MANAGEMENT OF THE HEAD TRAUMA PATIENT

INTRODUCTION

- Head trauma is a common presentation in wildlife rehabilitation centres
- Various causes:
  - Motor vehicle accident (HBC)
  - Projectiles (bullets)
  - Predator attacks
  - Falls
  - Crush injuries
- When an external force to the head causes injury to the brain → traumatic brain injury (TBI)

OUTLINE

- Introduction
- Primary injury and Secondary injury
- Systemic contributions
- CPP and ICP
- Assessment & Stabilization
- Extracranial therapy and Intracranial therapy
- Ancillary therapies
- Pharmacology
- Supportive Care
- Recap and prognosis
PRIMARY INJURY

- Direct result of the mechanical damage at the time of the incident
- Mechanical forces: concussion, contusion, compression, shear, laceration, distraction
  - Physical disruption of intracranial structures
  - Hemorrhage and hematoma, diffuse axonal injury
- Can be focal or diffuse
- This direct damage to the brain tissue is beyond your control
  - Generally has already occurred prior to presentation

SECONDARY INJURY

- Occurs over the minutes to days following the incident
- Combo of systemic extracranial insults and intracranial physical/biochemical changes leads to secondary injuries:
  - Intracranial hemorrhage
  - Brain swelling
  - Increased ICP
  - Ischemic injury to brain
    - Refers to injury as a result of inadequate blood supply
    - Increased fluid inside the skull
- Focus of treatment is on prevention of secondary injury

SYSTEMIC CONTRIBUTIONS

- Extracranial events can worsen the cerebral injury as a result of compromised perfusion
  - Hypotension
  - Low blood pressure
  - Hypoxia
    - Not enough O2 in the body
  - Hypo- or hyperglycemia
    - Low or high blood sugar
  - Hypo- or hypercapnia
    - Low or high CO2 in the body
  - Hyperthermia
    - High body temperature
CEREBRAL PERFUSION PRESSURE

- Cerebral perfusion pressure (CPP) is the primary determinant of blood flow to the brain
- Basically the difference between systemic blood pressure and the pressure inside the skull
  - Represents the pressure gradient driving blood flow to the brain

In TBI you work to maintain CPP by maintaining a good systemic blood pressure and by keeping the pressure inside the skull low. This allows good blood flow to the brain!

INTRACRANIAL PRESSURE

- Increased pressure inside the skull - a common sequelae to head trauma
- Skull is a rigid compartment containing 3 components:
  - Brain, blood, cerebral spinal fluid
  - Increase in one leads to decreased volume in one or both of the other components (compensatory mechanism)
- There are limits to this compensation!
  - If pressure in the skull increases beyond the limit of the compensatory mechanisms, blood flow in the brain is compromised

INITIAL ASSESSMENT

- ABC’s
  - Airway, Breathing, Cardiovascular
- Hypovolemia (decreased circulating blood volume) and hypoxemia (insufficient O2 in the body) strongly correlated with increased ICP
  - Hypercapnia (high CO2 in the blood) leads to dilation of cerebral blood vessels → increased ICP
  - Ensure airway is patent
  - Know that breathing abnormalities may be associated with concurrent injuries
  - Establish normovolemia
NEUROLOGIC ASSESSMENT

- State of consciousness
  - Alert
  - Obtunded (dull, but can be aroused by non-noxious stimuli)
  - Stuporous (semiconscious/somnolent, rousable with a noxious stimulus)
  - Comatose (unconscious, cannot be roused by a noxious stimulus)
  - Brain dead (no electrical activity in the brain, no brainstem reflex function)

- Breathing pattern
- Pupil size and responsiveness
  - Not as helpful in wildlife as in dog/cat medicine
- Seizures, changes in behaviour, turned head, circling, head pressing – all indicate brain injury
- Ocular position and movements
- Skeletal motor response
  - Affected by level of consciousness

STABILIZATION

- Extracranial stabilization
  - Maintenance of adequate intravascular volume
  - Oxygenation
  - Ventilation

- Intracranial stabilization
  - Optimizing blood flow to the brain
  - Minimize increases in ICP
  - Minimize elevations in metabolic rate in the brain
  - Because this increases demand for O2
EXTRACRANIAL THERAPY

Fluid Therapy
- Primary goal is to restore intravascular volume to ensure adequate blood flow to the brain
- Historically recommended to limit fluid volume in TBI to avoid exacerbating cerebral edema but dehydration does not reduce cerebral edema (Sande and West, 2010)

It is judicious to avoid excessive fluid administration, however erring on the side of dehydration is also firmly contraindicated
- Human TBI patients
  - Hypotension significantly impacts morbidity and mortality (Chestnut et al. 1993)
  - Resuscitation protocols are aimed at avoiding hypovolemia
- IV catheter ideally
  - Subcutaneous fluids alternatively
  - Isotonic fluids ONLY; never give hypertonic saline or mannitol subcutaneously

EXTRACRANIAL THERAPY

Fluid therapy
Parameters to monitor:
- Normalization of HR
- Pulse quality
- Mucous membrane color and CRT
- Blood pressure

EXTRACRANIAL THERAPY

Oxygenation
- O2 supplementation is recommended for most TBI patients
- Decreased O2 delivery is a key perpetrator of secondary brain injury
- Estimate O2 status
  - Respiratory rate and pattern
  - Mucus membrane and tongue color
  - Thoracic auscultation
  - Pulse oximetry
- O2 face mask is generally ideal
EXTRACRANIAL THERAPY

Ventilation
- It is not enough to simply provide oxygen to the patient – you need to ensure they are ventilating
- CBF and cerebral blood volume (CBV) are heavily influenced by CO2 levels in the normal brain
  - Hypercapnia → dilation of blood vessels in the brain → ↑ blood flow through the brain → ↑ pressure
- Ventilation is required to transport oxygen to the tissues, and carry CO2 away
  - Hyperventilation as a way of decreasing ICP?
  - Risk of ischemic injury to the brain

INTRACRANIAL THERAPY

- The goal is to minimize increases in ICP and maximize CPP
  - Elevate the head 15-30 degrees
  - Limit obstructions to venous drainage
    - No collars, wraps, jugular catheters
    - Avoid bending the neck

HYPEROSMOTIC AGENTS

Mannitol and Hypertonic Saline (HTS)
- How do they work?
  - Water in the body will always want to flow from an area of low concentration to an area of high concentration to create an equilibrium (to “dilute” the concentrated fluid)
  - Putting a hyperosmotic agent into the bloodstream puts a high concentration of particles in the blood
  - The blood brain barrier allows water to cross but will not allow mannitol or HTS to cross
  - So, water in the brain (edema) will flow across the barrier into the blood in order to dilute the blood
- The end result is less fluid inside the head → decreased pressure
INTRACRANIAL THERAPY

• HTS vs Mannitol?
  • Mannitol is still considered the first-line treatment for increased ICP in TBI
  • BUT there is growing support in the literature that HTS may be superior
    • Resulted in greater absolute decrease in ICP and a more sustained effect compared to mannitol (study in human TBI patients) - (Mangat et al. 2015)

INRACRANIAL THERAPY

• Mannitol
  • 0.5 grams/kg given IV over 15-20 minutes
  • Plumb’s Veterinary Drug Handbook 8th ed.
  • NEVER GIVE SUBCUTANEously

• Hypertonic Saline
  • 2-5 ml/kg given IV over 15-20 minutes
  • 2013 AAHA/AAP Fluid Therapy Guidelines for Dogs and Cats
  • NEVER GIVE SUBCUTANEously

• Always follow with isotonic crystalloid fluid therapy to ensure adequate tissue hydration
  • Remember to be judicious in your fluid administration
ANCILLARY THERAPIES

Glycemic control
• Studies have shown that hyperglycemia is associated with worsened neurologic outcomes (Sande and West, 2010)
  • Human literature: debate as to whether degree of hyperglycemia is a reflection of severity of the injury or a cause of worsened secondary injury

Hypothermia
• Employed as a tx in human TBI patients
  • Mechanism of how it limits secondary brain injury is not well defined
  • Hypothermia should be avoided as it increases cellular metabolism and vasodilation in increases ICP
• Keep patients at a “low-normal” body temperature (i.e. not cold, but at the low end of the normal range) (Sande and West, 2010)

PHARMACOLOGY

Analgesia
• Controlling pain is critical to preventing increases in ICP
  • Meloxicam (NSAID)
    • Important part of treatment for its analgesic AND anti-inflammatory properties!
  • Mammals: loading dose of 0.2 mg/kg, then 0.1 mg/kg q24h IM/SC/PO
  • Rabbits/Rodents: loading dose 0.3 mg/kg, then 0.3 mg/kg q24h IM/PO/SC
  • Birds: 1 mg/kg q12h PO (IM once ok but repeated causes muscle necrosis)
  • Reptiles: 0.2 mg/kg q24-48h IM/PO
• Opioids titrated to effect
  • Excellent analgesia, minimal cardiovascular effects
  • Hydromorphone 0.05-0.2 mg/kg IV/IM/SC q4h

Anticonvulsants
• Adverse effects of seizure activity: hyperthermia, hypoxemia, cerebral edema
  • All lead to increases in ICP
• Diazepam
  • Mammals (inc. rabbits and rodents) 0.1-0.5 mg/kg PO
  • Birds: 1-2 mg/kg PO
• Midazolam
  • Mammals (inc. rabbits and rodents) 0.1-0.5 mg/kg IM/SC/IV
  • Birds: 1-2 mg/kg IM/SC/IV/IN
PHARMACOLOGY

- GI protectants
  - Routine stress ulcer prophylaxis is recommended in trauma patients (Sande and West, 2010)
  - Famotidine
    - Mammals: 0.5 mg/kg PO/SC/IV q12h
  - Omeprazole
    - Mammals: 0.5-1 mg/kg PO q24h
    - Rabbits/rodents: 20 mg/kg SC q12h
  - Sucralfate
    - Large mammals: 1g PO q12h
    - Small mammals: 210-500 mg PO q6-12h
    - Rabbits/rodents/Birds: 25 mg/kg PO q8-12h
- Motility modifying drugs
  - Delayed gastric emptying can occur
    - Leads to poor nutrition, bacterial colonization of the GIT, reflux, risk of aspiration pneumonia

SUPPORTIVE CARE

- Nutrition
  - Very important consideration in TBI!
  - TBI leads to a hypermetabolic and catabolic state
  - Enteral nutrition helps maintain integrity of the gut, helps the immune systems, attenuates metabolic response to stress
  - NG tube placement if they will not (or cannot) eat
- Frequent turning, clean dry bedding, physical therapy
  - Particularly important for an animal that cannot ambulate!
  - Helps prevent pressure sores, urine scalding, limb contracture
- "If an animal is unable to ambulate you must ensure that they are still able to urinate" - put them on a "urine watch"
  - Monitor urine output frequently
  - If they are unable to urinate this is an emergency and veterinary attention is needed immediately
STEROIDS – YES OR NO?

- The results of the Medical Research Council CRASH (corticosteroid randomisation after significant head injury) trial showed increased risk of death in head injury patients treated with corticosteroids
- Concluded that corticosteroids should not be routinely used in the treatment of head injury (Roberts et al. 2004; Edwards et al. 2005)

RECAP

- Goal of treatment is to minimize the effects of secondary brain injury – in particular, preventing increases in ICP.
- Provide fluid support (IV if possible, otherwise SC)
- Provide oxygen support and maximize ventilation
- Prevent hyperthermia (no heating pads!)
- Keep the head slightly elevated, without bending the neck
- Hyperosmolar therapy (mannitol or HTS)
- Control pain, treat seizures, provide GI protectants and motility enhancers as needed
- A lot of supportive care!

NO STEROIDS!

PROGNOSIS?

- Immediate and aggressive treatment is key
- Many patients can recover if systemic and neurologic abnormalities are identified and addressed early
- Animals have a remarkable ability to compensate!
- Don’t make hasty conclusions on prognosis based on the initial appearance on presentation!
RESOURCES