Head Trauma and Traumatic Brain Injury

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Physiology of the Normal Brain

Neuronal function is essential to ongoing life. The loss of neuronal function in the course of injury or illness therefore represents a major loss. Neurological damage from any cause may result in a progression from a period of elevated intracranial pressure, resulting in a reduction in cerebral perfusion pressure, and ultimately compromising cerebral blood flow sufficient to produce permanent neuron loss.

In the normal animal, the brain receives 14-15% of resting cardiac output, and uses 15-20% of the body's oxygen consumption. Blood flow to gray matter is higher than to white matter. The brain has a high resting energy requirement, and no energy or oxygen stores. This makes the brain extremely susceptible to injury following events resulting in cellular damage, or disruption to normal oxygen delivery and metabolism within the cranial vault.

Cerebral blood flow is determined by five factors

1. PCO_2 – A change in cerebral CO_2 is the main and most sensitive regulator of cerebral blood flow. Carbon dioxide is a potent cerebral vasodilator, and leads to an increase in cerebral blood volume.
2. PO_2 – normal oxygen tension in cerebral circulation aids in maintaining mild vasoconstriction in cerebral blood vessels.
3. Blood pressure autoregulation – The cerebral circulation is regulated in such a way that a constant total cerebral blood flow is maintained under varying conditions. This is called autoregulation. The factors affecting total cerebral blood flow are:
   (a) Arterial blood pressure at the brain level.
   (b) Venous pressure at the brain level.
   (c) Intracranial pressure.
   (d) The viscosity of the blood.
   (e) The degree of active constriction or dilatation of cerebral arterioles. Cerebral arteriolar tone is determined by products of cerebral metabolism, vasomotor nerves, angiotensin, and by substances produced by the vascular endothelium.

   Autoregulation maintains cerebral blood flow constant between a systolic arterial blood pressure of between 50-150 mmHg, outside of which, cerebral blood flow becomes linearly related to blood pressure. Autoregulation is less efficient in states of ischaemia, hypoxia, hypercabia, and increasing blood viscosity.
4. The level of neuronal stimulation – Increasing neuronal activity and hence metabolic rate will increase cerebral blood flow, for example, with seizures, hyperthermia etc. As with autoregulation, this relationship is uncoupled with in conditions of trauma, infarction, subarachnoid hemorrhage, and cardiac arrest.
5. Systolic blood pressure

From the above, it may be seen that disruption to cerebral blood flow will be altered under conditions of head trauma, and traumatic brain injury, as trauma will alter, carbon dioxide concentrations, oxygen concentrations, blood pressure autoregulation, the level of neuronal stimulation, and systolic blood pressure. The aim of much of our treatment of brain trauma therefore, concentrates on restoring and maintaining cerebral blood flow.

Pathophysiology of Brain Trauma

Brain injury may be conceptually divided into primary and secondary injury. Primary brain injury occurs immediately following brain tissue impact, and initiates multiple cascades that result in secondary brain injury. Primary and secondary processes associated with brain injury in dogs and cats are outlined in the following table.

Primary and Secondary Processes Associated with Brain Injury

Primary Brain Injury
• Direct damage to brain parenchyma • Direct damage to blood vessels
Secondary Brain Injury
• ATP depletion • Intracellular accumulation of sodium and calcium • Increased cytokine production • Elevated extracellular glutamate • Oxygen free radical production • Lactic acidosis • Nitric Oxide accumulation • Arachadonic acid, kinin, complement, coagulation and fibrinolytic pathway activation

The progression from primary to secondary brain injury is described below:

1. **Trauma induced neuronal damage** – neuronal damage that is directly cause by trauma includes physical damage to neurons, including axons, cell body injury, damage to supporting structures (Perkinje cells, blood vessels), compression from hematomas (epidural, subdural, subarachnoid, parenchymal), and damage to blood vessels.

2. **Ischemic damage** – neuronal and vascular damage in the brain leads to brain oxygen deprivation. Oxygen deprivation leads to anaerobic glycolysis and ATP depletion. ATP depletion results in intracellular flux of calcium, sodium and water, and intracellular loss of potassium. Increases in intracellular calcium results in activation of intracellular phospholipases, proteases, xanthine oxidase, and nitric oxide synthase, leading to cell protein and phospholipid degradation, arachadonic acid and free radical production and cell death. Anaerobic glycolysis also results in production of lactic acid, which lowers intracellular pH. Cellular acidosis compromises cell functions, including mitochondrial ATP production and may enhance free radical formation. Ischemia of brain tissue is significant in most patients during the first 24 hours following traumatic brain injury, and has a significant impact on neurological outcome.

3. Both of these mechanisms can lead to edema of the brain parenchyma, which peaks at 24-48 hours post trauma. Brain edema may be classified as vasogenic, cytotoxic, and interstitial.

   (a) **Vasogenic edema** occurs secondary to increased vascular permeability, or increased intravascular pressures. This leads to compromise of the blood-brain barrier through disruption of endothelial tight junctions.

   (b) **Cytotoxic edema** occurs secondary to hypoxia and impaired cellular function and metabolism as outlined above.

   (c) **Interstitial edema** occurs when fluid shifts from the ventricles into the peri-ventricular interstitium due to obstruction of cerebrospinal fluid outflow (obstructive hydrocephalus)

Increased Intracranial Pressure

Following head trauma, the primary and secondary events involved in brain injury – direct trauma, ischemia, and edema, in combination with alterations in carbon dioxide, oxygen, and blood flow dynamics, lead to an elevation in brain volume, and increased intracranial pressure.

Most patients with head trauma develop increased intracranial pressure, the clinical signs of which usually complicate measures to localize and monitor the degree of brain injury. The Monro-Kellie doctrine states that within the confines of the inelastic skull, blood (10%), cerebrospinal fluid (10%), and brain parenchyma (80%) normally exist in equilibrium with each maintaining a stable volume and pressure. Sudden increases in brain parenchyma, blood or cerebrospinal fluid pressure or volume do not allow time for equilibrium and adjustment, leading to increased intracranial pressure. If severe enough, increased intracranial pressure can result in herniation of the falx cerebri, tentorium, cerebellum; or skull fractures. Most cases of parenchymal herniation are fatal.

Causes of increased intracranial pressure

- Hemorrhage.
- Cerebral edema.
- Arterial and venous dilatation – increasing tissue carbon dioxide will increase arteriolar dilatation.
- Increasing cerebral metabolic rate – results in increased brain production of carbon dioxide, which results in cerebral vasodilatation.
- Fever, seizures and some anesthetic agents (halothane will cause vasodilatation).
- Concurrent disease states such as pulmonary disease, hemorrhage etc, alter gas exchange.

In summary, elevated carbon dioxide concentrations, decreased oxygen concentrations, and increased cerebral metabolic rate are present in the patient with head trauma, and will all increase cerebral blood volume. Cerebral edema and hemorrhage lead to an increase in brain parenchymal mass. The net result is a rapid rise in intracranial volume, and elevated intracranial pressure.

When intracranial pressure becomes elevated, the cerebral blood flow reduces due to compression of blood vessels within the cranial vault. The resultant ischemia stimulates the vasomotor centre of the brain, which raises systemic blood pressure, the so-called “Cushing

reflex". Over a considerable range, the rise in systemic blood pressure is proportionate to the rise in intracranial pressure, thereby maintaining blood flow to the brain. However, when intracranial pressure exceeds the arterial pressure, cerebral circulation ceases.

Treatment of the Patient with Head Trauma

1. First Aid recommendations

(a) Initial emergency primary life support (ABC) at the scene

(b) Minimize patient movement during transport

(c) Place animal on board before transport to clinic

2. Airway – Ensure the patient has a patent airway

- Airway protection is vital. Early onset pneumonia due to aspiration of foreign material, mucus, blood and regurgitated contents worsens neurological outcome. Early intervention to protect an airway is essential to minimize the risk of aspiration and pneumonia.
- Provide oxygen by cage, flow past, mask or ET tube. The aim is to supply 100% oxygen on presentation.
- Avoid nasal oxygen as sneezing increases intracranial pressure, and the possibility of basilar skull fractures.
- Assess oral and nasal cavity for the presence of fractures, palatine defects, and hemorrhage. Control hemorrhage using pressure, topical adrenaline or cautery.
- Intubation and mild elevation of the head reduces chance of aspiration of gastric and oral secretions, blood and debris. If the patient requires intubation, let head lie on table, and elevate maxilla only to avoid occlusion of the jugular veins, and elevation of intracranial pressure. Hyperextension of the head during intubation can cause injury to blood vessels at the base of the brain, and can cause intracranial hemorrhage.
- If an anesthetic induction agent is required to facilitate intubation, propofol is the agent of choice. Propofol does not disrupt brain metabolism – blood flow coupling, thereby minimizing the risk of additional neurological damage.

3. Breathing

- If the patient is comatose, or semi-comatose, induce anesthesia with propofol (if required), intubate, and provide assisted ventilation. Ventilate the patient to achieve an end-tidal CO_2 of 26-32 mmHg. This aids in prevention of excessive carbon dioxide associated vasodilatation in the brain, as well as avoiding excessive vasoconstriction in the brain associated with hypocapnea. The patient should remain on 100% oxygen at all times for up to 2 hours post presentation.
- If patient is conscious and ventilating adequately – provide oxygen. If patient is not ventilating adequately, anesthetize the patient and provide ventilatory assistance.
- Oxygenation assessment using pulse oximetry can be misleading; the following table is used as a guide.

Interpretation of Pulse Oximeter Values

SaO_2	PaO_2	Interpretations
>95% <89% 75%	>80% <60% <40%	Normal Serious Hypoxemia Lethal Hypoxemia

4. Circulation

- **Treat hypovolemic shock or intravascular volume deficits using an isotonic crystalloid solution** such as Hartman's, using a technique of small volume resuscitation, to avoid excessive fluid accumulation in the brain. Administer boluses of Hartman's at 10-20 ml/kg IV over 10 minutes, and reassess the requirement for ongoing rapid fluid administration, monitoring heart rate, respiratory rate, blood pressure, and mucus membrane characteristics to determine the appropriate end-point for intravascular volume resuscitation. Once the patient is hemodynamically stable; begin maintenance fluid rates, using isotonic fluid therapy.
- The aim of all circulatory support is to maintain cerebral perfusion pressure ***at least*** above 60 mmHg (which means maintaining a mean arterial pressure above 70 mmHg) – below this level, brain blood flow is inadequate, and results in cerebral hypoxia and ischaemia. In humans, sustained systolic blood pressures below 90 mmHg are associated with neurological outcome that is two times worse, than in patients with normal systolic blood pressure following intravascular volume resuscitation. Therefore, it is vital that we re-examine our patients to ensure they have cardiac indices (heart rate, blood pressure, mucus membrane color and refill) within normal limits to minimize the chances of worsening neurological outcome.
- Isotonic crystalloids are widely used and are currently recommended as the fluid of choice. There is no significant difference in survival rates when isotonic crystalloids are used in volume resuscitation when compared to hypertonic saline and colloid administration, despite a theoretical benefit with the use of hypertonic saline in combination with dextrans/pentaspan. However, hypertonic saline may lead to a compromise in brain function, and areas of focal damage, as sodium is the major osmotic agent contributing to brain edema. In addition, recent studies indicate that although fluids of high osmolality in the brain may delay the onset of peak traumatic brain edema following brain injury, they generate a high osmotic potential in the brain, and eventually contribute to massive brain edema, and ultimately a worsening of neurological outcome.
- Hypotonic fluids such as 0.45% NaCl, or 5% glucose, will allow excess water to diffuse across the blood brain barrier when plasma osmolality is reduced, and will contribute to brain edema. They are not recommended.
- Patients are usually hyperglycemic following head trauma, the magnitude of which is directly proportional to the severity of brain damage. Hyperglycemia can potentiate neurological damage in head trauma, by increasing the production of cell breakdown products and cytotoxic mediators of inflammation. Glucose-containing fluids should be avoided.
- Elevate the head no more than 30 deg above the level of the heart to aid in increasing venous drainage from the head, and reducing intracranial pressure. Elevation greater than 30° may lead to jugular vein occlusion, and increases in intracranial pressure.

5. Control seizures with intravenous diazepam to effect

Propofol or phenobarbitone may be used in patients with seizures refractory to diazepam administration. The presence of seizures is strongly suggestive of intracranial hemorrhage. Note that the presence of nausea is also supportive of intracranial hemorrhage. Early recognition of symptoms such as nausea, vomiting, and seizures permits early detection of patients at risk for rapid deterioration. Administration of anti-epileptic medications and anti-nausea medications early in head trauma may reduce the risk of worsening neurological injury.

6. Perform an Initial Neurological Examination

- Perform a neurological evaluation using the Small Animal Coma Scale to determine the severity of neurological deficits on presentation.
- Evaluation of the Small Animal Coma Scale can aid in identifying patients that will require intubation and ventilatory support, outcome, and those patients that may require additional therapies during re-institution of feeding.

Small Animal Coma Scale

Motor Activity	
Normal gait, normal spinal reflexes	6
Hemiparesis, tetraparesis, or decerebrate rigidity	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity with opisthotonos	2
Recumbent, hypotonia of muscles, depressed/absent spinal reflexes	1
Brain Stem Reflexes	
Normal pupillary light reflexes, and oculocephalic reflexes	6
Slow PLR, with normal to reduced oculocephalic reflexes	5
Bilateral miosis, normal to reduced oculocephalic reflexes	4
Pinpoint pupils, reduced to absent oculocephalic reflexes	3
Unilateral unresponsive mydriasis, reduced/absent oculocephalic reflexes	2
Bilateral unresponsive mydriasis, reduced/absent oculocephalic reflexes	1
Level of Consciousness	
Occasional periods of alertness, responsive to environment	6
Depression/delirium, capable of responding to environment but response may be inappropriate	5
Semi-comatose, responsive to visual stimuli	4
Semi-comatose, responsive to auditory stimuli	3
Semi-comatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1

An improving coma score indicates response to therapy; static or decreasing scores may dictate further therapy is required.

A patient having a coma score less than seven is deemed as having severe neurological injury; a coma score between seven and twelve is deemed to have moderate neuro-

logical injury and a patient with a score above twelve as having mild neurological injury. Prognosis should be based on the results of several neurological examinations, localization of the neurological abnormalities, and the presence of concurrent medical and surgical problems with the patient. A single coma score should not be used as justification for euthanasia.

- **Localize the lesion** to one of the five (5) brain regions as follows:

(a) Telencephalon – cerebral cortex, cerebral white matter, basal nuclei

(i) Abnormal behavior, depression, seizures, head pressing, pacing.
(ii) Posture normal.
(iii) Gait normal, mild ipsilateral hemiparesis.
(iv) Contra-lateral postural deficits.
(v) Cranial nerves normal, possible contra-lateral vision impairment (cortical blindness, with normal pupillary light reflexes (PLR).

(b) Diencephalon – thalamus, hypothalamus

(i) Altered mental status, aggression, disorientation; disordered endocrine and autonomic functions.
(ii) Posture normal.
(iii) Gait normal to hemi- to tetra-paresis (UMN signs).
(iv) Contra-lateral postural deficits.
(v) Cranial nerve deficit CN II (Optic) no PLR, menace ipsilateral side.

(c) Mesencephalon (midbrain), Metencephalon (pons), Myencephalon (medulla oblongata)

(i) Altered mental status – depression, stupor, coma.
(ii) Posture normal; possibly turning and falling.
(iii) Gait – ipsilateral hemiparesis (unilateral lesion) or spastic tetraparesis (bilateral lesion), ataxia, grossly abnormal gait.
(iv) Ipsilateral or contra-lateral postural deficits.
(v) Cranial nerve deficits may be present in CN III-XII.
(vi) Respiratory pattern may vary from bradypnea, hyperventilation, agonal, or Cheyne-Stokes respiration.

(d) Metencephalon (cerebellum)

(i) Mental status normal.
(ii) Posture normal.
(iii) Gait – tremors, dysmetria, ataxia.
(iv) Postural reactions normal, possibly dysmetria.
(v) Cranial nerves normal, possibly reduced menace reaction ipsilateral to lesion, nystagmus.

(e) Vestibular

(i) Mental status normal.
(ii) Posture – head tilt.
(iii) Gait – normal to ataxia, hemiparesis if central vestibular.
(iv) Posture – ipsilateral or contra-lateral deficits.
(v) Cranial nerve deficits may be affected, CN III, V, VII, nystagmus may be present

7. **Relieve anxiety and pain.** Drugs used to relieve anxiety and pain includes sedatives, and narcotic agents. The most widely used include a combination of butorphanol and diazepam, or fentanyl and midazolam. These drugs can facilitate intubation during the provision of ventilatory support, aid in reducing sustained activation of the neuro-hormonal response to stress, and decrease cerebral oxygen demand. However, they may compromise cerebral oxygen delivery via their cardiovascular depressant effects. Opioids decrease brain sensitivity to carbon dioxide, which results in cerebral vasodilatation, and increasing brain volume. Ventilatory effort and effectiveness needs to be closely monitored following sedation with opiates. Provision of supplemental oxygen, intubation and ventilatory assistance is recommended if patients respiratory or neurological status deteriorates following analgesic therapy.

8. **Diuretics** are administered once the patient is hemodynamically stable. **Mannitol** is a small molecular weight (700Da) substance that is present in a hypertonic solution. It is large enough, that, in the normal brain, it does not cross the blood-brain barrier, and therefore, when administered intravenously, exerts a strong osmotic stimulus for movement of fluid from the intracellular and interstitial spaces in the brain, into the systemic circulation.

    - Give mannitol following correction of hypovolemia.
    - The small particle size of mannitol decreases blood viscosity, and increases blood flow through the brain, assisting oxygen delivery. This in turn, results in arteriolar vasoconstriction in the brain, and decreased intracranial pressure. Mannitol also reduces CSF production, scavenges free radicals, and produces an osmotic transfer of fluid from the interstitial and intracellular compartments to the intravascular space.
    - Mannitol decreases intracranial pressure in normal patients primarily due to reflex vasoconstriction, a reflex that occurs within a few minutes. Osmotic shift effects are seen approximately 15 minutes following administration.
    - Dose: 0.25-1 gm/kg, up to 2 ml/min (20% solution) q 4 hrs; give no more than 3 doses in any 24 hr period to avoid hyperosmolar syndromes.
    - Maximum effect occurs 15-30 minutes following administration; persists for 3-4 hours.
    - Indications include deteriorating neurological status, depressed LOC on presentation.
    - Administration of furosemide at 2 mg/kg IV a few minutes prior to mannitol administration may be synergistic in reducing intracranial pressure.
    - If patients neurological status deteriorates following mannitol administration, repeated doses should be withheld.

9. **Corticosteroids** are of no proven benefit patients with traumatic brain injury. They may cause hyperglycemia, and gastrointestinal ulceration. Methylprednisolone sodium succinate may be considered in patients not responding to conventional therapy that are not hyperglycemic, or in patients with concurrent spinal trauma. Give intravenously at 30 mg/kg, and repeat at 15 mg/kg at 2 hrs and 6 hrs following the initial dose.

Monitoring the Patient with Head Trauma – The First 24 Hours

Patients with traumatic brain injury following head trauma are critical patients that require frequent monitoring and reassessment. Treatment plans frequently need to be re-evaluated in light of the findings of serial clinical examinations, including TPR assessment, neurological assessment, coma score, and the presence of concurrent medical and surgical conditions associated with trauma.

In general, the following rules apply to the monitoring of the patient with traumatic brain injury:

1. Repeat primary survey q 10-30 minutes, depending on the patient status, until the patient is stable.
2. Monitor PCV/TP and glucose q 30-60 minutes until the patient is stable.
3. Perform secondary survey/thorough clinical evaluation of the patient q 30-60 minutes as for any patient with trauma.
4. Perform coma scale evaluation q 30 minutes, until the patient is showing signs of continued improvement.
5. Re-evaluate the treatment plan following each clinical and neurological assessment to ensure the most appropriate treatment is being given.
6. Fluid therapy – should continue following initial volume resuscitation at a rate of 1.5-2 x maintenance levels. The fluid of choice for the first 24 hours is LRS. Monitoring of systolic blood pressure is preferred, in order to better tailor a fluid therapy plan. Maintain systolic blood pressure above 90 mmHg, and preferably in the normal range (110-140 mm Hg).
7. Nursing care includes turning the patient q 2 hrs, nutrition, enemas, and assisted bowel function. Physiotherapy, limb massage, and passive limb flexion should also be used to minimize muscle contracture and wasting, and ischemic damage to gravity dependant limbs.
8. Nutrition – In head trauma, the functional gut-brain link is altered, causing gastric dysrhythmias, nausea, vomiting and intolerance to feeding. Administrations of prokinetic drugs such as metoclopramide are advised in all patients with head trauma. Begin nutrition with a semi-liquid diet via syringe, or esophagostomy or gastrotomy tube within 12-24 hrs of trauma.

The Deteriorating Patient – What To Do

Continued evaluation of the patient with head trauma and traumatic brain injury is an essential component of therapy. If the patient continues to show signs of neurological deterioration despite therapy, it is necessary to follow a methodical plan in an effort to determine the cause of the deterioration, and effect a timely therapeutic plan for intervention.

- **Assess the patient's respiratory function** – ensure the patient has a patent airway; establish a patent airway. Assess respiratory rate, effort, and effectiveness by visual examination, mucus membrane color, and pulse oximetry if available. Provide ventilatory assistance by oxygen supplementation and manual ventilation if required.
- **Assess the patient's cardiovascular function** – as outlined above, poor tissue perfusion is a major contributing factor in poor neurological outcome. Assess mucus membrane color

and refill time, pulses, blood pressure (if available), heart rate, and body temperature. Symptoms of persistent hypovolemia include elevated heart rate, poor pulse quality, low peripheral blood pressure, poor capillary refill time, and low body temperature. These symptoms may be present due to:

  - Inadequate volume resuscitation – treat by administering intravenous fluid therapy in a pulsatile, low-volume manner as described above. It is important to remember that administration of diuretics such as mannitol and furosemide can result in blood volume depletion. Frequent evaluation of the patient is required following diuretic therapy in order to prevent this complication.
  - The presence of pleural space disease – perform a chest tap on both sides of the chest to rule out the presence of a pneumothorax, hemothorax, or tension pneumothorax. Manage as required.
  - The presence of blood loss into the abdominal cavity, thoracic cavity, or long bone fracture sites. Manage as required – drain pleural fluid, abdominal compression bandage, splint and immobilize fractures.
  - The presence of pain – manage with adequate analgesia and/or sedation.
  - The presence of hypothermia – decreases vascular tone (see below).

- **Evaluate neurological status** – deteriorating neurological status despite adequate airway, breathing and circulation is an indication for further administration of diuretic therapy with mannitol and/or furosemide. Failure to respond to further diuretic therapy should prompt the clinician to consider ancillary therapy.

Other therapy that may be considered includes hyperventilation to achieve an end tidal carbon dioxide concentration of 26-32 mmHg, and hypothermia to decrease cerebral oxygen requirements.

Induction of a barbituate coma may be detrimental to survival due to the induction of hypotension and hypoventilation and is generally avoided.

Therapeutic hypothermia (cooling to a rectal temperature of 32°C has been used in several human studies in an effort to reduce cerebral metabolic rate, reduce brain oxygen supplementation, and assist neurological recovery. Therapeutic hypothermia however, may result in an increased incidence of infection (especially respiratory) due to a reduction in expression of proteins involved in innate immunity, and a reduction in the effectiveness of hemodynamic therapy with intravenous fluids, due to a reduction in vascular tone. These two factors may contribute to increased morbidity and a worsening of neurological outcome. Patients with hypothermia require more intensive monitoring of their blood pressure, and respiratory function, to ensure that benefit is obtained from the procedure.

Surgery or cranial decompression, including durotomy may be used to relieve intracranial pressure in patients not responsive to medical therapy. Neurosurgical therapy aims to minimize secondary brain damage through evacuation of an intracranial space occupied by a blood clot or hemorrhage, and the reduction of intracranial volumes and pressure. The prognosis after decompression depends on the clinical signs and symptoms on admission, the patients age, and the existence of major extra-cranial injuries. Patients having brainstem injury, and patients having sustained trauma greater than 48 hours prior to intended surgery are poor candidates. Interestingly, the presence of skull fractures can improve neurological outcome by lowering of intracranial pressure.

Experimental therapies include administration of mexiletine and gingko biloba extracts – both of which have been shown to reduce lipid peroxidation and brain edema following traumatic brain injury in rats.

Serum magnesium levels have been shown to correlate well with traumatic brain injury, with a graded decrease in serum magnesium concentrations, with increasing severity of brain injury. It is unclear whether administration of magnesium in traumatic brain injury improves neurological outcome.

Insulin-like growth factor-1 (IGF-1) administration following head trauma has been shown to reduce cell stress and damage, and apoptosis (programmed cell death), as well as attenuating post-trauma weight loss and anorexia following had trauma in rats, and may emerge as an important therapeutic modality in coming years.

Prognosis of the Patient with Head Trauma

- Cerebral, cerebellum injuries carry a better prognosis than brainstem lesions.
- Clinical progress may take up to 4 weeks.
- Seizures are the only long term possible complication.

The Eye in the Patient with Head Trauma

The ocular examination is an important part of the evaluation of the patient with head trauma. It is frequently the only "window" we have to assess intracranial pressure in veterinary medicine. It is therefore important to know what various changes in ocular appearance mean. The following is a guide to ocular findings in traumatic brain injury:

Ocular Finding	**Clinical Significance**
Normal pupil size, normal pupillary light reflexes	Normal
Small to pinpoint pupils, responsive to light	Ocular trauma, cerebral trauma; prognosis fair to good
Small to pinpoint pupils, non-responsive to light	Midbrain lesion (Mesencephalon); pontine injury (Metencephalon); prognosis guarded
Bilateral dilated pupils, fixed and non-responsive	Occulomotor nucleus injury due to hemorrhage or compression; prognosis grave
Unilateral dilated, non-responsive pupil	Unilateral occulomotor compression
Nystagmus (requires functioning of CN III, IV, VI)	Cerebellar, pontine, or vestibular contusion; brainstem lesion
Unilateral medial strabismus	Compression on CN VI; high pontine injury

The Cranial Nerve Examination

Cranial Nerve	Origin	Function	Test	Normal Response	Abnormal Response
CN 1 Olfactory	Pyriform Cortex	Sense of smell	Smelling of non-irritating volatile substances	Behavioral reaction – interest or aversion	No reaction
CN 2 Optic	Lateral geniculate nucleus (vision); pretectal nucleus (PLR)	Vision, PLR	Menace, obstacle test, visual placing, following movement, PLR, ophthalmoscopy	Blinks, avoids objects, visual placing, PLR, ophthalmoscopy normal	No blink, poor avoidance, no visual placing, direct PLR absent, retina or optic disc abnormal
CN 3 Occulomotor	Midbrain tegmentum	Constriction of pupil, dorsal, ventral, and medial rectus muscles, ventral oblique muscle	PLR, pupil size, eye position, eye movement	Pupils symmetrical, PLR, eyes central in palpebral fissure	Mydriasis, ipsilateral; no direct PLR, ventrolateral strabismus, no movement except laterally
CN 4 Trochlear	Midbrain tegmentum	Dorsal oblique muscle – rotates eye position ventro-medially	Eye position or movements	Eye central, moves in all directions	Abnormal position of retinal vessels
CN 5 Trigeminal	Pons (motor); Pons, medulla, C1 spinal cord segment (sensory)	Motor – muscles of mastication; Sensory – face rostral to ears	Motor – ability to close mouth; Sensory – palpebral reflex, pinch face	Closed mouth, good jaw tone; good response to noxious stimulation, palpebral reflex intact	Jaw hangs open, poor jaw tone; atrophy of masticatory muscles; loss of palpebral, poor facial sensation
CN 6 Abducent	Medulla oblongata	Lateral rectus and retractor muscles; lateral movement of eye, retraction of the globe	Eye movements, eye position	Eye centered in palpebral fissure; moves laterally and retracts	Medial strabismus; lack of lateral eye movements
CN 7 Facial	Medullar oblongata	Muscles of facial expression	Facial symmetry, palpebral reflex	Normal facial movements	Asymmetry of the face, deviation of nasal philtrum, reduced palpebral
CN 8 Vestibulocochlear	Vestibular nucleus, medulla oblongata	Equilibrium, hearing	Posture, gag, eye movements, rotatory and caloric tests, startle test	Normal posture and gait, brief post-rotatory nystagmus, positive startle test	Head tilt, head twist, circling, prolonged or absent post-rotatory nystagmus; poor startle response
CN 9 Glossopharyngeal	Medulla oblongata	Sensory and motor to pharynx and palate, salivary glands	Gag reflex	Swallowing	Poor gag reflex, dysphagia
CN 10 Vagus	Medulla oblongata	Sensory and motor to pharynx and larynx, thorax and abdominal viscera	Gag reflex, laryngeal reflex, oculocadiac reflex	Swallowing, cough, bradycardia	Poor gag reflex, dysphagia, inspiratory dyspnea, no retraction of laryngeal folds
CN 11 Accessory	Medulla oblongata, and cervical spinal cord	Trapezius, sternocephalicus and brachycephalicus muscles	Palpate for atrophy of muscles; EMG	Normal muscles	Atrophied muscles; denervation
CN 12 Hypoglossal	Medulla oblongata	Movements of the tongue	Protrusion of tongue, retraction of tongue	Strong withdrawal of tongue, able to lick in all directions	Tongue deviates to one side, atrophy, weak withdrawal

Divisions of the Central Nervous System – An Aid in Localizing Brain Lesions

Major Structures	Functions
Telencephalon	
• Cerebral cortex	
– Frontal lobe	• Intellect, behavior, fine motor activity
– Parietal lobe	• Touch, pain, propioception
– Occipital lobe	• Vision
– Temporal lobe	• Behavior, hearing
• Basal nuclei	• Muscle tone, initiation and control of motor
Diencephalon	
• Hypothalamus and Pituitary gland	• Autonomic control, appetite, thirst, temperature control, electrolyte balance, sleep, behavior, endocrine functions
• Thalamus	• Touch, pain, propioception, reticular activating system
• Olfactory system	• Olfaction
• Optic chiasm	• Vision, PLR
Mesencephalon	
• Midbrain – reticular activating system	• Consciousness and sleep
• Occulomotor nucleus	• Extra-ocular muscles, pupil constriction
• Trochlear nucleus	• Extra-ocular muscle
• Red nucleus	• Motor activity, origin of rubrospinal tract
• Ascending and descending tracts	• Motor and sensory
Metencephalon	
• Pons – trigeminal, reticular activating system, ascending and descending tracts	• Sensation and motor to mastication, vital centres – respiration, motor and sensory
• Cerebellum	• Coordination of movement and muscle tone, equilibrium
Myencephalon	
• Medulla oblongata – CN 6-12	• See cranial nerves table
Spinal cord	
• Gray matter	
– Dorsal horn	• Sensory neurons and reflexes
– Ventral horn	• Lower motor neurons and reflexes
– Intermediolateral	• Autonomic neurons
• White matter	
– Spinocerebellar tracts	• Unconscious propioception
– Dorsal columns	• Conscious propioception, sharp pain
– Spinothalamic tracts	• Pain and temperature
– Propriospinal tracts	• Communication between spinal segments
– Rubrospinal tracts	• Voluntary motor
– Corticospinal tracts	• Voluntary motor – fine movements
– Vestibulospinal tracts	• Posture motor
– Reticulospinal tracts	• Posture motor, some voluntary motor

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