



Nursing Care & Triage for HEAD TRAUMA PATIENTS

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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 fur-

TABLE 1. Types of Cerebral Edema

TYPES	RESULTS FROM:
Cytotoxic	Damaged cellular membranes Failure of cellular ion pumps
Interstitial	Rupture of the cerebrospinal fluid-brain barrier
Osmotic	Abnormal pressure gradient within the brain (water moves into brain)
Vasogenic	Vasodilation and failure of the blood-brain barrier

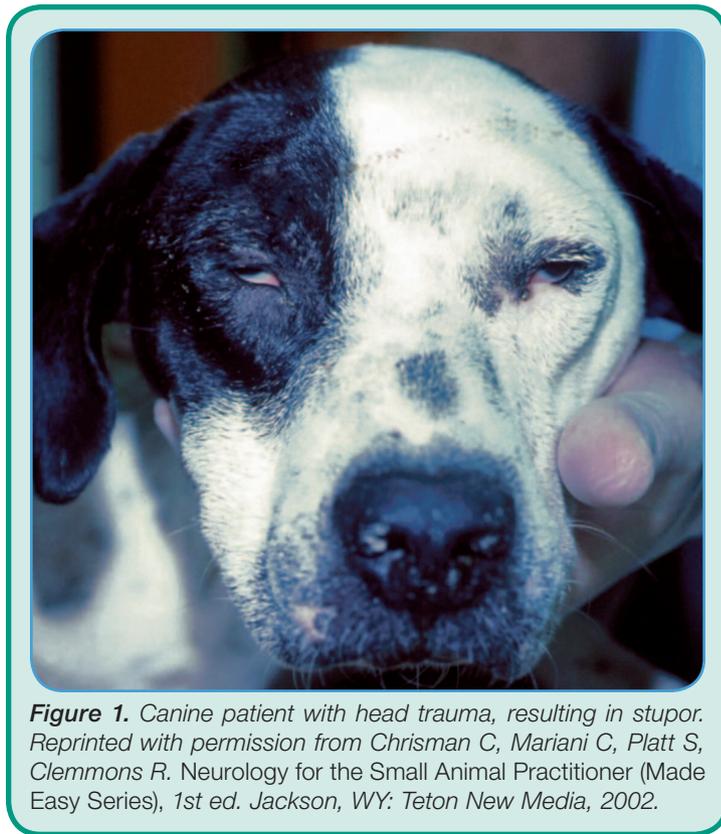


Figure 1. Canine patient with head trauma, resulting in stupor. Reprinted with permission from Chrisman C, Mariani C, Platt S, Clemmons R. Neurology for the Small Animal Practitioner (Made Easy Series), 1st ed. Jackson, WY: Teton New Media, 2002.

ther damage by stimulating various biochemical pathways. The primary mediators that become involved in this injury include nitric oxide, glutamate, and oxygen free radicals.¹

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.²

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.

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%.

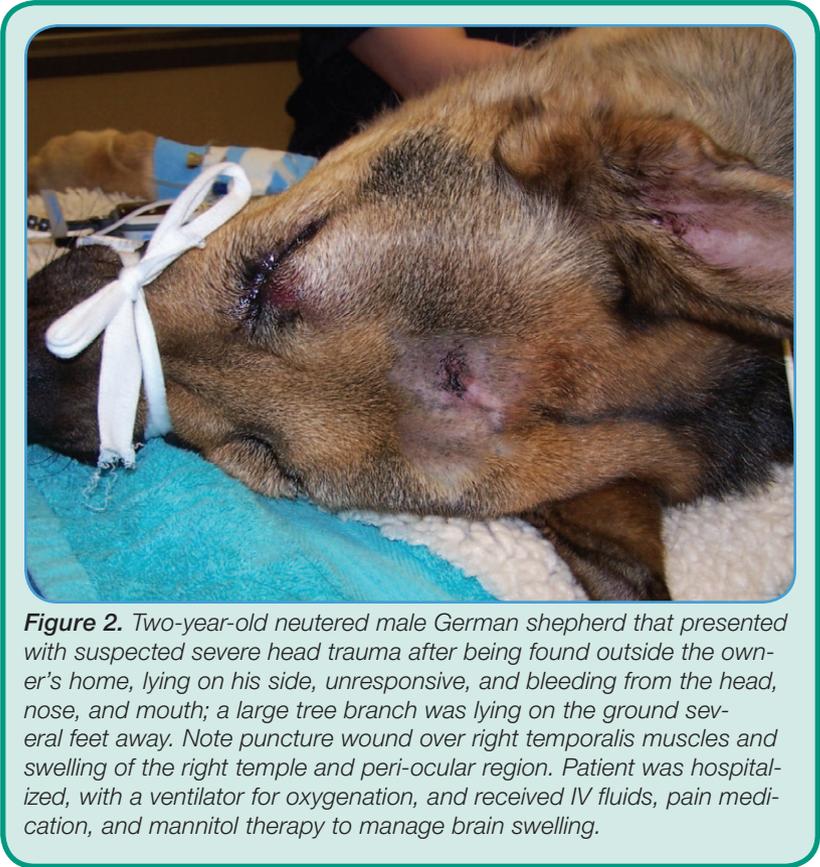


Figure 2. Two-year-old neutered male German shepherd that presented with suspected severe head trauma after being found outside the owner's home, lying on his side, unresponsive, and bleeding from the head, nose, and mouth; a large tree branch was lying on the ground several feet away. Note puncture 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.

ABNORMAL BREATHING	POTENTIAL LOCATION OF INJURY
Cheyne-Stokes breathing pattern (hyperpnea, with phases of apnea)	Severe cerebral or rostral brainstem lesions
Hyperventilation	Midbrain lesions
Irregular breathing patterns and apnea	Medulla oblongata lesions
Rapid and shallow breathing pattern	Pontine lesions

PHYSICAL EXAMINATION

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.
 - » *Mydriatic pupils* can indicate stress, ophthalmic disease, and use of certain medications, such as atropine.

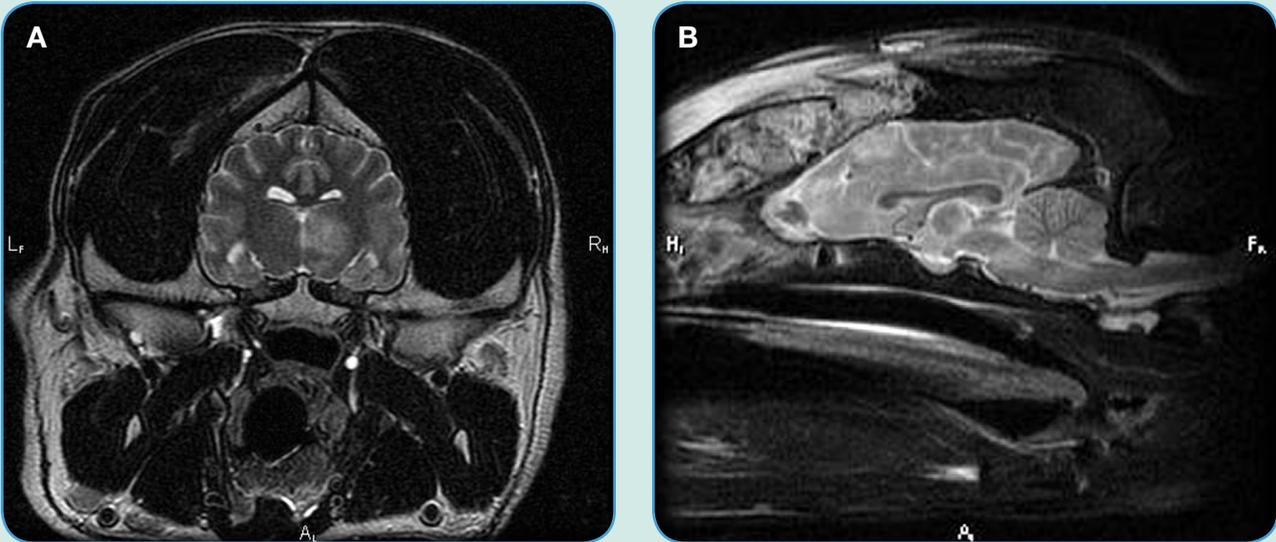


Figure 3. MRI from Figure 2 patient shows T2 (axial [A] and sagittal [B] views); T1 pre and post contrast, GRE, FLAIR, and proton density sequences were also obtained and showed evidence of severe brain trauma along with contrecoup injury; multifocal inflammation and hemorrhage can be seen in the forebrain, thalamus, and brainstem, and a fracture is present on the frontal sinus.

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.⁵

DIAGNOSTICS & TREATMENT

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

TABLE 3. Levels of Consciousness

LEVEL	PHYSICAL EXAMINATION RESULTS
Alert & Responsive	Normal behavior
Obtunded	Response to stimuli decreased; patient awake
Stuporous	Response to painful/noxious stimuli limited
Comatose	Response to stimuli nonexistent; patient unconscious

EIGHT STEPS OF NURSING CARE

1. **Place an IV catheter** immediately after initial assessment of patients that have experienced head trauma (also discussed in Step 3 under **Initial Stabilization**).
2. **Elevate the cranial end of the body**, not just the head, by 30 to 40 degrees, which helps decrease intracranial pressure and decreases the risk of aspiration pneumonia. If only the head is elevated, kinking the neck, the jugular veins may become restricted, causing intracranial pressure to increase.
3. **Place patients in a cage or kennel** with ample bedding and rotate the patient every 4 hours to help prevent decubital ulcers.
4. **Conduct range-of-motion exercises** every 6 to 8 hours to help avoid muscle wasting because these patients are unable to move normally or exercise.
5. **Treat eyes** with ocular wash and artificial tear ointment every 4 hours to provide lubrication for patients that may be unable to blink, which keeps ulcers and dry eye from developing.
6. **Wipe out the oral cavity** of comatose patients every 4 to 6 hours with water or an oral cleansing spray; these patients may have difficulty swallowing, resulting in saliva and debris buildup. Diluted liquid glycerin can help keep the mouth moist, while a suction machine can remove larger amounts of secretions.
7. **Express the bladder** every 3 to 6 hours, or place a urinary catheter if the patient is unable to walk or stand and eliminate. Monitor urine output every 4 hours to ensure the patient is producing adequate amounts of urine.
8. **Hand feed patients** every 4 to 6 hours while they are in a sternal position. If the patient is unable to swallow, consider placing a feeding tube and then administer a gruel through the tube every 4 to 6 hours.² Avoid nasogastric tubes because they cause irritation to the nares, which may cause sneezing and, subsequently, an increase in intracranial pressure.

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

MEDICATION	DOSAGE	PREPARATION
Furosemide	Give single dose (0.7 mg/kg)	15 minutes following administration of mannitol. ¹
Hypertonic Saline 7.5%	4 mL/kg⁶	Dilute 23% hypertonic saline solution with a colloid solution to create 7.5% hypertonic saline.
Mannitol	0.25 to 2 g/kg: IV bolus administered over 10–20 min; can be repeated Q 4–6 H ⁵	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.

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.⁵

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

1. Platt S, Garosi L. *Small Animal Neurological Emergencies*, 1st ed. London: Manson Publishing, 2012, pp 363-382.
2. Terry B. Head trauma. *Veterinary Technician Journal* 2010; 31(12). Available at <https://www.vetlearn.com/veterinary-technician/head-trauma-ce>.
3. Campbell M. *Traumatic brain injury*. CVC San Diego Proceedings 2010.
4. Basilio P. What to assess when triaging patients with head trauma. *Vet Forum* 2008. Available at <https://www.vetlearn.com/veterinary-forum/what-to-assess-when-triaging-patients-with-head-trauma>.
5. Chrisman C, Mariani C, Platt S, Clemmons R. *Neurology for the Small Animal Practitioner (Made Easy Series)*, 1st ed. Jackson, WY: Teton New Media, 2002, pp 50-54.
6. Lorenz M, Coates J, Kent M. *Handbook of Veterinary Neurology*, 5th ed. St. Louis: Elsevier Saunders, 2010, pp 348-352.



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