

# HEAD TRAUMA

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*The following is a lecture outline for advanced veterinary technicians discussing head trauma in the veterinary patient. The outline reviews the anatomy & physiology of the normal head and one which has been traumatized, and discusses in depth the principles of diagnosing and treating types of head trauma. Detailed information is given with respect to monitoring and nursing care required to successfully treat the head trauma patient. By understanding the physiological changes induced by the injury, the monitoring parameters, and the treatments available, the veterinary technician becomes one of the most important team members contributing to a patient's successful recovery from head trauma. The information is technical and detailed.*



*If you have any questions, please feel free to Email our hospital at [cecomment@westbridgevets.com](mailto:cecomment@westbridgevets.com).*

## THE "NORMAL" HEAD

### STRUCTURE

- The intracranial space is 80% brain, 10% CSF, and 10% blood
- The brain is encased in bone
- Under normal circumstances an  $\uparrow$  in pressure can be compensated for by a  $\downarrow$  in CSF.

### PHYSIOLOGY

- The brain is able to regulate cerebral vasculature based upon metabolic values, pressure, and oxygen availability providing there is normally functioning vasculature and normal intracranial pressure. (e.g. Increased cellular activity leads to an  $\uparrow$   $\text{CO}_2$  which in turn leads to vasodilation and increased blood flow.
- Local vasculature controlled by metabolic activity, pH, and adenosine concentration. Global cerebral blood flow mainly determined by  $\text{pCO}_2$ .
- Ischemic response (relatively late response):  $\downarrow$  cerebral perfusion leads to  $\uparrow$   $\text{CO}_2$  which in turn leads to an elevated heart rate and intense systemic vasoconstriction to support CPP.
- The normal brain can maintain cerebral perfusion pressure over a systemic blood pressure of 50-150mm Hg.
- Cerebral perfusion pressure (CPP) = Systemic arterial pressure (SAP) – Intra-

- cranial pressure (ICP)
- Normal consciousness requires both the reticular activating system (RAS of brainstem) and the cerebral cortex.

## THE "TRAUMATIZED" HEAD

### PRIMARY EFFECTS

- **Concussion** = Transient loss of function without obvious structural damage
- **Contusion** = Petechiation and Hemorrhage in cortex +/- edema and necrosis; Leading to focal signs which can resolve or progress (Brainstem hemorrhage more likely with twisting impact.) [Hematomas are uncommon in animals]
- **Skull fractures**: Depressed vs. Non-displaced
- **Direct axonal damage**
- The only primary injury that can be treated are depressed skull fractures (and hematomas when they occur: require MR to diagnose)
- **Focus treatment on preventing secondary effects.**



### SECONDARY EFFECTS

- **Trauma** leads to pain, fear, and seizures which result in increased cerebral metabolism. (Massive depolarization leads to increased ATP usage) The brain compensates by vasodilating.
- If there is concurrent respiratory compromise you can get an elevated pCO<sub>2</sub> which leads to further vasodilation and a decreased pO<sub>2</sub> which leads to hypoxia, anaerobic metabolism, and cell and capillary damage. Cell and capillary damage will result in edema formation.
- **Hemorrhage** within the brain will directly lead to increased intracranial pressure. Hemorrhage outside the brain will lead to whole body reflex vasospasm resulting in hypoxia and increased cell and capillary damage.
- **Excitotoxicity**: ↑ Glutamate concentration (an excitatory neurotransmitter) → ↑ Ca<sup>2+</sup> intracellularly → ↑ ATP utilization, uncoupling of oxidative phosphorylation (↓ ATP production), and activation of several enzyme systems → degradation of DNA, RNA, proteins, and membrane phospholipids and oxygen free radical production.
- **Altered vascular reactivity**: Direct damage to endothelium as well as loss of regulation of vasoactive substances. When vascular reactivity is lost, local or global cerebral blood flow is dependent on cerebral perfusion pressure alone.
- **Herniation** is a shift of tissues between compartments from high to low pressure. Any cause of increased intracranial pressure may lead to herniation. In head

trauma, mostly concerned about tentorial herniation.

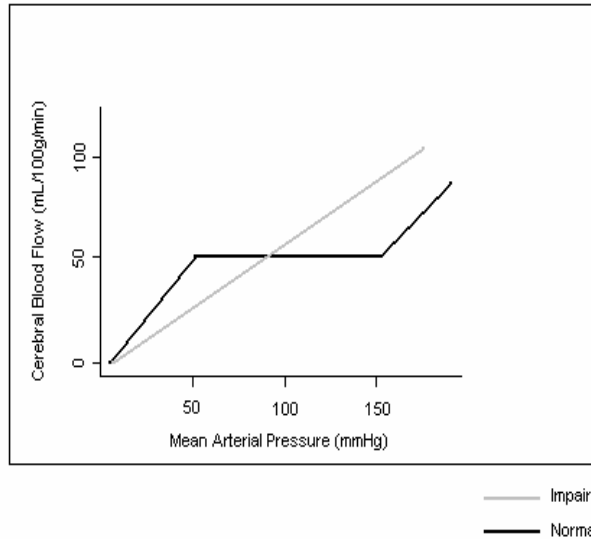


Diagram 1: Adapted from Proulx Compendium Article

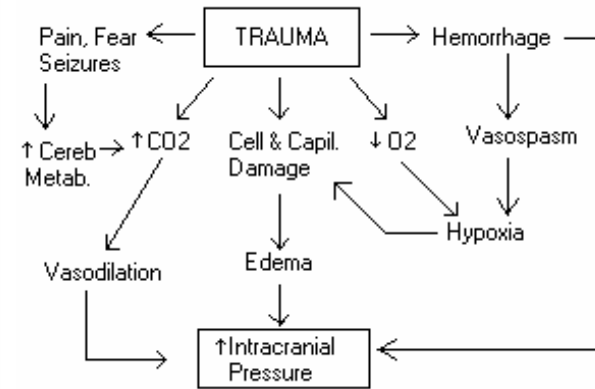


Diagram 2: Adapted from Lecture by Dr. Dennis O'Brien

## MONITORING HEAD TRAUMA

- **Increased intracranial pressure (ICP)** causes decreased cerebral perfusion pressure ( $CPP = MAP - ICP$ ) the body responds by increasing mean arterial pressure. The body's baroreceptors sense an increased blood pressure and decrease heart rate. This phenomenon is called the Cushing's response. ( $\uparrow ICP \rightarrow \uparrow MAP + \downarrow HR$ )
- **ICP can be measured by intraventricular, subarachnoid, subdural, and epidural catheters**- similar to the way central venous pressure is measured. Complications include infection, hemorrhage, malfunction, obstruction, and malposition; however, these methods are the best way to know what is happening to the brain, when it happens. If you do not have a direct ICP monitor, there will be a lag between when the pressure changes and when you see the resultant systemic or neurologic response.
- **Heart rate, systemic blood pressure, neurologic status**, and other parameters associated with the trauma should also be monitored. (Note: Post trauma- Clear discharge from the ear can only be CSF. A clear discharge from the nose could be serous or CSF. Differentiate with a glucose level: Serous should be low, CSF will be 70-80% of BG)

## PROGRESSION

- **Hemorrhage** - Rapid change or no progression
- **Edema** – Maximum signs at 24 hours, will usually resolve within 96 hours.
- **Herniation** – Slow

## NEUROLOGIC STATUS

As ICP increases, there will be a progression of signs which begin in the forebrain and progress to the brain stem with eventual herniation under the tentorium.

### *Diencephalic Stage (Forebrain):*

- **Attitude:** Depression to Stupor
- **Pupils:** Small but responsive (A loss of forebrain inhibition)
- **Doll's Eye reflex intact**
- **Gait:** Normal to Paretic (weakness)
- **Respiration:** Normal to Cheyne-Stokes (Tachypneic and Deeper followed by Apnea)

### *Midbrain Stage (Red nucleus (-) inhibition and some cranial nerves):*

- **Attitude:** Stupor to Coma
- **Pupils:** Dilated and Fixed (Loss of CN III)
- **Doll's Eye weak +/- Ventrolateral Strabismus (CN's III, IV)**
- **Decerebrate Rigidity (Loss of Red Nucleus)-** stiff legs, arched back and neck
- **Respiration:** Hyperventilation

### *Pons Stage (Further pressure on Brainstem):*

- **Attitude:** Coma
- **Pupils:** Midrange and Fixed w/ no PLR (Loss of III and Sympathetic)
- **Doll's Eye lost**, eyes midposition (CN's III, IV, VI)
- **Decerebrate Flaccid (Loss of VIII)**
- **Respiration:** Rapid shallow or apnea

### *Medulla Stage:*

- **Coma to Death**
- **Pupils:** Fixed and Dilated
- **Flaccid**
- **Apnea**

## TREATMENT OF HEAD TRAUMA

- Therapy is directed towards preventing the secondary effects.
- The only primary effect that can be effectively treated would be depressed skull fractures which would require surgery.

### SUPPORT RESPIRATION AND CIRCULATION- *Give FLUIDS!*

- Remember CPP = MAP – ICP
- Do NOT restrict fluids (Dehydration only slightly decreases ICP)
- **Hypertonic saline with colloid** will have rapid and prolonged effect
  - **Caution:** bolus of hypertonic saline may cause a pulmonary vagoreflex resulting in drop in HR. If it occurs, discontinue hypertonic saline and administer vagolytics (atropine) if indicated.
- Isotonic (or close to isotonic) solutions for volume resuscitation:  
Body=295mOsm, 0.9% NaCl=310mOsm.
- **Do NOT use hypotonic solutions**
- **Do NOT use fluids containing glucose**

### TREAT RESPIRATORY COMPROMISE

- Oxygen
- Treat chest trauma as needed (Thoracocentesis, chest tube...)
- However, sneezing and coughing cause ↑ ICP so beware nasal oxygen

#### *Hyperventilate?* (Currently not recommended as routine treatment)

- Hypocapnia → Vasoconstriction → ↓ cerebral perfusion
- However, avoid hypercapnia
- Maintain pCO<sub>2</sub> at 30-35mmHg
- If intubation necessary, IV lidocaine (2mg/kg for dogs and 0.25mg/kg for cats) may be used to blunt the increased ICP associated with intubation

### CELL AND CAPILLARY DAMAGE/ CYTOTOXIC CASCADE

#### *Steroids - To give or not to give?*

- **Potential beneficial effects** include interfering with lipid peroxidation and formation of inflammatory molecules.
- **In human trials, no benefit**
- **Side effects: Hyperglycemia** – Detrimental in head trauma
  - Lead to increased anaerobic metabolism and lactic acid production
  - Hyperglycemia is correlated with poorer outcome
- Other side effects: **GI ulceration, pancreatitis, and sepsis**
- In general, **DO NOT USE** unless there is concurrent spinal trauma such that the

positive effects outweigh the potential negative effects.

## **DECREASE METABOLIC DEMAND**

### **Seizures: Treat aggressively!!!**

- Valium
- Barbiturates: May be protective, but ↓ respiration

### **Pain management (+/-Sedation):**

- Decreases cerebral metabolism but may lead to hypoventilation and hypoxemia
- Phenothiazines: ↓ BP, +/- ↓ Seizure threshold
- Opiates: ↓ respiration, ↑ ICP

### **Hypothermia:**

- Moderate hypothermia reduces secondary brain injury and improves behavioral outcome
- Hypothermia decreases ventricular glutamate and IL-1 $\beta$  (inflammatory mediators) and decreases basal energy requirements
- Possible complications (T<30°C) include clotting disturbances, hypotension, bradycardia, and cardiac arrhythmias. Also, shivering may increase ICP (should be treated with muscle relaxants or atracurium) or you may get rebound increases in ICP during rewarming (Rewarm slowly)

### **Fever:**

- Find underlying cause
- Treat with antibiotics if indicated
- Nonsteroidals, cool fluids, wet blankets, and ice packs

## **REDUCE EDEMA**

**Elevate Head:** Do not kink jugular; whole body on slant

### **Mannitol:**

- Immediate effect – plasma expanding: decreases Hct, ↓ viscosity, ↑ CBF, ↑ cerebral oxygen delivery.
- Delayed approx. 15-30 min.: Osmotic diuretic, radical scavenger
- Hemorrhage? – Global effect outweighs local effect
- Dose 0.25-1 g/kg IV bolus at 2mL/kg/min; repeat if needed
  - Bolus more effective than CRI
- Watch hydration and sodium level (Mannitol causes free water loss in excess of sodium)

## **SURGERY**

- Depressed skull fractures
- Open wounds
- Hematoma
- ↑ ICP, unresponsive to medical therapy

## **NURSING CARE**

- Frequent turning
- Nutrition
- Prevention of pressure sores
- Attention to bladder and bowel function

## **Complications of head trauma**

- **ASPIRATION PNEUMONIA** – Depressed swallowing reflexes
- **MENINGITIS** – Prophylactic antibiotics not indicated, but if used, need broad spectrum that crosses blood brain barrier
- **EPILEPSY** – Usually within 2 years of injury

## **Research/ Experimental Therapy**

### **TIRILAZAD MESYLATE (+ Lazaroids)**

- Nonglucocorticoid steroid analog of methylprednisolone sodium succinate
- Inhibitor of lipid peroxidation in nervous tissue
- Antioxidant
- Showed promise in experimental studies but no benefit in clinical trial

### **MAGNESIUM**

- After experimental brain trauma, free magnesium concentrations decrease
- In rat model, if Mg was administered 30 minutes after brain injury, improved outcome

### **EXCITATORY AMINO ACID ANTAGONISTS (Dextromethorphan, Nitroglycerin)**

- Some newer compounds are experimentally improving neurologic outcome, delaying onset of brain edema, and improving brain tissue ion homeostasis

### **DMSO**

- Free radical scavenger, stabilizes lysosomal membranes, and anti-inflammatory
- Dose of 0.5-1.0 gm/kg IV over 30 minutes has been shown to decrease ICP and

improve outcome in laboratory animals

### **ACETYLCYSTEINE**

- Antioxidant
- Improves neuronal survival and vascular reactivity during reperfusion following cerebral ischemia

### **DEFEROXAMINE MESYLATE**

- Iron chelator and inhibitor of lipid peroxidation
- 25-50mg/kg IM or slow IV
- May have a role in early prevention of reperfusion injury

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