Nursing Care & Triage for HEAD TRAUMA PATIENTS

Oriana D. Scislowicz, BS, LVT

Although animals with head trauma are frequently presented to emergency hospitals, veterinary teams at general practices encounter these patients as well. Therefore, understanding triage and emergency assessment and treatment of head trauma is important for every veterinary professional in practice.

TYPES OF HEAD TRAUMA
Head trauma often results from falls, gunshot wounds, car crashes, and altercations with other animals.

When assessing a head trauma patient, it is helpful to understand the differences between primary and secondary head injuries.

Primary head trauma immediately follows impact and consists of direct damage to the brain parenchyma, such as contusions, lacerations, and diffuse axonal injury. There also may be damage to blood vessels in the brain, which can cause subsequent intracranial hemorrhage and vasogenic edema (Table 1).

Secondary injuries result from increased intracranial pressure—the pressure exerted within the skull by hemorrhage and swollen brain tissue—that causes further damage by stimulating various biochemical pathways. The primary mediators that become involved in this injury include nitric oxide, glutamate, and oxygen free radicals.

When inflammation and bleeding occurs within the brain, cerebrospinal fluid—the fluid that bathes the spinal column and brain—and intracranial venous blood are directed out of the skull and back into the body in order to compensate for the other space occupying lesions. If the body has already exhausted all of its compensatory mechanisms and intracranial pressure continues to rise, intracranial hypertension can develop.

INITIAL STABILIZATION
1. Stabilize the ABCs (airway, breathing, and circulation)—the most important step upon a head trauma patient’s admittance to the hospital. Ensure that the airway is patent by:
   • Observing the respiratory pattern
   • Determining whether breathing appears normal
   • Confirming appropriate airflow.
   During assessment, check circulation, including evaluation for pulse deficits, hypovolemia or dehydration.
2. Do not forget pain—the fifth “vital sign.” Addressing pain provides some relief to the patient and aids in the recovery process.

<table>
<thead>
<tr>
<th>TABLE 1. Types of Cerebral Edema</th>
</tr>
</thead>
<tbody>
<tr>
<td>TYPES</td>
</tr>
<tr>
<td>Cytotoxic</td>
</tr>
<tr>
<td>Interstitial</td>
</tr>
<tr>
<td>Osmotic</td>
</tr>
<tr>
<td>Vasogenic</td>
</tr>
</tbody>
</table>

Vital Signs
- Blood pressure
- Heart rate
- Pain
- Respiratory rate
- Temperature
• Increased blood pressure can cause alarm because it may be caused by an increase in intracranial pressure, especially if accompanied by bradycardia.
• However, pain may be the underlying cause of hypertension and should be assessed and managed during stabilization.

3. Establish IV access and assess blood pressure; then consider administering fluids while the patient is being stabilized.
• The goal of volume resuscitation with colloids or hypertonic saline is to achieve a mean arterial pressure (MAP) of 80 to 100 mm Hg (or 120–150 mm Hg systolic).
• Cardiovascular support is important because cerebral perfusion pressure depends greatly on MAP. In particular, if intracranial pressure increases, this support is critical.
• IV catheterization helps facilitate rapid administration of medications, such as mannitol, which aids in decreasing intracranial pressure.²,³

4. Auscultate the patient’s lungs and observe the respiratory pattern, which can provide information with regard to the location of brain injury (Table 2), although diagnostics, such as magnetic resonance imaging, provide the most complete picture of brain trauma (see page 38, Advanced Imaging: Its Place in General Practice). To help prevent respiratory and cardiac arrest, if breathing abnormalities are present, consider:²
  • Providing oxygen supplementation
  • Intubating the patient
  • Providing continuous ventilation.

5. Assess oxygenation via pulse oximetry or arterial blood gas analysis. Keep in mind that, even if the patient is not cyanotic, it may be unstable and hypoxic.⁴ SpO2 levels (percentage of hemoglobin in blood saturated with O₂) should be greater than, or equal to, 95%.

Table 2. Head Trauma Patients: Abnormal Breathing & Location of Injury²

<table>
<thead>
<tr>
<th>ABNORMAL BREATHING</th>
<th>POTENTIAL LOCATION OF INJURY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheyne-Stokes breathing pattern (hyperpnea, with phases of apnea)</td>
<td>Severe cerebral or rostral brainstem lesions</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Midbrain lesions</td>
</tr>
<tr>
<td>Irregular breathing patterns and apnea</td>
<td>Medulla oblongata lesions</td>
</tr>
<tr>
<td>Rapid and shallow breathing pattern</td>
<td>Pontine lesions</td>
</tr>
</tbody>
</table>

Figure 2. Two-year-old neutered male German shepherd that presented with suspected severe head trauma after being found outside the owner’s home, lying on his side, unresponsive, and bleeding from the head, nose, and mouth; a large tree branch was lying on the ground several feet away. Note puncture wound over right temporalis muscles and swelling of the right temple and peri-ocular region. Patient was hospitalized, with a ventilator for oxygenation, and received IV fluids, pain medication, and mannitol therapy to manage brain swelling.

Once the patient is stable, a more thorough physical examination can be completed. Make sure to avoid:
• Accidental displacement of fractures and/or exacerbation of spinal injuries by failing to be careful when manipulating the head and neck.
• Pressure on, and blood collection from, the jugular vein, both of which can decrease venous return from the brain, which increases intracranial pressure.

1. Assess level of consciousness (Table 3)—the first step in the physical examination.¹

2. Examine the patient’s eyes, which provides a multitude of information, including severity of brain injury.
• Strabismus and nystagmus: If strabismus is present, the cranial nerves or brainstem may be damaged. If physiologic nystagmus is absent, severe brainstem damage may be present. However, lack of physiologic nystagmus in a comatose patient does not necessarily indicate brainstem damage.
• Pupillary light response (PLR): A slow PLR usually indicates a guarded to poor prognosis; an absent PLR indicates a grave prognosis.
• Pupil size and behavior: Pupil size, along with PLR, can help evaluate a patient’s status and prognosis.
  » Miotic, or “pinpoint,” pupils usually result from cerebral injury or edema, and indicate a guarded to fair prognosis.
  » Hydriatic pupils can indicate stress, ophthalmic disease, and use of certain medications, such as atropine.
In rare circumstances, they may indicate impending cardiopulmonary arrest. Unilateral, then bilateral, unresponsive mydriatic pupils (bilateral being worse) indicate a poorer prognosis than miotic pupils per the Modified Glasgow Coma Scale.

» **Anisocoria** often signals oculomotor nerve damage or compression, direct eye injury, and/or uveitis.

» **Pupils that change** from miotic to mydriatic and become unresponsive to light signal brain herniation.

» **Mid-size pupils** that are unresponsive to light point to a brainstem injury, and indicate a grave prognosis.

- **Menace response:** If the patient appears blind, the eye, optic nerve, or brain may be dysfunctional. The menace response should result in the patient blinking. When performing this test, do not move too much air toward the eye, which can create a false positive. Lack of menace response may be due to:
  » Eye, optic nerve, or brain trauma or dysfunction of the facial nerve
  » The animal being obtunded (Table 3)
  » Patient age—many neonates have not yet developed a menace response.²

3. **Evaluate body position** and monitor posture closely—minute changes often indicate an injury that is becoming worse.

- **Opisthotonus:** Patients affected by this condition have severe hyperextension, with the head, neck, and spinal column arched. Opisthotonus in head trauma patients often indicates severe brain injury and, therefore, a grave prognosis.

- **Schiff-Sherrington posture:** In patients with Schiff-Sherrington posture, which usually manifests as thoracic limb extensor rigidity, a thoracolumbar lesion often is present.

- **Decerebellate posture:** This posture, characterized by extension of the thoracic limbs and flexion of the pelvic limbs, can indicate cerebellar lesions or herniation.

- **Decerebrate rigidity:** This posture, characterized by rigid extension of all limbs and opisthotonus (extension of the head and neck) associated with a stuporous or comatose mental status, has a less promising prognosis than decerebellate posture.²

4. **Evaluate the chest and abdomen** for pulmonary contusions, pneumothorax, bone fractures, and abdominal injuries, all of which may be seen in patients presenting with head trauma. Abnormal SpO₂ and auscultation, which should be identified during initial stabilization, may help detect respiratory injuries. Radiographs and ultrasonography may prove useful in evaluation of traumatic injuries.³

### DIAGNOSTICS & TREATMENT

Once a patient has been stabilized and assessed, and had a thorough physical examination, further diagnostics can be pursued.

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>PHYSICAL EXAMINATION RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alert &amp; Responsive</td>
<td>Normal behavior</td>
</tr>
<tr>
<td>Obtunded</td>
<td>Response to stimuli decreased; patient awake</td>
</tr>
<tr>
<td>Stuporous</td>
<td>Response to painful/noxious stimuli limited</td>
</tr>
<tr>
<td>Comatose</td>
<td>Response to stimuli nonexistent; patient unconscious</td>
</tr>
</tbody>
</table>

---

**Figure 3.** MRI from Figure 2 patient shows T2 (axial [A] and sagittal [B] views); T1 pre and post contrast, GRE, FLAIR, and proton density sequences were also obtained and showed evidence of severe brain trauma along with contracoup injury; multifocal inflammation and hemorrhage can be seen in the forebrain, thalamus, and brainstem, and a fracture is present on the frontal sinus.
Routine Blood Analysis
Blood can be drawn (but not from the jugular vein) for blood cell counts, chemistry panels, and venous and arterial blood gas values:
• **Packed cell volume (PCV)** and total solids assess for the presence of hemorrhage.
• **Blood gas analysis** assists in evaluating ventilation, oxygenation, acid–base status, and perfusion.
• **CO₂ levels** help monitor changes in respiratory function as a result of intracranial pressure changes or trauma to brainstem respiratory centers. Note that, currently, there is no easy, noninvasive way to measure intracranial pressure.

Brainstem Integrity Tests
Several brainstem integrity tests can be performed:
• **A caloric test** lavages warm water into the external ear canal. The observer looks for nystagmus; if present, it most likely indicates that the medulla oblongata, pons, and midbrain are intact.
• **Brainstem auditory evoked response (BAER) testing** detects electrical activity in the cochlea and auditory pathways in the brain; abnormal results may indicate damage to the brainstem.
• **Electroencephalography (EEG)** helps determine the integrity of the cerebral cortex and brain death. CSF analysis should not be performed on head trauma patients because it increases the risk of brain herniation. 5

Medical Therapy
**Fluids** should be given throughout the course of treatment for head trauma patients. Use crystalloid fluids with caution because they can exacerbate cerebral edema.

- **Mannitol or hypertonic saline** is used to treat increased intracranial pressure. Mannitol is chosen to treat intracranial pressure in cardiovascularly stable patients, while hypertonic saline is chosen for patients with intracranial pressure accompanied by shock or hypovolemia because it greatly expands intravascular volume. See Table 4 for dosages and preparation. Remember that:
  • Mannitol will cause dramatic diuresis
  • Hypertonic saline may not be the best choice for patients experiencing hyponatremia or hypernatremia because it can rapidly increase sodium levels, harming brain tissue. 2

**Furosemide** can be used in conjunction with mannitol to help manage initial expansion of intravascular volume following mannitol administration. See Table 4 for dosage.

### Table 4. Medical Therapy: Dosages & Preparation

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>DOSAGE</th>
<th>PREPARATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>Give single dose (0.7 mg/kg) 15 minutes following administration of mannitol. 1</td>
<td></td>
</tr>
<tr>
<td>Hypertonic Saline 7.5%</td>
<td>4 mL/kg²</td>
<td>Dilute 23% hypertonic saline solution with a colloid solution to create 7.5% hypertonic saline.</td>
</tr>
<tr>
<td>Mannitol</td>
<td>0.25 to 2 g/kg: IV bolus administered over 10–20 min; can be repeated Q 4–6 H</td>
<td>Warm mannitol, ideally on a fluid warmer covered with a towel or drape; apply a 0.22 mcm Hemo-Nate filter (utahmed.com) to the end of the syringe, between syringe and needle.</td>
</tr>
</tbody>
</table>
Monitor furosemide usage closely—it can lead to cerebral ischemia by depleting intravascular fluid volume.2

Surgical Therapy
In head trauma patients, surgery can help patients that have hematomas and, sometimes, skull fractures (identified by imaging). However, in contrast to humans, subdural hematomas are not the most common type of intracranial hemorrhage in dogs; instead, dogs have more evidence of contusions, which cannot be treated surgically. Patients requiring surgery should be referred to a surgeon who specializes in this area of veterinary medicine.

Monitoring
As with other critical patients, animals with head trauma should have the following monitored:
• Mucous membranes and capillary refill time
• Heart rate
• Respiratory rate and effort
• Pulse rate and quality
• Temperature and blood pressure.

1. Monitor blood pressure, which is critical in head trauma patients because hypotension results in decreased cerebral perfusion and, subsequently, brain ischemia.

2. Beware of the Cushing’s reflex—a response to increased intracranial pressure that results in reduced heart rate and increased blood pressure. If the veterinary technician suspects its presence, the attending veterinarian should be notified promptly because a Cushing’s reflex can be a sign of imminent brain herniation.

3. Check body temperature regularly because patients with brain injuries may have difficulty regulating their own temperature. Provide outside heat or cooling support as needed.

4. Monitor level of awareness, pupil size, and PLR regularly. Hypovolemic patients may initially present with an overall decreased mental status. When providing IV fluids to these patients, it is important to regularly check their level of awareness and mentation.

Prognosis
The prognosis for head trauma patients can range greatly, depending on the severity of injury. However, it is possible, especially with thorough care, to nurse these patients back to a quality of life acceptable to their owners and even, in some cases, a full recovery. Improvements can continue over the following 9 to 12 months. However, for up to 2 years, post-injury patients can experience epilepsy as a result of head trauma.3

In summary
Caring for patients with head trauma can be exceptionally rewarding for veterinary team members due to the high level of nursing care required and the strong connection created between the patient and veterinary caregiver during recovery. There is also the opportunity to share knowledge with pet owners, most of whom will be providing nursing care at home. This creates a strong bond between pet owners, patients, and the veterinary team, which most team members consider one of the most rewarding aspects of their careers.

ABC = airway, breathing, circulation; BAER = brainstem auditory evoked response; CO₂ = carbon dioxide; CSF = cerebrospinal fluid; EEG = electroencephalography; MAP = mean arterial pressure; O₂ = oxygen; PCV = packed cell volume; PLR = pupillary light response

References

Oriana D. Scislowicz, BS, LVT, is a veterinary technician in a neurology specialty practice in Richmond, Va. She received her BS in psychology from Virginia Commonwealth University and her AAS from Blue Ridge Community College. She is the President Elect of the Virginia Association of Licensed Veterinary Technicians.