Head Trauma

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KEYWORDS

• Head trauma • Traumatic brain injury • Intracranial pressure • Dog • Cat

KEY POINTS

- The goal of managing head trauma is the prevention and treatment of secondary injury.
- Systemic assessment and stabilization should occur before neurologic assessment.
- Validated assessment methods, such as the Modified Glasgow Coma Score and Animal Trauma Triage Score, may serve as guidelines for predicting outcome.
- Treatment is guided toward minimizing secondary injury and considering respiratory and cardiovascular systems, analgesia, anxiety, nutrition, and recumbency care.
- Assessments should be performed frequently to determine if a change in patient status has occurred.

INTRODUCTION

Head trauma is a common cause of morbidity and mortality in small animals. In dogs with severe blunt trauma, head trauma occurs in approximately 25% and is associated with increased mortality.[1] Reported mortality rates in dogs with head trauma range from 18% to 24%.[1,2] Approximately 50% of dogs and cats present with head trauma owing to motor vehicle accidents and crush injuries, respectively. Other causes include falls from height, bite wounds, gunshots, and other accidental or intentional human-inflicted trauma. Head trauma may lead to traumatic brain injury (TBI), defined as a structural or physiologic disruption of the brain by an external force. Rapid recognition and response is required to ensure the best outcome. Dogs and cats compensate remarkably well to losses in cerebral tissue.[3] Although the initial appearance of a head traumatized patient may be discouraging, even patients with severe neurologic deficits can recover with appropriate care. This article reviews the pathophysiology of head trauma, patient assessment and diagnostics, and treatment recommendations.

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PATIENT EVALUATION OVERVIEW

Normal Brain Physiology and Compensatory Mechanisms

Cerebral perfusion pressure (CPP) is the pressure gradient driving cerebral blood flow (CBF), including delivery of oxygen and metabolites. CPP is defined as the mean arterial pressure (MAP) minus the intracranial pressure (ICP): CPP = MAP – ICP. CBF is a function of CPP and cerebral vascular resistance (CVR): CBF = CPP/CVR. CVR is dependent on blood viscosity and vessel diameter: $CVR = L\eta/\pi r^4$, where L is vessel length, η is viscosity, and r is vessel radius.[4] A major mechanism controlling CVR is pressure autoregulation, the intrinsic ability of the vasculature to maintain a constant CBF and ICP over a wide range of pressure (MAP of 50–150 mm Hg). Chemical autoregulation with $Paco_2$ also influences CVR via vessel diameter.[4]

ICP is the pressure inside the skull exerted by the intracranial contents. The Monro-Kellie hypothesis states that the sum of the volumes of brain parenchyma, intracranial blood, and cerebrospinal fluid is constant. Any change in volume without a compensatory decrease in another component will cause an increase in pressure. With head trauma, hemorrhage or edema add to the volume. Increases in volume are buffered by fluid shifts in the brain vasculature and cerebrospinal fluid. This accommodation is known as intracranial compliance, or the change in volume per unit change in pressure.

In normal conditions, intracranial compliance is high and changes in intracranial volume minimally affect ICP. However, when the volume buffering capacity is exceeded, increases in volume directly increase ICP. High ICP decreases CPP, leading to ischemia and neuronal death.[4]

Brain injury is divided into primary and secondary injury. This distinction is valuable in understanding pathophysiology and highlights the goal in managing head trauma: prevention and treatment of secondary injury.

Primary Injury

The physical disruption of intracranial structures at the time of impact. Concussion is the mildest injury characterized by a loss of consciousness with no associated histopathologic lesions. Contusion is bruising of the brain parenchyma. Laceration is disruption of the brain parenchyma and is the most severe form of primary injury. Hemorrhage, hematoma formation, and subsequent compression of the brain parenchyma may also occur. Locations of hematoma formation include within brain parenchyma (intraaxial) and in the subarachnoid, subdural, and epidural spaces (extraaxial). In a study of dogs with mild head injury, 89% had skull fractures and 11% had intracranial hemorrhage.[5] In dogs and cats with severe head injury, nearly all (96%) had evidence of intracranial hemorrhage.[6] Primary injury is beyond the clinician's control and sets the stage for secondary injury.

Secondary Injury

Occurs minutes to days after the initial insult and involves a complex cascade of local and systemic derangements. Brain trauma results in excessive excitatory neurotransmitter release leading to further neuronal damage or death in a vicious cycle of *excitotoxicity*. The accumulation of neurotransmitters such as glutamate cause an influx of sodium and calcium resulting in depolarization and further release of neurotransmitters. Excessive sodium causes cytotoxic edema, whereas calcium activates destructive proteases, lipases, and endonucleases.

Other local contributors of secondary injury include depletion of adenosine triphosphate, production of reactive oxygen species, nitric oxide accumulation, and lactic acidosis.[7]Systemic derangements worsen brain injury by compromising cerebral

perfusion. Hypotension, hypoxia, systemic inflammation, hyperglycemia or hypoglycemia, hypercapnia or hypocapnia, hyperthermia, and abnormalities in electrolytes or acid–base balance all contribute.[8]

Increased intracranial pressure

Intracranial hypertension perpetuates secondary injury. If severe, it may lead to brainstem compression, resulting in a depressed mental, cardiac, and respiratory function. Brain herniation and death are possible. This triggers the Cushing's reflex or cerebral ischemic response. With severely increased ICP, CBF decreases allowing CO_2 to accumulate locally. The vasomotor center of the brain detects the increase in CO_2 and triggers sympathetic discharge causing peripheral vasoconstriction to increase MAP and maintain CPP. Baroreceptors sense the hypertension, triggering a reflex bradycardia. In a patient with decreased mentation, a combination of hypertension and bradycardia indicates a potentially life-threatening increase in ICP requiring prompt treatment.[4]

Other findings with increased ICP include sudden decrease in mentation, pupillary light reflex, decerebrate posture (opisthotonus with hyperextension of all 4 limbs), and loss of physiologic nystagmus.[9] With severe head trauma, blood pressure autoregulation can be lost focally or globally, partially or completely. A partial loss resets the lower MAP extreme to a higher value (eg, from 50 mm Hg to 80 mm Hg). Without pressure autoregulation, CBF becomes directly proportional to systemic blood pressure, highlighting the importance of maintaining optimal blood pressure when treating head trauma cases.

SYSTEMIC ASSESSMENT

Head trauma patients are triaged and assessed for life-threatening injuries and systemic derangements, which contribute to secondary injury. Approximately 60% of human TBI patients have concurrent injuries.[9] See **Fig. 1** for overview of patient assessment.

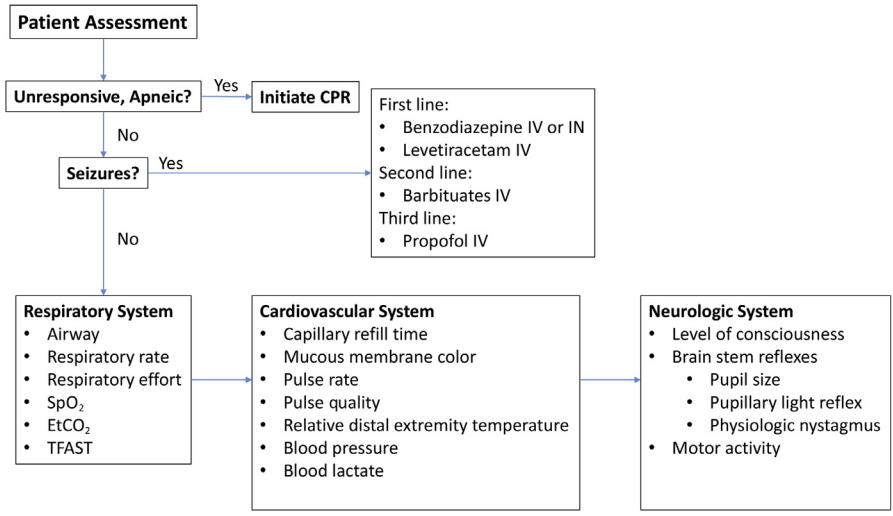

Fig. 1. Overview of patient assessment after head trauma. CPR, cardiopulmonary resuscitation; $EtCO_2$, end-tidal carbon dioxide; SpO_2, saturation of peripheral oxygen; TFAST, thoracic focused assessment with sonography for trauma.

Trauma patients may present in shock, and a full neurologic assessment should occur after patients are resuscitated and stabilized. The main goal is to establish normovolemia and appropriate oxygenation and ventilation. Assessment of the cardiovascular system should focus on the perfusion parameters (mentation, mucous membrane color, capillary refill time, pulse rate, pulse quality, and relative distal extremity temperature). Respiratory rate, respiratory effort, and thoracic auscultation may indicate respiratory compromise. Point-of-care thoracic ultrasound imaging may detect pneumothorax, pleural effusion, and parenchymal disease (contusion).[10]

Neurologic Assessment

Neurologic examination is performed without the influence of analgesia and interpreted based on the patient's stability and condition. For example, miosis may be due to traumatic uveitis or Horner's syndrome; segmental reflexes may be diminished by limb pathology such as fractures. Initial examination is focused on mentation, brain stem reflexes, and motor activity/posture to assign a score using the Modified Glasgow Coma Scale (MGCS), which has been validated in dogs (**Box 1**). The MGCS assesses 3 categories: motor activity, brainstem reflexes, and level of consciousness, with a total score of 18 (each category with a maximum score of 6) being normal. The MGCS is useful for serial monitoring and can be performed as frequently as every 30 minutes in critical patients. MGCS is a useful prognostic tool. Studies have shown that an MGCS of 8 within the first 48 hours of hospitalization approximates a 50% probability of survival.[2,11]

The animal trauma triage (ATT) score is another severity scoring system with several studies confirming its strong prognostic value.[1,11–14] The ATT score is based on 6 categories (body systems) scored on a scale of 0 to 3 with 0 being normal. In dogs with head trauma, a score of 9 approximated a 50% probability of survival.[11] A recent retrospective study determined the strongest predictor for non-survival was a decreased MGCS. This study also found that poor perfusion, higher ATT score, intubation or the need for hypertonic saline (HTS) were all associated with a worse outcome.[11] While scoring systems may help guide owners and veterinarians, caution must be exercised, as they are not designed to predict survival in individual patients.

Imaging

Extracranial

Head trauma patients often present with concurrent injuries and should be thoroughly evaluated with imaging such as thoracic and abdominal radiographs, abdominal and thoracic focused assessment with sonography for trauma, and/or a full body computed tomography (CT) scan (ie, trauma CT).[15,16]

Intracranial

Intracranial imaging is crucial to identification and treatment of TBI. In the emergency setting, CT scanning is the modality of choice because it does not require general anesthesia, and is accessible, fast, and relatively cost effective.[17] CT scanning is helpful in identifying fractures, parenchymal damage, hemorrhage (intraaxial and extraaxial), and herniation, as well as which patients may require surgical intervention.[5,17] A full body CT can provide information about intracranial injuries as well as systemic injuries without the need to rotate the patient for additional views, and may be similar in cost to multiple radiographic projections.[18]

Although CT is the primary imaging modality for TBI, MRI is more sensitive for smaller lesions. Because MRI requires general anesthesia, takes longer, and is less

Box 1
Modified Glasgow Coma Scale monitoring sheet

Date	Time	MA	BSR	LOC	Total Score	Initials

Motor Activity (MA)

6 = Normal gait/spinal reflexes
5 = Hemiparesis/tetraparesis
4 = Recumbent, intermittent extensor rigidity
3 = Recumbent, constant extensor rigidity
2 = Recumbent, constant extensor rigidity, opisthotonus
1 = Recumbent, depressed or absent spinal reflexes and muscle tone

Brain stem reflexes (BSR)

6 = Normal pupillary light response (PLR), normal oculocephalic reflexes
5 = Slow PLR, normal to depressed oculocephalic reflexes
4 = Bilateral miosis, normal to depressed oculocephalic reflexes
3 = Pinpoint pupils, depressed to absent oculocephalic reflexes
2 = Unilateral, unresponsive mydriasis, depressed to absent oculocephalic reflexes
1 = Bilateral, unresponsive mydriasis, depressed to absent oculocephalic reflexes

Level of consciousness (LOC)

6 = Occasional periods of alertness and responsive to environment
5 = Depression or delirium, capable of responding, but response may be inappropriate
4 = Semicomatose, responsive to visual stimuli
3 = Semicomatose, responsive to auditory stimuli
2 = Semicomatose, responsive only to repeated noxious stimuli
1 = Comatose, unresponsive to repeated noxious stimuli

readily available, it may not be appropriate for unstable patients.[19] A recent study in dogs demonstrated the prognostic value of early MRI in dogs with TBI, including prediction of posttraumatic epilepsy.[20]

PHARMACOLOGIC TREATMENT OPTIONS

Fluid Therapy

Although the optimal fluid type for resuscitation in head trauma is not established, the goals of fluid resuscitation are rapid reversal of hypovolemia, prevention of

hypotension, and maintenance of CBF while avoiding intracranial hypertension. Patients should not be purposefully dehydrated to reduce cerebral edema. Permissive hypotension or hypotensive resuscitation is often used in trauma, but is inappropriate for head trauma. Human guidelines recommend targeting a systolic blood pressure of at least 90 mm Hg. A retrospective study of people with severe brain injury found that a single episode of hypotension (systolic blood pressure <90 mm Hg) was associated with a 150% increase in mortality.[21]

Head trauma presents challenges regarding fluid therapy. In health, the blood–brain barrier (BBB) tightly regulates changes in intracranial volume. After trauma, disruption of the BBB may lead to pathologic fluid shifts, vasogenic edema, and cytotoxic edema. Until BBB disruptions heal, the brain may be less tolerant of fluids and at increased risk for fluid overload. Impaired autoregulation increases the brain sensitivity to changes in volume status. Fluid therapy is further complicated by the lack of specific monitoring for ICP. Striking a balance between optimizing cardiac output and minimizing tissue edema remains a challenge. Numerous fluid choices are available, with their own sets of advantages and disadvantages.[22]

Isotonic crystalloids

Isotonic crystalloids should be titrated to reach goals as discussed. Dose recommendations start with a one-quarter of the shock volume of fluids (20 mL/kg in dogs, 15 mL/kg in cats). Some support 0.9% NaCl because it contains the least amount of free water, but it also an acidifying solution and may worsen acid–base imbalance. Isotonic crystalloids redistribute over the intravascular and interstitial spaces, so large volumes can exacerbate tissue edema.[7]

Hypertonic saline

HTS offers several benefits in head trauma. By rapidly increasing blood osmolality, HTS expands the intravascular space by shifting fluid from the interstitial and intracellular spaces, allowing administration of smaller volumes. Other theoretic benefits of HTS are discussed elsewhere in this article in the hyperosmolar therapy section. Dosage recommendations for 7.5% and 3% NaCl are 4 mL/kg and 5.3 mL/kg, respectively. Although the response to HTS is rapid, fluid redistribution limits duration of action to less than 75 minutes.[7,23]

Colloids

The rationale for colloid administration in head trauma is compelling. By supporting plasma oncotic pressure, colloids should minimize extravasation of fluid from the intravascular space and tissue edema. The duration of action of colloids is longer than crystalloids. However, large metaanalyses have failed to demonstrate a clear benefit of any colloids in any patient group.[24] Post hoc analysis of the landmark SAFE trial (Saline versus Albumin Fluid Evaluation) found that resuscitation with 4% albumin significantly increased mortality compared with 0.9% NaCl in TBI.[25] Although the exact mechanism is unknown, leakage of albumin through a disrupted BBB may create oncotic shifts, promoting edema formation and leading to increased ICP and mortality. There are no large randomized trials regarding synthetic colloids in TBI. The authors infer similar disadvantages as albumin for synthetic colloids until further studies are performed. Other investigators recommend the use of synthetic colloids and some consider them the fluid of choice for head trauma.[4,7] Regardless of fluid type, frequent reassessment and titrating to effect to avoid overload is essential.

Hyperosmolar Therapy

Hyperosmolar agents create an osmotic gradient across the intact BBB to shift water from the interstitial space to the intravascular space to decrease ICP. Mannitol and HTS are routinely used to reduce ICP. Some recent metaanalyses favor HTS, but the choice remains controversial.[26–28]

Mannitol

Mannitol, a sugar molecule, acts as an osmotic diuretic. Immediately, the osmotic effect expands the plasma volume reducing viscosity and improving microcirculatory flow. Persisting for an estimated 75 minutes, the reduction in viscosity causes a reflex vasoconstriction of pial arterioles as does hyperventilation. The osmotic gradient across the BBB forms slowly over 15 to 30 minutes, persists for 2 to 5 hours, and shifts fluid from the brain into the intravascular space. Mannitol may also act as a free radical scavenger.[7] The diuretic effect is not desirable in hypotensive patients and the loss of fluids must be addressed. Patients should be volume resuscitated before mannitol administration.

Extravasation of mannitol owing to ongoing cerebral hemorrhage leading to increased ICP is a common concern, but remains unproven. In people, no difference in outcome has been found between patients with intracerebral hemorrhage that did or did not receive mannitol.[29]

Accumulation of mannitol in the extravascular space leading to a "reverse osmotic shift" is unlikely with appropriate dosing. The benefits of mannitol far outweigh the potential risks. Recommended dosing is 0.5 to 1.0 g/kg intravenously over 15 to 20 minutes.[4] Historically, furosemide was administered concurrently in hopes of decreasing cerebrospinal fluid production, counteracting the initial plasma expansion, and potentiation of the osmotic gradient.[30] However, these benefits are unproven and furosemide may increase the risk of dehydration and hypovolemia.[31]

Hypertonic Saline

HTS shares similar mechanisms with mannitol including expanding the plasma volume and reducing viscosity. Proposed advantages of HTS over mannitol include volume expansion leading to improved cardiac output and blood pressure, a reduced likelihood of HTS crossing the BBB, improved regional CBF by reducing endothelial swelling, and modulation of neuroinflammatory pathways.[32–34] HTS is less desirable in a hyponatremic patient. Recommended dosing is 4 mL/kg and 5.4 mL/kg for 7.5% and 3% NaCl, respectively.[7] Although the debate continues between mannitol versus HTS, HTS seems ideal for the hypovolemic patient; both are reasonable in the euvolemic patient. If the patient fails to respond to one, the other should be considered.

Anesthetics, Analgesics, and Sedatives

Analgesia is essential in management of head trauma, and anesthesia is often required for procedures such as surgery, diagnostic imaging, and mechanical ventilation. Because pressure autoregulation may be lost after trauma, the brain is particularly vulnerable to hypotension and alterations in $Paco_2$. A balanced approach minimizes the risk of secondary injury and provides analgesia without excessive sedation.[35] Inhalant anesthetics have a dose-related effect on ICP. As concentrations increase greater than 1.0 to 1.5 the minimum effective alveolar concentration, ICP increases.[36] Anesthesia-induced hypoventilation and hypercapnia also increases ICP. The risk of increasing ICP can be minimized by titrating inhalants to effect and providing

adequate ventilator and cardiovascular support. At lower concentrations, the vasodilatory effects of inhalants may improve cerebral perfusion.[37]

If the ICP is increased, inhalants are contraindicated and total intravenous anesthesia is recommended.[35] Direct comparison studies have shown that injectable anesthetics such as propofol improve cerebral perfusion and maintain pressure autoregulation better than inhalant anesthetics.[38–41] Propofol may also be neuroprotective via modulation of GABA receptors and antioxidant effects.[42] However, propofol may also cause hypotension and hypoventilation. Careful titration, meticulous monitoring, and supportive care are essential.

Analgesia is essential for patient comfort and preventing further increases in ICP. Pain and anxiety increase cerebral metabolic rate, which increases CBF, cerebral blood volume, and ultimately ICP. Opioids are the analgesic of choice because they are cardiovascular sparing and easily reversible. Respiratory depression is possible, but minimized with careful titration. The patient's ability to protect their airway (gag reflex and/or ability to swallow) should be assessed frequently to decrease the risk of aspiration pneumonia. Continuous rate infusions of full mu agonists such as fentanyl are recommended to provide consistent analgesia and to avoid the adverse effects seen at higher blood levels. Recommended dosing for fentanyl is 2 to 6 μg/kg/h. Opioid agonist/antagonists such as buprenorphine cause less cardiovascular, respiratory, and central nervous system depression, but provide only moderate analgesia and are more difficult to reverse.[35] Having less sedation allows more accurate patient assessment and may be particularly important in patients with subtle changes or at risk for rapid changes in neurologic status.

Benzodiazepines are a valuable adjunct to the balanced approach. They provide anxiolysis and sedation with minimal intracranial, cardiovascular, and respiratory effects. They also enable dose reduction of other agents, such as propofol, minimizing adverse effects.[35] Midazolam, but not diazepam, significantly reduced the propofol dose required for intubation in dogs, whereas both were effective in cats.[43,44]

Ketamine is an anesthetic drug with potent analgesic and hypnotic action. By inhibiting the NMDA receptor, ketamine may have neuroprotective effects as NMDA receptor signaling plays a key role in neuronal death.[45] Other potential advantages are stimulation of the cardiovascular system and minimal respiratory depression. The benefits of ketamine have led to reexamination of its role in neurotrauma. Historically, ketamine was contraindicated owing to drug-induced increases in ICP. Older literature performed in patients with nontraumatic intracranial lesions showed an increase in ICP, and ketamine's supposed effect on ICP was perpetuated in anesthetic texts and literature.[46–49] However, recent studies of ketamine use in TBI do not support the increase in ICP.[45] Other studies in human TBI show higher mean CPP and lower vasopressor requirements with ketamine.[50,51] Ketamine may be a useful adjunct in veterinary head trauma, but further studies are warranted before specific recommendations can be made.

Alpha-2 agonists are easily reversible and provide sedation, anxiolysis, and analgesia without respiratory depression. However, their use in head trauma is controversial. Although dexmedetomidine may have neuroprotective properties, clinical studies in human patients with severe TBI have been mixed.[52–55] A 2016 metaanalysis of dexmedetomidine concluded that although the literature was limited in quantity and quality, dexmedetomidine seems to be both efficient and safe as a sole or adjunct agent in human neurocritical care patients.[56] However, dexmedetomidine was associated with significantly more hypotension in a prospective human study with 198 patients.[57] In a study with isoflurane-anesthetized dogs, dexmedetomidine significantly decreased

CBF and cardiac output, but without evidence of global cerebral ischemia.[58] Medetomidine did not increase ICP in healthy dogs under isoflurane anesthesia.[59] Given the mixed results and the risk of clinically significant reductions in heart rate and cardiac output, alpha-2 agonists should be avoided unless analgesics with fewer adverse effects are unavailable or are providing inadequate pain relief.[7] Recommended dosing is listed in **Table 1**.

Anticonvulsants

In human medicine, there is an established correlation between the severity of TBI and the development of posttraumatic seizures as well as an increased incidence of epilepsy in TBI patients compared with the general population.[60–62] In veterinary patients, the incidence is less well-documented, but a recent study found a higher epilepsy rate in dogs (3.5%–6.8%) with head trauma as compared with a standard population epilepsy rate of 1.4%.[63]

Seizures may occur at different time points relative to the injury, either early (within 7 days) or late (after 7 days). Preventing seizure development may limit the detrimental effects of seizure activity such as increased ICP and increased metabolic demands.

A recent Cochrane review evaluated prophylactic antiepileptic medications for the prevention of early and late seizures and impact outcome in humans.[64] There was minimal evidence that seizure prophylaxis reduced early seizures and no evidence in reduction of late seizures or improvements in outcome. The current recommendation is treatment for 1 week.[65] There are no studies in veterinary medicine investigating the use of seizure prophylaxis in a population of TBI patients. If seizures develop, it is reasonable to initiate emergency treatment with benzodiazepines followed by a maintenance antiepileptic medication. In the authors' experience, continued neurologic assessment may be easier to facilitate with levetiracetam.

Corticosteroids

Based on previous experimental evidence, steroids were often used in the treatment of TBI.[66,67] However, the results of the CRASH trial demonstrated an increased risk of death at both 2 weeks and 6 months in human adults.[68] Corticosteroids are not recommended for TBI patients.

Gastric Ulcer Prophylaxis

Patients with neurologic injury, including TBI, are at an increased risk of gastric ulceration and bleeding.[69] A recent metaanalysis concluded that ulcer prophylaxis with

Table 1
Recommended doses for anesthetics, analgesics, and sedatives

Drug	Recommended Dose
Propofol	1–6 mg/kg, then 100–400 μg/kg/min
Fentanyl	Dogs: 2–6 μg/kg, then 2–6 μg/kg/h Cats: 1–3 μg/kg, then 1–3 μg/kg/h
Buprenorphine	0.01–0.02 mg/kg q8h
Midazolam	0.1–0.5 mg/kg
Ketamine	0.1–1.0 mg/kg, then 2–10 μg/kg/min
Dexmedetomidine	0.5–3 μg/kg/h, then 0.5–1 μg/kg/h

either proton pump inhibitors or histamine-2 receptor antagonists was effective in preventing gastrointestinal bleeding in humans. There was no increase in the risk of nosocomial pneumonia.[70] In veterinary medicine, drugs including proton pump inhibitors, such as pantoprazole or omeprazole, and histamine-2 receptor antagonists, such as famotidine may be used.

NONPHARMACOLOGIC TREATMENT OPTIONS

Oxygen and Ventilation

Respiratory compromise may result from pneumothorax, pulmonary contusion, aspiration pneumonia, or an abnormal respiratory drive. Normal oxygenation and ventilation are the goals of treatment. Hyperoxygenation and hyperoxemia may worsen reperfusion injury. Oxygen should be titrated to achieve normoxemia (Pa_{O_2} >80 mm Hg and SpO_2 >94%).[7]

Oxygenation supplementation should be individualized. Most patients tolerate flow-by with or without a mask during initial assessment and stabilization. Nasal cannulas are effective, but may cause sneezing or coughing, which can increase ICP. Nasal cannulas should be used as a last resort. Apply care when placing nasal cannulas in patients with head trauma. The distal tip of the catheter should not extend past the medial canthus because possible fractures may allow communication with the cranial vault. Oxygen cages may be ineffective if frequent or constant monitoring is required. Intubation or temporary tracheostomy should be considered in stuporous or comatose patients and those lacking a gag reflex.[4]

Under normal conditions, Pa_{CO_2} is the most powerful determinant of CBF. CO_2 affects ICP by regulating vessel tone and diameter. Between 20 and 80 mm Hg, CBF changes linearly with Pa_{CO_2}.[71] Decreases in Pa_{CO_2} lead to vasoconstriction, with a Pa_{CO_2} of less than 30 mm Hg causing excessive vasoconstriction, low CBF, and cerebral ischemia. Conversely, high Pa_{CO_2} leads to excessive CBF, increased CBF, and worsened ICP.[7] Hypoventilation and increased CO_2 may occur from damage to the respiratory center, oversedation, thoracic pain, mechanical airway obstruction, or respiratory muscle fatigue or paralysis.[72]

Prophylactic hyperventilation is not recommended. After trauma, ischemia from excessive vasoconstriction is common.[73] Hyperventilation worsens ischemia and secondary injury by promoting vasoconstriction. Subsequent alkalosis with a leftward shift in the oxygen–hemoglobin dissociation curve decreases oxygen delivery. Numerous studies have shown a poorer outcome with prophylactic hyperventilation during initial resuscitation in humans.[74,75] Normoventilation (Pa_{CO_2} 35–40 mm Hg) is recommended. Short-term conservative hyperventilation (Pa_{CO_2} >30 mm Hg) should only be used to reduce increased ICP. Guidelines for human patients recommend avoiding hyperventilation in the first 24 hours after trauma when CBF may be critically reduced.[71]

Nutrition

Head trauma is associated with a hypermetabolic and hypercatabolic state and early nutritional support is essential. Early enteral nutrition maintains gastrointestinal integrity, improves immune function, and attenuates the metabolic response to stress.[76] A retrospective study of 797 humans with severe TBI found that early nutrition (within 5 days) reduced 2-week mortality and the amount of nutrition was inversely correlated with mortality.[77]

The patient's ability to protect their airway, tolerance of a tube placement procedure, and anticipated duration of use must be considered when choosing the method

of providing nutrition. In stable patients, esophagostomy tubes are well-tolerated and associated with few complications.[78] Owners can be trained to use and maintain these tubes at home, allowing for at-home care.

Parenteral nutrition should be considered in patients at risk for aspiration.[4] Commercial products are available and well tolerated in hospitalized dogs in intensive care.[79]

Head Elevation

Mild head elevation of less than 30° is associated with decreases in ICP and increases in CPP without affecting MAP. Mild head elevation does not compromise cerebral oxygenation.[80] In veterinary patients, a rigid back board may be used to avoid kinking the neck and compressing the jugular veins, which could lead to increased ICP. See **Fig. 2** for an example of head elevation.

Therapeutic Hypothermia

The mechanisms that lead to secondary brain injury are inhibited by hypothermia. Apoptosis, excitotoxicity, increases in inflammatory mediators, free radical formation, microcirculatory dysfunction, and other mechanisms are implicated.[81] Therapeutic hypothermia (32°C–34°C), may protect against secondary injury. In humans, therapeutic hypothermia is the standard of care for patients after cardiac arrest and stroke and may be used in TBI with intracranial hypertension and status epilepticus.[82,83] However, a recent study of humans with intracranial hypertension treated with therapeutic hypothermia in addition to standard care failed to demonstrate benefit.[84] At this time, there is only 1 report of a veterinary TBI patient treated with therapeutic hypothermia.[85] At the authors' institution, hypothermic TBI patients are allowed to passively rewarm, but are not actively cooled.

Glycemic Control

In human patients, hyperglycemia leads to increases in mortality and duration of hospitalization, and worse neurologic outcome owing to accelerated secondary brain injury.[86] In veterinary patients, hyperglycemia is an indication of the severity of the injury, but not necessarily a prognostic indicator.[87] Studies do not support the use of insulin protocols owing to possible hypoglycemia.[88] See **Fig. 3** for an overview of patient stabilization.

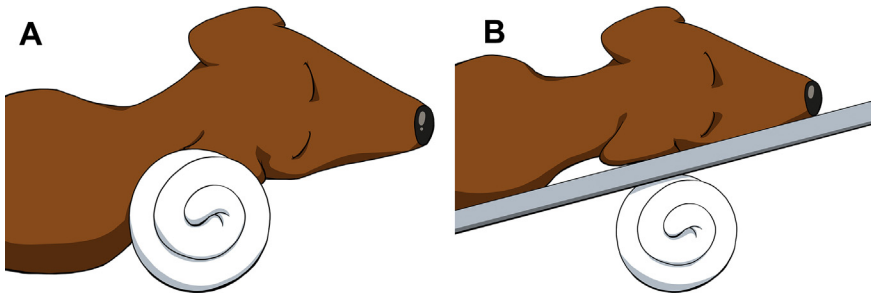

Fig. 2. Elevation of the head and neck is associated with decreases in intracranial pressure and increased cerebral perfusion pressure. (*A*) Inappropriate head elevation using a rolled up towel may lead to kinking of the neck, which can compress the jugular veins and increase intracranial pressure. (*B*) A rigid backboard allows head elevation without risk of compressing the jugular veins. (*Courtesy of* Silas Lee, Department of Information and Instructional Technology, Auburn, AL.)

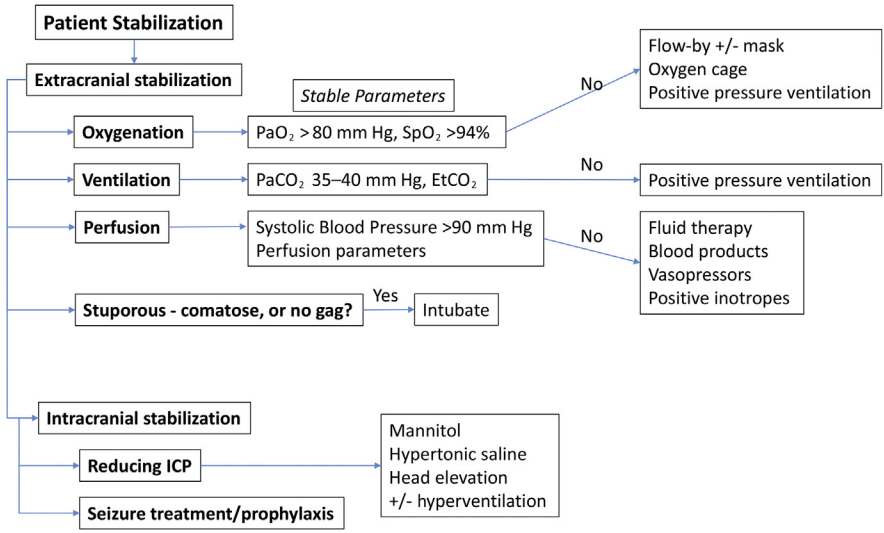

Fig. 3. Overview of patient stabilization after head trauma. $EtCO_2$, end-tidal carbon dioxide; ICP, intracranial pressure.

SURGICAL TREATMENT OPTIONS

Decompressive craniectomy may be performed in patients with refractory intracranial hypertension at risk for cerebral herniation.[89] A recent trial in human TBI patients comparing decompressive craniectomy with standard medical therapy for intracranial hypertension resulted in a lower mortality rate in the surgery group.[90] In veterinary patients, decompressive craniectomy should be considered in those failing aggressive medical therapy or a compressive lesion from fracture or hemorrhage.[91]

MONITORING

Repeated Physical and Neurologic Examinations

Frequent reassessments should be performed to direct therapy and diagnostics. Examinations should be performed hourly for the initial 6 to 12 hours with a gradual lessening of assessments as the patient stabilizes.

Intracranial Pressure

In human TBI patients, ICP monitoring is often used in titrating therapies to treat intracranial hypertension. However, a recent study comparing patients treated for intracranial hypertension with either ICP monitoring or clinical and radiographic information concluded that neither was superior in terms of outcome.[92] There are few studies evaluating ICP monitoring systems in dogs and cats, because expense and invasiveness preclude widespread use.[93–95]

Electrocardiography

Traumatic myocarditis causing arrhythmias is common in blunt trauma patients.[1,96] Patients presenting with evidence of concurrent thoracic trauma should be monitored for 24 to 48 hours.

Blood Pressure

Blood pressure should be monitored to ensure a minimum systolic blood pressure of 100 mm Hg to promote cerebral perfusion. Blood pressure should also be monitored if the patient becomes bradycardic to determine if the patient is experiencing a Cushing's reflex.

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