Management of Head Trauma and Traumatic Brain Injury
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Introduction
Patients with head trauma present with interesting diagnostic and therapeutic challenges. A good neurological assessment will help to localize the lesion and the severity of clinical signs will help to predict the prognosis. Therapeutic challenges for patients with traumatic brain injury (TBI) include tight regulation of blood pressure, oxygenation, and ventilation, in order to optimize cerebral blood flow (CBF). Direct measures to control intracranial pressure elevations include improving venous drainage, reducing pain, managing seizures, monitoring glucose levels, and regulating temperature. Extensive nursing care and frequent monitoring are imperative as neurological status can change abruptly. Worsening of the neurological status or clinical signs suggestive of imminent brain herniation, require intervention with hyperosmolar solutions and adjunctive therapies.

Key Physiologic Points
Head trauma can cause bruises, contusions, or other injuries to the brain, which are considered the primary insults; controlling the secondary insults such as hemorrhage, inflammation, and edema is the focus of the treatment of TBI. CBF is decreased after TBI and poor perfusion to damaged areas of the brain leads to a lack of oxygen and glucose, decreased ATP availability, and proton pump inefficiency. This in turn causes intracellular accumulation of Na\(^+\) and Ca\(^{++}\) and extracellular accumulation of K\(^+\), which leads to cell swelling, vasogenic and cytotoxic edema, oxidative injury, and further ischemia and neuronal necrosis.

Normal perfusion to the brain is auto-regulated within a systemic mean arterial pressure (MAP) of 50-150 mmHg. This auto-regulation is also maintained by vasomotor tone, which is regulated by changes in arterial oxygen and carbon dioxide concentrations. Auto-regulation is lost after TBI; therefore, careful management of systemic blood pressure, oxygenation, and ventilation are vital to optimize CBF and improve recovery.

Key Diagnostic Points
The level of consciousness, cranial nerve examination (particularly pupillary light response), and postural reactions make up the crucial elements for evaluating the TBI patient. Systemic signs such as abnormal ventilatory patterns and the Cushing’s reflex (hypertension and bradycardia) are imminent signs of severely elevated intracranial pressure (ICP) resulting in brain herniation and impending death.

Key Therapeutic Points
Adequate oxygen delivery to the brain is vital in patients with TBI. Maintaining normotension is critical (goal: MAP of 90-100 mmHg) in the face of an elevated ICP. Supplemental oxygen should be administered to achieve an \(\text{SpO}_2 > 95\%\). Normal ventilation and regulation of \(\text{CO}_2\) is also imperative. Other preventative measures to control ICP include promoting venous drainage (elevating the head above the bed),
providing sedation/analgesia, as well as avoiding jugular venipuncture, hyperglycemia, and hyperthermia. Evidence of elevated ICP should be treated with mannitol or hypertonic saline.

**Key Prognostic Points**

For patients with thalamocortical injury and minimal brain stem injury, the prognosis is excellent; however, hospitalization and intensive care might be required. Prognosis improves markedly once patients have survived 24 hours after their initial injury. However, patients can change neurological status abruptly and > 72 hours are necessary before the patient is considered to be “out of the woods”. Patients with significant brainstem injury (comatose patients with dilated non-responsive pupils and no oculocephalic reflex) have a grave prognosis for survival.

**Patient Assessment**

Head trauma patients must be appropriately assessed to determine the extent of their TBI, to localize the lesion, and to determine their prognosis. These patients are assessed based on their level of consciousness, cranial nerve examination, motor activity, postural reactions, and systemic signs. It is important to remember that patients with head trauma may have cervical or other trauma and movement of the head should be minimized during assessment.

**Level of Consciousness**

Altered mentation is a hallmark of head trauma. The following is a list of terms and their definitions should be used to describe head trauma patients:

- Delirious – inappropriate response to environmental stimuli
- Obtunded/depressed – decreased responsiveness to environmental/verbal stimuli
- Stuporous – respond only to noxious/painful stimuli
- Comatose – unresponsive to noxious stimuli

Decreased level of consciousness, or ultimately coma, can result from injuries to the brainstem and the ascending reticular activating system (ARAS) or the thalamocortex. Differentiating the site for decreased consciousness is important to determine the patient’s prognosis. Deficits in the cranial nerves indicate the presence of brain stem involvement and portend a poorer prognosis.

**Cranial Nerve Examination**

A complete cranial nerve examination is recommended with specific attention paid to facial and nasal septum sensation, menace response, palpebral reflex, and gag reflex. A large amount of information can also be obtained from the pupillary light and oculomotor reflexes.

_Pupillary light reflex (PLR)_ evaluates cranial nerve (CN) II and CN III. The ability to constrict the pupil indicates that CN III remains intact. It is imperative that even subtle changes in PLR be recognized as prognosis varies greatly based on the presence (good) or absence (bad) of a PLR. Small pin-point pupils suggest a lack of higher input from the thalamocortex with a resultant increase in pupil tone. Dilated non-responsive pupils
indicate a complete lack of CN III input and suggest a grave prognosis. Unilateral changes can be noted in the presence of unilateral injuries.

*Physiologic nystagmus (oculocephalic reflex, doll’s eye reflex)* evaluates CN III, IV, VI, VIII and the medial longitudinal fasciculus, which runs the length of the brainstem. This test evaluates a large segment of the brainstem and is a good ‘global’ assessment of the brainstem. The presence of any sort of nystagmus (positional, horizontal, vertical or rotatory) suggests that input from the brainstem is present. To elicit this response, small patients can be held in the air, while the clinician rotates and observes the patient for ocular movement. For larger patients, the head must be moved from side to side while observing for nystagmus. However, always use caution not to move the neck of patients with potential cervical trauma.

**Motor function**

*Gait* is generated in the brainstem; therefore, brainstem lesions can affect the ability to ambulate. Significant brainstem lesions can be associated with a complete loss of voluntary motor function (plegia) and hypotonia. Lesions in the thalamocortex do not affect gait generation; however, weakness of the limbs on the contralateral side of the lesion can be seen.

*Circling* indicates intracranial disease. Cerebrocortical disease (wide circles) must be differentiated from vestibular disease (tight circles and accompanied by a head tilt). Circling is generally to the side of the lesion.

**Postural Reactions and Spinal Reflexes**

Postural reactions are evaluated by assessing proprioception, tactile placing, and/or hopping if the patient is stable. It is important to note that the spinal tracts cross in the rostral brainstem. Therefore, postural deficits will be noted on the contralateral side with thalamocortical involvement and on the ipsilateral side with brainstem or spinal cord lesions. Lateral recumbency, loss of muscle tone, and absent spinal reflexes (e.g., withdrawal or patellar reflex) suggest a very poor prognosis.

**Positional Abnormalities**

Certain postures are associated with particular lesions. *Decerebrate rigidity* results from disconnection of the higher centers of the brain and the brainstem. Patients will display opisthotonus characterized by an arching of the head backwards and the limbs and tail held in rigid extension. These patients are comatose, lack PLRs, and have dilated or pinpoint pupils. This posture suggests an extremely guarded prognosis. *Decerebellate rigidity* results from an injury to the rostral lobe of the cerebellum. These patients are alert and aware, but posturally the neck and forelimbs are in extension and the hind limbs are unaffected (normal or hyper-flexed).

**Systemic Signs**

Concomitant hypertension and bradycardia associated with an elevated ICP is known as the *Cushing’s reflex*. CBF is difficult to determine; therefore, cerebral perfusion pressure (CPP) is used as a surrogate marker for CBF. The following equation describes cerebral perfusion pressure: \( \text{CPP} = \text{MAP} - \text{ICP} \). Based on this equation, if ICP rises, MAP must also increase in order to maintain an adequate CPP. Elevations in ICP lead to
progressive decreases in CPP and poor perfusion of the pons and medulla. CO₂ also accumulates and massive sympathetic stimulation generates a markedly increased systemic blood pressure. The large rise in MAP stimulates a baroreceptor-mediated bradycardia. The Cushing’s reflex should be suspected in any patient presenting with decreased mentation and bradycardia. Blood pressure should always be measured in patients with head trauma and TBI. The Cushing’s reflex is suggestive of markedly elevated ICP and impending brain herniation.

The respiratory center is located within the brainstem. Therefore, many different respiratory patterns can be seen with TBI. Markedly abnormal respiratory patterns such as Cheyne-Stokes breathing (i.e., alternating apnea and hyperventilation) suggest a brainstem lesion and carry a poorer prognosis.

Monitoring Head Trauma Patients

Frequent re-evaluation of the head trauma patient is imperative as changes in neurological status can happen abruptly and require immediate intervention. Minimally, TPR, blood pressure, SpO₂, and mentation should be monitored regularly in patients with TBI. Initially, this might be required every 5-15 minutes and thereafter every 1-4 hours. If possible, arterial, venous, or end-tidal CO₂ should also be monitored in patients with abnormal breathing patterns or in patients that are intubated. Patients should also be assessed for changes in cranial nerve or postural reflexes every 4-6 hours or sooner if their mentation deteriorates.

Treatment of Head Trauma Patients

Any patient showing a combination of 2 out of the 3 following signs should be treated for elevated ICP and imminent brain herniation: 1) decreased level of consciousness; 2) absent physiologic nystagmus or PLR (bilateral or unilateral with no evidence of ocular trauma or Horner’s syndrome); or 3) the Cushing’s reflex. Treatment can include mannitol or hypertonic saline or both.

Mannitol is an osmotic diuretic and should be administered at a dose of 0.5 g/kg IV over 10 minutes for impending brain herniation. Higher doses of mannitol (1-2 g/kg IV) might be required and have recently shown improved effects in comatose patients. A constant rate infusion (CRI) of mannitol should be avoided and is not necessary. Mannitol initially causes a large shift of water into the intravascular space with a transient increase in systemic and intracranial blood pressure. Other effects of mannitol include improved cerebral blood flow due to changes in rheology (red cell deformability), as well as oxygen free radical scavenging.

Hypertonic saline is a hyperosmolar solution that reduces brain edema. It also promotes cerebral blood flow and modulates the inflammatory response. Dose recommendations vary based on the concentration of hypertonic saline used. A 3% NaCl solution is administered as an intravenous bolus of 5-10 mL/kg over 10-30 minutes. This can be followed by a CRI at 0.5-1.5 mL/kg/hr, which is titrated to effect while maintaining sodium concentrations < 160 mmol/L. Other recommendations include a 4-6 mL/kg IV bolus of 5% NaCl or a CRI of 1 mL/kg/min IV or 4 mL/kg IV bolus of 7.5% NaCl administered slowly. A 23.4% NaCl solution has also been used in human patients and evaluated in research animals as a bolus of 0.25-0.5 mL/kg over five minutes or as a CRI of
0.03-0.08 mL/kg/hr. Concerns when using hypertonic saline include hypernatremia and hyperchloremia; sodium levels should always be maintained < 160 mmol/L.

Recently studies investigating mannitol and hypertonic saline have been compared and results suggest that hypertonic saline administration results in a better outcome in human head trauma patients. The magnitude of the difference and the clinical implications for veterinary patients remain uncertain. Ultimately, if an effect is not seen with one agent, the other agent should be administered.

*Intravenous lidocaine* has also shown positive effects on lowering ICP. Lidocaine is postulated to prevent sodium influx into ischemic neurons, thereby reducing secondary brain injury. Lidocaine can be administered at 0.75 mg/kg IV when ICP is refractory to mannitol or hypertonic saline administration.

Other aspects of treatment for head trauma patients are numerous and are outlined below:

**Normotension:** Adequate CBF is critical for patient survival and optimal cerebral perfusion is necessary for improved patient outcome. Autoregulation is the control of blood pressure within the brain. Autoregulation is achieved within a MAP 50-150 mmHg; however, the injured brain might no longer be able to autoregulate successfully. As such, management of blood pressure is essential. The goal for the TBI patient is to maintain MAP between 90-100 mmHg to ensure appropriate oxygen delivery to the brain. Blood pressure should be supported with crystalloids, colloids, or hypertonic saline. Small volume resuscitation with titrated fluid resuscitation (IV boluses over 15 minutes repeated to effect) is recommended; unnecessarily large volumes of crystalloid solutions should be avoided as fluid overload may exacerbate brain edema.

**Normal oxygenation and ventilation:** Because alterations in oxygen and carbon dioxide concentrations can lead to deleterious secondary brain insults, it is imperative that these levels are maintained within a normal range. If SpO₂ is < 95%, oxygen supplementation should be provided by flow-by, face mask, or nasal prongs (if tolerated) to ensure the SpO₂ is maintained > 95%. Likewise, PaCO₂ should be maintained as close as possible to 40 mmHg. If arterial blood gas measurements are not available, a venous CO₂ can be measured and should be 40-45 mmHg. If a patient's CO₂ is not within the recommended ranges, altered ventilation secondary to severe TBI should be suspected and intubation might be required.

**Airway protection:** In patients with an absent gag reflex or severe hypoventilation, endotracheal intubation might be required to protect the airway, prevent aspiration, and ensure normal ventilation. If the patient is intubated, end-tidal CO₂ monitoring can be employed to determine whether the patient is ventilating normally or requires manual ventilator support.

**Manual ventilation:** Intubation and manual ventilation allows manipulation of CO₂ levels. Hypercapnia results in vasodilation and can exacerbate elevations in ICP. Ideally, patients should have CO₂ levels maintained near 40 mmHg. With normal lung function, end tidal CO₂ and PaCO₂ will differ by approximately 5 mmHg; therefore, maintaining end tidal CO₂ around 35 is appropriate. Hyperventilation and hypocapnia conversely promote vasoconstriction and can alleviate elevations in ICP while other treatment options take effect. Hyperventilation causing hypocapnia is only a temporary option in the face of imminent brain herniation as prolonged vasoconstriction will lead to decreased cerebral perfusion.
**Improve venous drainage:** Further elevations in ICP can be avoided by ensuring adequate venous drainage from the cranial part of the body and brain. Elevate the head and forelimbs approximately 15-30 degrees above the bed and ensure that the jugular veins are not compressed by bandages or collars. It is also important to prevent coughing, sneezing, or vomiting as these will also elevate ICP. If intubation is required, provide a smooth induction by premedicating the patient and spraying the larynx with lidocaine spray to avoid coughing. Avoid placement of nasal oxygen cannulas and instead administer oxygen via nasal prong (if easily tolerated), mask, or flow-by oxygen. Consider antiemetic therapy including metoclopramide or maropitant to prevent nausea, retching, or vomiting.

**Temperature regulation:** Low body temperature will decrease cerebral oxygen consumption and hypothermia is considered protective for the brain. It is recommended that patients with TBI be maintained at a cool body temperature, but above 98°F (36.5°C) to avoid shivering, which increases metabolic oxygen consumption. Active cooling is not often performed; however, permissive hypothermia can be employed, which means not actively warming patients that are hypothermic (unless they are shivering).

**Pain management:** Opioids are the mainstay of therapy for pain management of patients with head trauma. Excessive opioids can cause hypoventilation or nausea; therefore, opioids should be titrated to effect. Consider using a short acting opioid such as injectable fentanyl, which can be titrated easily. Otherwise, buprenorphine can be used as a first line agent. If mu-agonist opioids (e.g., morphine, hydromorphone) are required for additional pain relief, use small incremental doses to avoid nausea/vomiting or hypoventilation. Only once the patient is stable for 24 hours should NSAIDs be considered.

**Glucose control:** Maintenance of normoglycemia is optimal for patients with TBI. Hyperglycemia should be avoided since it drives cerebral lactate production resulting in acidosis and potentiation of cell death. Hyperglycemia has been associated with worse neurologic injuries in dogs and cats, and a poorer outcome in people. Additionally, hypoglycaemia should absolutely be avoided since it will have the most deleterious effects including cerebral metabolic effects in TBI patients.

**Nursing care:** Extensive nursing care can be required for TBI patients. Regular turning of the patient to avoid pressure sores is necessary. Physiotherapy can also be required if the patient is recumbent for a period of time. Depending on the extent of neurological dysfunction, regular application of ocular lubrication might be necessary for patients unable to blink voluntarily. Suctioning of the mouth might also be required for patients with difficulty swallowing.

**Nutrition:** Nutritional requirements should also be addressed and might necessitate placement of a feeding tube. A nasal feeding tube can be considered if it can be safely placed without causing sneezing, coughing, or gagging. Otherwise, an esophagostomy tube could be placed as an alternative, but with the requirement for general anesthesia. An esophagostomy tube would be the preference for patients with concurrent facial or nasal trauma, which would preclude placement of a nasal feeding tube.

**Glucocorticoid therapy:** A landmark large randomized controlled study (CRASH trial) evaluated the use of corticosteroids in human head trauma patients and was published in 2004. The results demonstrated that the risk of death (from all causes) was higher in the patients given corticosteroids. Therefore, steroids are not recommended in patients with TBI.
**Anti-convulsant therapy:** Patients with head trauma and TBI can present with or develop seizures. Seizures increase cerebral metabolic oxygen consumption and cause hyperthermia, both deleterious to patients with TBI. Seizures should be aggressively managed with a diazepam bolus (0.5 mg/kg IV) followed by a constant rate infusion of diazepam (0.5-1.0 mg/kg/hr). Phenobarbital (2-4 mg/kg every 12 hours) or **levatiracetam** (XX mg/kg every 8 hours) should be initiated for continued seizure management. These drugs might be weaned in patients after 6-12 months without seizure activity.

**Anesthetic Considerations**

Should anesthesia be necessary, barbiturates decrease cerebral metabolic oxygen consumption and are a good choice for anesthesia of head trauma patients. Benzodiazepines have no effect on ICP and minimal effect on the respiratory and cardiovascular systems and are good adjunctive sedatives. Propofol can also be considered; however, hypotension and respiratory depression must be anticipated and managed appropriately. The effects of ketamine on ICP are unpredictable and etomidate is associated with cerebral hypoxia and ischemic injury; therefore, both drugs should be avoided in head trauma patients. Inhalant anesthetics reduce cerebral metabolism; however, they cause vasodilation as well as anesthetic-induced hypoventilation and hypercapnia resulting in increased ICP. If ICP is already elevated, inhalant anesthetics should be excluded from anesthetic protocols and total IV anesthesia using barbiturates, benzodiazepines, and/or propofol selected instead.

**Outcome**

As patients regain mobility during the recovery period, new neurological signs might develop. It is common for patients to begin to circle towards the side of the lesion during the recovery period. Patients can also be blind after head trauma, which can be temporary or permanent depending on lesion localization and the extent of the injury. Owners should be forewarned that vision can be affected permanently. After head trauma, patients can also develop behavioral changes, which are impossible to predict. Re-training of commands and re-introduction of routines might be necessary. Seizures can develop at any point in the patient’s life due to scar formation and the traumatized area of the brain serving as a nidus for seizure activity.

**Prognosis**

Prognosis is dependent on the site and extent of neurological damage. However, many head trauma patients (80-90%) can have an excellent prognosis. These patients may require prolonged hospitalization and intensive care to allow time for return to normal function.