereby decreasing morbidity and mortality. Traumatic spinal cord injuries may occur as a result of direct blunt and/or penetrating trauma. The spinal cord and its exiting nerve roots may also be compressed as a result of a number of chronic degenerative processes such as osteoarthritis or degenerative disc disease. N.B. HEAD INJURY may not be associated with neurological deficits! Scalp injuries CLOSED â€“ contusion. OPEN Injuries tend to be focal â€“ external signs of trauma are frequently noted at site of contact. â€“ skull initially bends inward at point of contact (if force is sufficient, skull fracture can occur) â€“ cranium absorbs some of applied energy, while some energy is transmitted to brain by shock waves that travel and distort/disrupt intracranial contents. â€“ injury from compression requires significant force because skull â€“ treatment, and other emergency measures. â€“ importance of ABC â€“ ultimate outcome of brain injury is as much (or more) dependent on early. ABC as any other organ. Even moderate hypotension can convert reversible brain injury to irreversible ischemic brain damage. Gunshot Wound Head Trauma | American Association of Neurological Surgeons. www.aans.org/Patients. Page of. Gunshot wound head trauma patients are aggressively resuscitated upon initial arrival at the hospital. If blood pressure and oxygenation can be maintained, an urgent CT scan of the head is obtained. The decision to proceed with surgical treatment of the gunshot wound is based on the following factors. If a hematoma is confirmed by CT scan, an emergency craniotomy for clot evacuation, removal of debris and devitalized tissue may be performed. It is common for pressure to build up within the skull, so a cranieotomy (a procedure in which a large portion of the skull is temporarily removed to decrease pressure inside the skull) is also often performed. Introduction/Background Head trauma (i.e., head injury) is a significant public health concern and is a leading cause of morbidity and mortality in children and young adults. According to the Centers for Disease Control and Prevention, head trauma resulted in over 2.5 million emergency department (ED) visits in the United States in 2014 (63% increase from 2006) with nearly 290,000 hospitalizations and 57,000 deaths [1]. Common mechanisms of injury include falls, motor vehicle accidents, and acts of violence. Part II: Emergency Management of Severe, Moderate, and Minor Head Trauma. Even patients with an initial normal neurological examination and normal head CT can develop a hematoma requiring surgical intervention. Based on our review of current literature, most authors suggest routine CT scanning on all patients with GCS scores of 14 or less. For patients with GCS scores of 15, consideration may be given to the risk factors listed in Tables 1 and 2; however, a more specific recommendation cannot be offered at this time.