

Nursing the head trauma patient (part I)

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Figure 1: Patient who sustained head trauma as a result of a road traffic accident.

Head trauma patients are commonly seen within veterinary practice, and the treatment of these patients can prove challenging. In order to achieve a positive outcome in these cases, they require intensive treatment and nursing care. Nursing plays a vital role in monitoring these patients, and alerting the veterinary surgeon to any changes in their condition. The mainstays of therapy include intravenous fluids and hyperosmolar agents, with the administration of corticosteroids being somewhat outdated.

INTRODUCTION

Head trauma is commonly seen in veterinary emergency clinics. Animals sustain head trauma in numerous ways, including road traffic accidents, falling from heights, kicks from horses, being stepped on by owners (see Figure 1). Animals with head trauma required immediate medical attention. The patient may also have concurrent injuries, such as circulatory or respiratory problems, which need to be addressed during the initial treatment and stabilisation period.

There are two types of head trauma or brain injury: primary and secondary. Primary head trauma, such as a skull fracture or cerebral haemorrhage, describes the injury to the brain tissue from direct trauma and the forces applied to the brain at impact, these forces include acceleration, deceleration and rotational forces (Freeman and Platt, 2012). The brain is unable to tolerate these forces because of its composition and lack of internal support. Secondary head trauma occurs following the primary trauma. Following impact, a cascade of biomolecular events occur causing continued and progressive brain pathology. The presence of haematomas and oedema from the primary injury distorts normal brain parenchyma and decreases cerebral blood flow. In addition, a series of cellular reactions occur at the time of impact, and continue after the injury (Freeman and Platt, 2012). An important goal of treating head trauma is to limit or prevent secondary trauma.

BASIC ANATOMY

The brain is encased within the skull, which does not allow any room for inflammation or swelling. The skull cavity contains parenchymal tissue – the brain contents (80%), blood (10%) and cerebrospinal fluid ([CSF]; 10%). The main sections of the brain are the cerebrum, cerebellum, and brainstem.

PATHOPHYSIOLOGY

Like all organs, the function of the central nervous system

(CNS) is dependent on sufficient blood flow and oxygen and energy supply. The brain is a very active organ with a particularly high oxygen and energy demand (Sigrist, 2011). It consumes about 20% of the total body oxygen and more than 25% of the glucose (Sokoloff, 1981). Neurons are also not able to retrieve their energy anaerobically to a sufficient extent. Since the brain has only very limited storage capacity for glucose and oxygen, a minimal lack of energy can lead to brain damage.

Cerebral blood flow depends on the ratio between cerebral perfusion pressure (CPP), and the cardiovascular resistance. In a healthy brain, the cerebral perfusion pressure is monitored closely to maintain the oxygen and energy supply to the nerve cells (Guyton and Hall, 2000). Through this autoregulation, cerebral blood flow is kept constant despite changes in blood pressure and cerebral vascular resistance. Cerebral blood flow is effectively self-regulated, with cerebral perfusion pressure somewhere between 50-150mmHg (Sigrist, 2011). At CPPs above or below this range, the cerebral blood flow becomes directly proportional (Busija, 1980). In various diseases of the brain, including traumatic brain injury, autoregulation may be impaired focally or generally, which will also lead to direct dependence of cerebral blood flow of mean arterial pressure (MAP): $CPP = MAP - ICP$.

Intracranial pressure (ICP) is the pressure exerted between the skull and the intracranial tissues, normal intracranial pressure being 5-10mmHg (Bagley, 1996). Where there is inflammation or bleeding in the brain, intracranial venous blood and CSF are shunted into the body in an attempt to compensate for increasing intracranial pressure. If the body has done everything it can to compensate but the ICP continues to increase, intracranial hypertension (ICH) can develop. An increase in ICP results in decreased cerebral perfusion pressure (CPP) and cerebral blood flow, decreased oxygen flow to, and diminished supply of brain cells with oxygen and glucose. This leads to secondary changes and cell damage. Intracranial hypertension will lead to alterations in levels of consciousness, respiratory and circulatory abnormalities, and may cause death of the patient by brain herniation.

Severe, acute increases in ICP will trigger the Cushing reflex, a characteristic rise in MAP and reflex decrease in heart rate. Briefly, this occurs due to an initial drop in CPP caused by the increase in ICP at a given MAP. The resulting decrease in CPP triggers massive catecholamine release, which increases MAP, restoring CPP (Fletcher, 2012). The increase in MAP triggers baroreceptors in the carotid body and aortic arch, which causes reflex vagal stimulation, slowing the heart rate. The presence of the Cushing's Reflex in a patient with head trauma is a sign of a potentially life threatening increase in ICP and should be treated promptly (Fletcher, 2012).

INITIAL ASSESSMENT, DIAGNOSTICS AND MONITORING

Initial assessment should involve evaluation of the patient's respiratory and cardiovascular systems.

A Is the airway blocked? Ensure that the airway is free and

there is no debris or swelling in the oral cavity.

- B Is the patient breathing normally? If the patient is unconscious and has apnoea or dyspnoea, intubate the patient and perform manual ventilation, if needed.
- C How is the circulation? What are the heart rate, pulse rate and blood pressure? Ensure that the heart and pulse rates are synchronous. If there is a discrepancy, electrocardiography should be performed to check for arrhythmias. If the heart rate or blood pressure is not within normal limits, notify the veterinary surgeon. Emergency intervention, which may consist of fluid resuscitation and/or pain management, may be indicated (Terry, 2010).

PHYSICAL EXAMINATION

Once stable, a complete physical examination should be performed to document the patient's condition at presentation. Patients with severe head trauma can deteriorate quickly; therefore, it is extremely important to note all changes in the patient's condition. The head and neck should be manipulated minimally during the physical examination. Manipulation can displace fractures, worsen spinal cord injuries, or occlude the jugular vein, which can decrease venous return from the brain and, in turn, increase ICP, leading to ICH. Patients with head trauma should receive supplemental oxygen until proper oxygenation is confirmed.

A physical examination should start with the assessment of the patient's level of consciousness, changes to which can reveal the severity and progression of the injury. The levels of consciousness are as follows:

- Alert and responsive – the patient exhibits normal behaviour;
- Obtunded – the patient is awake but responds less to stimuli;
- Stuporous – the patient responds only to painful/noxious stimuli;
- Comatose – the patient is unconscious and does not respond to any stimuli (Sigrist, 2011).

Examination of the eyes can provide important information about the severity of brain injury. Any deviation from the normal eye position is called strabismus, which usually is caused by damage to the cranial nerves or brainstem (Terry, 2010).

It is extremely important to check normal eye position. Rhythmic eye movement that is vertical, rotary or horizontal, fast or slow, is called nystagmus. Physiologic nystagmus (or oculocephalic reflex) can be initiated in healthy patients by moving the head horizontally or vertically, resulting in rapid eye movement (also called fast phase) towards where the head is positioned (Terry, 2010). Absence of physiologic nystagmus indicates severe brainstem damage and correlates with a poor prognosis (MacIntyre et al, 2005). Any other type of nystagmus is considered abnormal.

In addition, the pupils' response to light (the pupillary light reflex (PLR)) should be assessed. Shining a bright light into the eyes should cause the pupils to constrict; they should dilate when the light is removed. A slow PLR suggests a



Figure 2: Feline patient with anisocoria, as a result of head trauma (RTA).

guarded to poor response (MacIntyre et al, 2005). Absence of a PLR suggests a grave response. Continual monitoring of the PLR can help assess the ICP.

Checking the size of the pupils is also important. With head trauma, the pupils can be normal, constricted (miosis), dilated (mydriasis), or asymmetric (anisocoria). Miotic (pinpoint) pupils are usually due to cerebral injury or oedema, indicating a guarded to fair prognosis, but they can be associated with ocular causes, such as ocular injury, so this cause should be investigated (Platt, 2012). Mydriasis can be associated with stress, medications, ophthalmic disease, decreased cerebral perfusion and impending cardiopulmonary arrest, but also may indicate permanent midbrain damage or brain herniation and is associated with a poor prognosis. Mydriasis is not directly associated with brain injury but is important to recognise in debilitated patients because it can be associated with cardiopulmonary arrest. Progression from miosis to mydriasis indicates deteriorating neurological status and is an indication for immediate, aggressive therapy (Platt, 2012).

Anisocoria has several causes, including oculomotor nerve damage or compression, direct eye injury, and uveitis – inflammation of the uvea – (Terry, 2010). Mid-size pupils that are unresponsive to light usually indicate a brainstem injury and grave prognosis. Changes in the pupils' size should be closely monitored and recorded in the medical history/chart. The menace response is the involuntary blink of the eyelids in response to movement towards the eyes. If the menace response is intact, the patient will then blink when something approaches the face, indicating sight. The staff member who evaluates this response should be careful not to move air towards the face, which can cause the eyes

to blink, possibly leading to a false-positive test result. Neonates can be difficult to assess because they may not yet have developed a menace response (Terry, 2010). Blindness may indicate that either the nerves to the eyes and brain are too inflamed to work or there is a problem with the eye(s). Vision problems usually indicate major nerve problems with the head. Treatment decisions are not made based only on blindness. In many cases of blindness due to trauma, vision can be restored (Terry, 2010).

The patient's body position can be used to help the veterinary surgeon determine the severity of the brain injury and the prognosis. In a position called opisthotonus or decerebrate rigidity, the patient is recumbent and comatose with all limbs rigidly extended, and the head back. Opisthotonus indicates severe brainstem injury and usually carries a grave prognosis (Sturges and LeCouter, 2009). Decerebellate posture (see Figure 2), which has a more favourable prognosis than decerebrate rigidity, may indicate an acute cerebellar lesion or herniation. (MacIntyre et al, 2005).

In decerebellate posture, the patient's forelimbs are extended and hind limbs flexed. Patients with this posture are usually conscious and have responsive pupils. Close monitoring of the patient is important because subtle changes in posture can indicate progression of the injury. Schiff-Sherrington syndrome, which may appear similar to decerebrate posture, is characterised by extended, rigid forelimbs and paralysed, flaccid hind limbs. This syndrome indicates a thoracolumbar spinal lesion. It is extremely important to carefully assess the patient to avoid confusion and misdiagnosis (Terry, 2010).

NEUROLOGICAL ASSESSMENT

The Modified Glasgow Coma Scale score (MGCS) is a quantitative measure that has been shown to be associated with survival to 48 hours in dogs with TBI (Fletcher, 2010), and provides a score that can be used to assess initial neurologic status as well as progression of signs. This scale incorporates three domains: level of consciousness, posture, and pupillary size/response to light, with a score of one assigned to each domain. The final score ranges from three to 18, with lower scores indicating more severe neurologic deficits. The initial neurological examination should be interpreted bearing in mind the patient's systemic status, as shock can cause significant neurologic dysfunction (Fletcher, 2012).

