In: **Clinical Neurology in Small Animals - Localization, Diagnosis and Treatment**, K.G. Braund (Ed.)
Publisher: International Veterinary Information Service (www.ivis.org), Ithaca, New York, USA.

Traumatic Disorders (6-Feb-2003)

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Central nervous (CNS) system trauma in dogs and cats is commonly encountered in practice. These disorders are among the most devastating of all neurological entities since traumatic injuries often result in death (from initial impact or from euthanasia) or permanent impairment of function. CNS trauma has received more intensive research over the past decade than any other area of human neurology. Considerable advances in knowledge have emanated from this research. We now understand that apart from the primary injury there are important secondary injury processes such as hypoxia, hypotension, mass lesions, increased intracranial pressure, ischemia, free radical production, excitotoxicity, and loss of calcium homeostasis that have a great influence on the clinical outcome. Today, drug research continues to identify therapeutic neuroprotective agents aimed at eliminating or reducing the effects of the cascade of detrimental biochemical and molecular perturbations.

In this chapter, the following topics will be reviewed:

Cranial Trauma
Spinal Trauma

Cranial Trauma

Cranial trauma is a relatively common entity in dogs and cats, usually resulting from a fall or an automobile accident, falls, kicks, bites, or penetrating objects (e.g., bullets, knife wounds, porcupine quills, etc) [1-5,114,116,119]. Head trauma may result in different types of primary injury occurring at the time of impact and include skull fractures, scalp lacerations, cortical contusions and lacerations, and intracranial hemorrhage. The extent of the primary brain injury is affected by the degree of the acceleratory/decelerative and rotational impact forces [6]. In people, such forces may cause tearing of nerve fibers at the moment of impact, called shearing injury or diffuse axonal injury [7], however, Summers and colleagues [8] suggest that this injury has not been confirmed in spontaneous trauma cases in animals. A "coup" contusion occurs in the brain at the site of impact. A "contrecoup" contusion occurs in the area of the brain opposite the point of impact.

As in acute spinal cord trauma, there are important secondary biochemical changes that occur within hours or days after the cranial insult. These events are thought to be associated with progressive hypoxic-ischemic injury of the brain due to multiple factors such as decreased blood flow autoregulation, inadequate cerebral perfusion pressure, altered cerebral metabolism, hemorrhage, increased cytokine and free radical production (e.g., superoxide, hydroxyl, hydrogen peroxide, singlet oxygen, and nitrous oxide), calcium and sodium influx into neurons and glia and endothelial cells along with potassium shift to the extracellular spaces leading to astrocytic swelling and "cytotoxic edema", declining intracellular magnesium levels, elevated levels of excitotoxins (e.g., glutamate and aspartate), and adenosine triphosphatase depletion [9]. The end result of such changes is progressive brain tissue damage and elevated intracranial pressure [6,10,11]. In human trauma patients, mortality is doubled when the deleterious effects of the secondary insults of hypoxia and hypotension are superimposed on severe head injury [9].
Pathological alterations are often heterogeneous and may include intracranial hemorrhage, bone fragments embedded within brain parenchyma, ischemic laminar necrosis of the cerebral cortex, profound hemorrhage into the substance of the brain, especially the midbrain with associated focal or multifocal necrosis of midline structures, and edema. Epidural, subdural, subarachnoid and intraparenchymal hemorrhages may be observed in dogs and cats following head injury [12,116] and bleeding into the inner ear is not infrequent [13]. Subdural hematomas may occur as focal intradural mass lesions or as diffuse lesions over the cerebral cortex, sometimes associated with massive accumulations of blood [12,14]. Subarachnoid hemorrhage is a common consequence of cranial trauma in animals and is usually associated with extensive parenchymal

damage [8]. Hemorrhage into the brain substance (intraparenchymal) from damaged vessels is commonly observed in many forms of cranial injury. This form of bleeding may be short-lived due to vessel spasm and microthrombi formation [15]. Brain hemorrhages may quickly become space-occupying masses (hematomas) that, like brain tumors, compress brain parenchyma, and if unchecked, may lead to widespread brain edema, brain herniation (see below), mid-line shifts, ischemia, brainstem compression and development of deep pontine hemorrhages (Duret hemorrhages) [8,16].
Cerebral edema, either vasogenic from vascular leakage due to openings in the blood-brain barrier (BBB) or cytotoxic, i.e., cell swelling without any loss of the normal impermeability of the BBB (the former is prominent in white matter while the latter affects gray and white matter [17]), can quickly increase intracranial pressure which will reduce cerebral perfusion and further exacerbate cellular hypoxia. Vasogenic edema most commonly occurs with focal injury, brain tumors or abscesses [8,18]. In patients with acute cranial trauma, cytotoxic edema secondary to hypoxia appears to be the dominant form [19]. Recent studies suggest that intracellular swelling of astrocytes is the major form of cytotoxic edema seen in many different kinds of brain injury and one potentially damaging secondary consequence may be an increased release of excitatory amino acids from swollen astrocytes [20]. Since such intracellular swelling is usually not a response to toxins, it has been suggested that the term "cellular edema" is preferable to "cytotoxic edema" [20]. The size of the brain may increase dramatically as a result of cerebral edema, and like hemorrhage (see above) may induce brain herniation. The brain can herniate in several ways in animals [8,21], the four most common being:

a. the cingulate gyrus herniates under the falx cerebri toward the unaffected hemisphere,
b. the occipital or temporal lobe (mainly the parahippocampal gyrus) herniates under the tentorium cerebelli (caudal transtentorial herniation),
c. the rostral cerebellar vermis herniates under the tentorium cerebelli (rostral transtentorial herniation), and
d. the cerebellum (especially the caudal lobe of the cerebellar vermis) herniates through the foramen magnum.

Myocardial degeneration and necrosis following cranial (and spinal) trauma have been reported in dogs [22]. The myocardial lesions are characterized by subendocardial degeneration of muscle fibers accompanied by mineralization, interstitial edema, and mononuclear infiltration. This neural effect on the myocardium is thought to be mediated via the sympathetic nervous system.

A wide variation of clinical signs commensurate with a multifocal syndrome is usually anticipated in animals with head trauma since lesions may be dispersed at multiple levels of the brain. Some animals may be completely normal after a brief period of unconsciousness lasting a few seconds. Other animals can be:

a. comatose - unconscious and unresponsive to repeated noxious stimuli;
b. semicomatose (stuporous) - semiconscious, responsive only to noxious stimuli, demented, with unconscious vocalization;
c. delirious - disoriented, irritable, fearful, capable of responding to the environment but the response may be inappropriate; or
d. depressed - lethargic, despondent but capable of responding to the environment in a normal manner.

These disturbances of consciousness are thought to result from lesions involving the ascending reticular activating system within the brainstem. Limbs of recumbent animals may be rigidly extended. Hyperextension of thoracic limbs and neck (opisthotonus) suggests decerebrate rigidity associated with a midbrain lesion. Pupil size may be normal, pinpoint (suggestive of mild or moderate midbrain compression) or dilated and unresponsive to light (suggestive of severe midbrain compression, e.g., from caudal transtentorial herniation). Normal conjugate eye movements (also called oculocephalic reflex, oculovestibular response, or doll's eye movements) may be depressed or absent when the head is rotated (this is suggestive of severe brainstem pathology). Other signs can include blindness, which may be transient (up to 24 hours) or permanent, various cranial nerve deficits (e.g., unilateral ventrolateral strabismus due to oculomotor damage or unilateral medial strabismus due to abducent nerve damage), vestibular and/or cerebellar signs, and abnormal respiration. Several forms of abnormal respiration may be recognized with cranial trauma [23,24]:

a. Cheyne-Stokes respiration characterized by periods of hyperventilation followed by periods of apnea. This form is often associated with damage to deep cerebral cortical structures, basal ganglia, internal capsule, or diencephalon;
b. central neurogenic hyperventilation characterized by rapid and regular respiration at a rate of about 25 per minute. This respiratory pattern is due to injury to the pons and lower midbrain, but also occur with cerebral hypoxia/acidosis;
c. apneustic respiration, characterized by a cyclic pattern of prolonged inspiration followed by expiration and an apneic

phase. This form is seen with lower brainstem (e.g., medulla oblongata) injury and carries a poor prognosis;

d. central alveolar hypoventilation characterized by shallow, slow but regular, ventilation most often seen with lesions in the medulla oblongata.

Note that respiratory distress due to non-cardiogenic pulmonary edema also has been reported in animals following cranial trauma [25]. The upper airway may also be compromised as a result of indirect injury to the soft tissues of the neck in animals with blunt trauma to the head [119]. In their report of 2 dogs with respiratory distress following horse kicks to the head, the authors suggested that a kick to the head produces rapid acceleration of the skull, tearing soft tissues of the neck (e.g., hyoid apparatus and larynx), and potential cervical spine fracture/subluxation.

Diagnosis typically is based on historical information relating to the accident, clinical evidence of cranial injury, such as abrasions or penetrating wounds, and/or clinical signs. Skull fractures can be demonstrated using radiography. Of the special imaging techniques, computed tomography may be the preferred modality for evaluating soft tissue changes, hemorrhage and bone [6], although magnetic resonance imaging has been used to visualize the compression and displacement of cerebral tissue and to assess the dynamic changes in cerebral tissue water in experimental subdural bleeding [16]. A recent report suggests that brain parenchymal changes assessed by CT were poor compared to changes seen at necropsy [116]. In addition, meningeal enhancement and mass effect were best visualized with MR. Magnetization transfer imaging is a modality capable of examining the non-water components of brain tissue by examining the effects they have on water protons [26]. It may be used qualitatively to increase the visibility of lesions seen during magnetic resonance angiography and following the administration of an intravenous paramagnetic contrast medium. Quantitatively, it can be used to quantify disease progression in trauma. Collection of cerebrospinal fluid is contraindicated because of the risk of brain herniation.

Treatment of animals with cranial trauma can be medical, surgical, or both. The ABCs of trauma resuscitation must be followed (airway, breathing, cardiovascular status) [6] with correction of hypoxia and hypotension. Dewey [6] recommends maintaining the partial pressure of oxygen (PaO_2) at or above 90 mm Hg for dogs and 100 mm Hg for cats, and that of carbon dioxide ($PaCO_2$) between 30 and 35 mm Hg, if arterial blood gas analysis is available. He also recommends use of pulse oximeters, nasal or transtracheal oxygen catheters for conscious patients that are not deteriorating, and intubation and ventilation for patients who are losing or have lost consciousness. Hyperventilation as a means of rapidly decreasing intracranial pressure through vasoconstriction of cerebral vessels is no longer recommended as a first line therapy for intracranial hypertension because of the risk of aggravating any pre-existing cerebral ischemia and further compromising cerebral oxygenation [11,27]. Blood pressure should be evaluated very carefully since systemic hypotension is often seen in head trauma patients and it has been considered a primary predictor of outcome in human patients [28] which will lead to decreased cerebral blood flow (especially if autoregulation of blood flow to the brain is impaired), decreased cerebral perfusion pressure, and tissue hypoxia/ischemia. In human trauma patients, vigilant monitoring of both arterial pressure (MABP) and intracranial pressure (ICP) in order to maintain adequate cerebral perfusion pressure (CPP) is a standard practice (by recording both MABP and ICP, the cerebral perfusion pressure can be determined {CPP = MABP – ICP}) and proven to be beneficial in reducing the incidents of secondary biochemical effects [29,30]. Maintaining CPP at 70 - 80 mm Hg seems to be a critical threshold in humans with cranial trauma. ICP monitoring should also become a standard procedure in animals with head trauma. To date, ICP recordings in animals have been limited [6,31,32]. Normal ICP in dogs and cats lies between 8 and 12 mm Hg [33,34]. Evaluation of ICP using non-invasive transcranial Doppler ultrasonography has recently been reported in dogs [35]. Recently, an implantable solid-state sensor that reliably measures ICP for months has been reported in experimental studies using dogs [36]. The potential clinical application of this sensor and its telemetry includes long-term monitoring of patients with head injury, mass lesions, and hydrocephalus.

Lactated Ringer's solution and 0.9% saline remain the isotonic crystalloid solutions of choice and are administered at a volume of 90 ml/kg/hour in dogs and 60 ml/kg/hour in cats [6,11], to effect. Dewey [6] recommends the colloid hetastarch for restoring normal blood pressure in head injury patients, at a dose of 10 - 20 ml/kg, to effect. It may be given as a rapid bolus in dogs, and in 5 ml/kg increments over 5 to 10 minutes in cats so as to avoid vomiting. Mannitol still remains the primary diuretic to control intracranial pressure [37]. Effective doses range from 0.5 - 1.0 g IV over a 10 - 20 minute period. It may be given as intermittent boluses every 3 - 6 hours [11], but limited to three boluses over a 24-hour period to avoid hypernatremia (See Endogenous Metabolic Disorders) and hyperosmolarity [6]. It has been suggested that concerns about mannitol's "rebound" effect on ICP, its exacerbation of continuing brain hemorrhage, and reverse osmotic shift have been exaggerated [6,11]. Animals should be evaluated approximately every 30 minutes until stable. A response to medical therapy should occur within 4 - 6 hours for a favorable outcome [11]. Corticosteroids and nonglucocorticoid steroid analogs such as the aminosteroid tirilazad mesylate (shown to inhibit lipid peroxidation in experimental studies) have not been shown to be effective in treating head trauma patients and are no longer recommended [6,11,38]. Barbiturates are considered as a rescue therapy in cases of refractory intracranial hypertension, and the use of hypothermia remains to be defined [6,11,39]. New

therapeutic perspectives aimed at controlling biochemical disorders at a cellular level are under active investigation. In one large-scale clinical human trial, pegorgotein, a scavenger of oxygen-derived free radicals, showed no significant reduction in mortality or outcome [40]. Additionally, results of a clinical human trial evaluating CP-101,606 (a postsynaptic antagonist of N-methyl-D-aspartate receptors bearing the NR2B subunit) indicated that CP-101,606 infused for up to 72 hours was well tolerated, penetrated the cerebrospinal fluid (CSF) and brain, and may improve outcome in the brain-injured patient [41].

Surgical management can be considered under the following circumstances:

a. animals with skull fractures and penetrating wounds,
b. comatose animals with miotic pupils whose condition has not improved after 24 to 36 hours of medical therapy,
c. animals whose signs are deteriorating despite aggressive medical treatment,
d. animals with persistent cerebrospinal fluid leakage.

Various surgical techniques are available for cerebral decompression [6,42,43]. In general, simple linear fractures or elevated fractures of the cranial vault do not require fracture management and are usually associated with less severe brain injury than are fractures of the base of the skull which may damage cranial nerves, result in leakage of cerebrospinal fluid, and may provide a portal for entry of infectious agents and subsequent meningitis. Tension pneumocephalus (e.g. ,presence of intraventricular air and a fistula between the craniectomy site and ventricular system) is an uncommon but life-threatening complication of craniectomy that requires urgent diagnosis and treatment [118].
Animals presented with seizures or status epilepticus can be treated with Valium using a dose of 5 to 10 mg IV or IM, and repeated as needed every 30 minutes. General supportive treatment includes monitoring of vital signs, maintaining normal body temperature, prevention of decubital ulcers by placing animals on a padded surface with frequent turning, and bladder emptying [44].

Potential complications include cardiac dysrhythmias, coagulopathies, neurogenic pulmonary edema, central diabetes insipidus, aspiration pneumonia from weakened swallowing reflexes, meningitis from open head wounds/skull fractures, and post-trauma epilepsy, usually within 2 years of head trauma [6,11]. Secondary hypoadrenocorticism associated with head trauma has been reported in a dog [45]. Panhypopituitarism was not confirmed. The hypoadrenocorticism was successfully treated with prednisone.

Prognosis is guarded. Some animals are normal after a brief period of unconsciousness. Others may have a stable condition for several days before showing signs of deterioration. Stuporous or comatose animals with dilated unresponsive pupils have a poor prognosis. A period of coma lasting 48 hours or longer is a grave prognostic sign. Deteriorating clinical signs such as depression progressing to coma, or normal or miotic pupils becoming dilated and unresponsive are ominous and indicative of progressive brain swelling or transtentorial herniation. It has been recently reported that CT findings in dogs and cats with head trauma are unreliable for determining prognosis [116]. A modified Glasgow Coma Scale (MGCS) (see Table 1) (i.e., modified from one used in people) has been proposed as an aid to assess prognosis in animals with cranial trauma [1]. Each category (level of consciousness, motor activity, brainstem reflex) receives a score of 1 to 6. A total score of 3 to 8, without sign of improvement, indicates a grave prognosis; 9 to 14, a poor to guarded prognosis; and 15 to 18, a good prognosis. In a recent study of 38 dogs, the MGCS predicted the probability of survival in the first 48 hours after head trauma in an almost linear fashion, with a 50% probability of survival in a patient with a score of 8 (Fig. 1) [46]. Gender, weight, age and presence of skull fractures did not predict survival. As the authors of this report stated, this study did not take into account any deaths after 48 hours and insufficient data were available for long-term follow-up. In human medicine, the Glasgow Coma Scale has been the most reliable indicator of the severity of injury and deterioration or improvement, and the initial score often influences early treatment [9].

Table 1. Modified Glasgow Coma Scale (MGCS) for Dogs and Cats

Category	Grade
Motor activity	
Normal gait, normal spinal reflexes	6

Category	Grade
Hemiparesis, tetraparesis	5
Recumbent, intermittent extensor rigidity	4
Recumbent, constant extensor rigidity	3
Recumbent, constant extensor rigidity, opisthotonus	2
Recumbent, depressed/absent spinal reflexes and muscle tone	1
Brain stem reflexes	
Normal pupillary reflexes, with normal oculocephalic reflexes	6
Slow pupillary reflexes, with normal to depressed oculocephalic reflexes	5
Bilateral miosis, with normal to depressed oculocephalic reflexes	4
Pinpoint pupils, with depressed to absent oculocephalic reflexes	3
Unilateral, unresponsive mydriasis, with depressed to absent oculocephalic reflexes	2
Bilateral, unresponsive mydriasis, with depressed to absent oculocephalic reflexes	1
Level of consciousness	
Occasional period of alertness and responsive to environment	6
Depression/delirium, capable of responding to environment but response inappropriate	5
Semicomatose, responsive to visual stimuli	4
Semicomatose, responsive to auditory stimuli	3
Semicomatose, responsive only to repeated noxious stimuli	2
Comatose, unresponsive to repeated noxious stimuli	1
Total score (sum of the three categories)	
Grave prognosis	3 - 8
Poor to guarded prognosis	9 - 14
Good prognosis	15 - 18

Courtesy of Dr. A Shores.

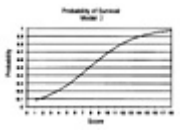

Figure 1. Graph of the probability of survival of a head trauma patient as it relates to the modified Glasgow Coma Scale score assigned to the patient upon admission. (Reprinted with permission from: Platt SR, Radaelli ST, McDonnell JJ. The prognostic value of the modified Glasgow Coma Scale in head trauma in dogs. J Vet Intern Med 2001;15:581-584.) - To view this image in full size go to the IVIS website at www.ivis.org . -

It should be noted that in the acute stage of severe head injury in humans, hyperglycemia (e.g., 200 - 250 mg/dl) and

elevation of serum levels of catecholamines (epinephrine, norepinephrine, and dopamine) are common components of the systemic stress response, significant indicators of severity, and significant predictors of outcome [47-49]. Another metabolic marker associated with poor outcome in children with acute head injuries is hypokalemia [50]. These markers are often seen in patients with lower Glasgow Coma scores. Results of recent clinical studies in dogs and cats with acute spontaneous head trauma indicated that blood glucose concentration was also significantly higher in animals with head trauma than in the controls [51]. While blood glucose concentration was significantly associated with severity of head trauma, it was not associated with outcome. Since hyperglycemia has been shown in both experimental and clinical studies to exacerbate the severity of brain injury during ischemic/hypoxic conditions [52-55] (possibly due to lactic acidosis), efforts are made to prevent it by carefully regulating the glucose and insulin intake in the nutritional management of human patients with head injury [56]. Routine neuroimaging studies are recommended for all animals with head injury, regardless of severity [115]. In this study, dogs with a MGSC score of 15 - 18, and thus graded as having "mild" head trauma and a good prognosis were shown to have myriad abnormalities using computed tomography, including skull fractures, parenchymal damage, hydrocephalus (unilateral and bilateral), hemorrhage, and mass effect.

Spinal Trauma

Spinal trauma of sufficient magnitude to cause vertebral fractures, luxations/subluxations, traumatic disk extrusion, or dural tearing usually results in spinal cord concussion, laceration, compression or distraction [57]. The severity of injury depends on the velocity, degree, and duration of the compressive/distractive force. Spinal injury of this type is one of the more frequent neurological disorders seen in clinical practice. Common causes include automobile accidents, falls, gunshot wounds, and fight injuries. Accidental penetrating injuries of the vertebral canal are extremely uncommon, however, acute onset tetraparesis as a sequela to an oropharyngeal stick injury has been reported in a dog [120]. Fractures and luxations of the spine generally occur at the junction of movable and stable vertebral segments, such as atlanto-occipital, cervicothoracic, thoracolumbar, and lumbosacral areas. In one review of spinal trauma in 41 dogs, the majority of vertebral fractures were in the lumbar region while most vertebral luxations occurred at the thoracolumbar junction [58]. Cervical spinal subluxation at the C5-C6 level has been reported in several dogs as a result of fight injuries, suggesting a possible anatomical predisposition for this type of injury [59]. The axis may be more commonly fractured than other cervical vertebrae. External traumatic injuries of the spinal column have been arbitrarily divided into (a) ventral compartment injuries involving the vertebral body, intervertebral disk, dorsal/ventral longitudinal ligaments, and intertransverse ligaments and (b) dorsal compartment injuries involving the lamina, pedicles, dorsal spinal processes, articular processes, and various ligaments, e.g., supraspinous, interspinous, and interarcuate [60]. Combined compartment injuries are often seen in animals following spinal trauma. A three-compartment model has also been proposed [61]: the dorsal compartment consists of the articular facets, pedicles, laminae, spinous processes, and the ligamentum flavum; the middle compartment contains the dorsal longitudinal ligament, the dorsal annulus, and the dorsal vertebral body; and the ventral compartment consists of the remainder of the vertebral body, the lateral and ventral portions of the annulus, and the ventral longitudinal ligament. When two or three of the compartments are damaged, the spine is considerably unstable. The spine is relatively stable if only one of the compartments is damaged, although in a recent spinal biomechanical study it was suggested that thoracolumbar spinal fractures involving only the vertebral body might significantly destabilize the spine [62].

Apart from the external causes of spinal cord trauma, acute injury can arise from internal factors including intervertebral disk disease [63] and congenital deformities such as atlantoaxial subluxation. Spontaneous spinal fractures attributable to severe osteopenia and hypocalcaemia have been recently reported in cats with nutritional secondary hyperparathyroidism (See Nutritional Disorders) [64], while a dorsally displaced Salter-Harris type I fracture of the cranial portion of the fourth cervical vertebra with the endplate present in the vertebral canal has been described in an adult dog with congenital hypothyroidism [65].

Neurological deficits that follow acute traumatic spinal cord injury result from direct (primary injury) mechanical disruption of neuronal pathways instantly after the traumatic event, as well as from delayed tissue injury (secondary injury) that develops over a period of hours to days after the primary insult. This delayed injury relates to a physiological cascade of events that begins shortly after the injury and includes ischemia, hypoxia, edema and various biochemical events that are harmful to the spinal cord [9]. The degree of ischemia is positively correlated with the severity of the injury and is progressive [66]. In association with reduction of spinal cord blood flow [67,68] there are several metabolic changes that occur almost immediately after spinal cord injury [69-72]. They include electrolyte disturbances such as deceased intracellular free magnesium concentration, increased intracellular calcium level, increased extracellular potassium level, and increased sodium permeability. Additionally, there is loss of high-energy phosphates, lactic acidosis, decline in intracellular pH, reduced oxygen tension, and inflammation and neuronophagia by polymorphonuclear leukocytes. There is now much evidence that the decline in spinal cord blood flow may involve release or activation of endogenous autodestructive factors,

including free radicals, lipid peroxidation, monoamines, free fatty acids, arachidonic acid metabolites (prostaglandins, leukotrienes, and thromboxanes), excitatory neurotransmitter accumulation (e.g., glutamate and aspartate), and endogenous opiate activation [9,73-75]. The collective result of these events is ischemia, edema, membrane destruction, cell death, and eventually, serious if not permanent neurological dysfunction [9].

Pathological findings include petechial hemorrhages progressing to hemorrhagic necrosis over a 24-hour period [76]. These changes are most severe in the gray matter of the spinal cord, with subsequent spread to the white matter. The gray matter vulnerability [77] may be due to several factors [76]:

a. in contrast to the slightly packed fiber tracts of the white matter, the neuropil of the gray matter is easily separated by fluid or blood,
b. because of the inelastic pial membrane, any increase in intramedullary pressure (secondary to hemorrhage/edema) is concentrated centrally, or
c. injured tissues have supranormal metabolic demands, and although gray matter/white matter blood flow ratio is 5:1, the gray matter metabolic needs may exceed the available blood flow.

In contrast, slowly, progressive spinal cord compression, as seen in the Wobbler syndrome or cervical spondylomyelopathy, in dogs with Hansen type 2 disk protrusion, or in dogs and cats with extramedullary masses, tends to be characterized by loss of axons and their myelin sheaths in all funiculi leading to a fibrous astrocytosis and eventually focal spinal cord atrophy [57]. There is no acute hemorrhage or necrosis as seen in acute spinal cord injury, although a reduction of ventral motor neurons in the cervical gray matter may occur from intermittent ischemia. Extensive partial demyelination was found in an experimental model of chronic spinal cord compression in kittens [78].

Animals with acute spinal cord injury should be handled carefully with minimal manipulation so as to avoid further cord injury from any unstable vertebrae. Traumatized animals should be placed in lateral recumbency upon admission and maintained in this posture during clinical/neurological examination and radiographic procedures. Struggling animals can be restrained and immobilized by being firmly taped to a rigid backboard [79]. Animals should be immediately evaluated for airway obstruction, shock, visible hemorrhage or limb fractures. Clinical signs typically are acute in onset, usually nonprogressive, and either stable or improve with time. In rare cases in which clinical signs are progressive, continued bleeding and/or excessive bony movement at the site of injury should be suspected. Clinical syndromes seen with spinal fractures and luxations are cervical, cervicothoracic, thoracolumbar or lumbosacral [80]. Localization of the lesion in such cases usually can be determined with the animal in lateral recumbency.

Radiography usually will demonstrate obvious fractures and luxations of the vertebral column [81]. The degree of luxation has no prognostic value since the luxation that is seen radiographically may have been much worse at the time of the injury. For example, a severe luxation resulting in spinal cord transection may return to normal immediately after the accident. Traumatic disk extrusion sometimes occurs with spinal injury and may be suggested radiographically by the presence of a narrowed disk space. Severe spinal cord contusion can occur in the absence of vertebral or diskal damage. In such cases, myelographic studies may help to delineate an area of spinal cord swelling which may be present up to 36 hours after the injury. Myelography may also delineate any tears in the dura mater [82]. The entire vertebral column should be evaluated radiographically to rule out more than one site of vertebral fracture, luxation or traumatic disk extrusion. Diagnosis of cervical spinal luxation using three-dimensional CT reconstruction has been reported [83]. MRI scans in spinal trauma patients can reveal focal edema within the spinal cord in absence of extraparenchymal compression [84]. Spinal cord and peripheral nerve evoked potentials may be sensitive indicators of severity and location of acute spinal cord compression [77,85,86]. Dogs with neck and back pain caused by acute and chronic spinal cord compression have significantly more oxytocin in their CSF than clinically normal dogs [87]. CSF levels of eicosanoids (prostaglandins, leukotrienes, and thromboxanes) are also increased in dogs during the first 7 days following acute spinal trauma [74]. In this study, there was a good correlation between CSF leukotriene C4 levels and the neurological severity.

Prompt medical treatment is mandatory. Methylprednisolone succinate (MPS) presently remains the drug of choice in people with acute spinal cord injury due to its neuroprotective effects against the physiological cascade associated with the secondary spinal injury events [88-92]. These beneficial effects occur when MPS is given within 8 hours of injury. Clinical studies in humans suggest that spinal cord damage may be exacerbated if MPS treatment is initiated more than 8 hours after injury [93,94]. The recommended dose of MPS in human patients is 30 mg/kg as a bolus administered over 15 minutes followed by an intravenous infusion of 5.4 mg/kg/hour for 24 hours (in patients who receive the bolus within 3 hours of

injury) or for 48 hours (in patients who received the bolus 3 - 8 hours post injury). A similar regimen for MPS (i.e., IV bolus of 30 mg/kg followed by 5.4 mg/kg/hour for 24 hours) [95] has been proposed for spinal cord injury in dogs. Bagley suggests an empirical modification [96]: an initial bolus of 30 mg/kg IV followed by additional doses of 15 mg/kg IV of MPS given at 2 and 6 hours after the initial dose. Surprisingly, results of controlled clinical trials using the human MPS regimen have yet to be reported in dogs and it still remains uncertain whether the use of MPS is actually helpful in dogs with spinal cord injury [73]. In one experimental canine model of spinal trauma, MPS had no clinical efficacy [97]. In cats, the recommended regimen for MPS is 30 mg/kg as an initial intravenous bolus, followed by 15 mg/kg at 2 and 6 hours, and continues with an intravenous infusion of 2.5 mg/kg/hour for 42 hours (see review by Olby [73]). The use of dexamethasone for treating animals with acute spinal cord trauma is no longer recommended due to doubts about its efficacy and because of its detrimental side effects [23,73]. Other potential neuroprotective drugs such as thyrotropin-releasing hormone, the 21-aminosteroids, kappa opioid agonists, and GM1 gangliosides have been reviewed and await clinical trials [43,73,98,99], although the 21-aminosteroids (drugs which are similar in structure to MPS but without the glucocorticoid receptor-mediated effects) have been shown to be beneficial in experimental spinal trauma studies in cats [100-103] but not in dogs [97]. Potential complications of glucocorticosteroids include vomition, hypotension, immunosuppression, gastrointestinal hemorrhage, ulceration, colonic perforation, and pancreatitis [73,79,104,105]. Prophylactic use of intestinal protectants, such as bismuth subsalicylate (i.e., Pepto-Bismol) in conjunction with frequent administration (at least four times daily) of either antacids (such as magnesium or aluminum hydroxide) or H_2 antagonists, such as cimetidine (i.e., Tagamet, 20 mg/kg, PO, tid) also may reduce the prevalence of gastrointestinal hemorrhage. Corticosteroids should be stopped immediately, when gastrointestinal complications are noted.

In patients with fractures and luxations, medical management is usually combined with prompt surgical intervention such as decompression of the spinal cord, vertebral reduction, and internal stabilization (e.g., use of Steinman pins and polymethylmethacrylate or coated polypropylene) [42,96,106,107,117]. The choice of stabilization technique is contingent on the location in the spinal column, size of the patient, and the surgeon's experience [106,117]. Acute surgical intervention is also required in cases with neurological deterioration associated with spinal cord compression from bone and disk fragments, hematoma, or unreduced subluxation [9]. Experimental studies indicate that surgical intervention should occur as early as possible, e.g., within an hour following compressive spinal cord injury [108]. In this report, there was no neurological recovery in dogs when compression lasted six hours or more. Durotomy or myelotomy have been recommended in animals with spinal cord swelling but without extradural compression [11]. Animals with luxations and mild paresis may require only internal stabilization without decompression. All animals undergoing surgery also should be confined for 2 to 3 weeks after surgery. Paretic animals without evidence of vertebral column injury can be managed with strict cage confinement for 4 to 6 weeks [79]. Additionally, external support bandages or casts applied for 4 to 6 weeks may be used as forms of non-surgical treatment [61,79,96,107]. Decompressive surgery and removal of a foreign body 3 days after injury was successful in a dog with acute spinal cord compression secondary to an oropharyngeal stick injury [120].

Recent studies indicate that an applied electric field (oscillating electric field stimulation, approximately 500 to 600 µV/mm) in which the polarity is reversed every 15 minutes can improve the outcome from naturally occurring severe, acute spinal cord injury in dogs [109]. The oscillating extracellular voltage gradient reduces the density and influences the orientation of astrocytes in injured spinal cord, which form a major component of the scar that forms in response to injury [110].

Prognosis of animals with acute spinal trauma is always guarded and is influenced by several factors, including the degree and location of spinal cord damage and stability of the fixation technique [104,106,117]. Spinal cord dysfunction reflects varying degrees of damage to the gray matter, white matter, or both. With cranial cervical and thoracolumbar cord injuries, clinical signs primarily reflect damage to ascending and descending tracts of the white matter. More heavily myelinated fibers show earliest dysfunction; thus, signs of progressive neurological deterioration in order of presentation are [2]:

a. loss of proprioception,
b. motor dysfunction such as paresis and paralysis, and
c. sensory disturbances including hypesthesia, hyperesthesia, and anesthesia.

Neurological recovery occurs in reverse order; however, proprioceptive loss may be permanent. In cranial cervical and thoracolumbar spinal cord regions, severe gray matter lesions have little clinical significance, since no major muscle groups or vital organs will be deprived of innervation. In contrast, gray matter lesions in caudal lumbar segments involve the lumbosacral intumescence, which provides innervation to the pelvic limb muscles and bladder. An extensive gray matter lesion in this region carries a very poor prognosis. Similarly, severe gray matter lesions in caudal cervical segments may result in death from respiratory failure due to damage to neurons giving rise to the phrenic nerves. Also, gray matter damage in cervicothoracic cord segments (the cervical intumescence) will carry a guarded prognosis because of denervation of thoracic limb muscles. In animals with disk disease, the degree and rate of clinical recovery are largely based upon the rate of disk extrusion. Gradual cord compression over several hours has a much more favorable prognosis than compression

occurring within seconds or minutes. The prognosis in many cervical injuries may be better than that in other spinal regions since the ratio of canal to cord diameter is greatest in the cervical region, and therefore a greater vertebral displacement can be tolerated [2]. Animals with gunshot wounds involving the spine have a very guarded prognosis [5]. One prognostic indicator of spinal cord injury is the vocal response to a noxious stimulus that is easily tested by applying digital pressure to the nail bed. Absence of deep pain perception (nociception) is an indication that a severe spinal cord injury has occurred. In general, animals that are paretic or paralyzed, but have normal sensation to a painful stimulus, have a favorable prognosis following medical and/or surgical treatment. Clinical signs can be expected to improve within 2 to 3 weeks. Animals that are paralyzed, with loss of bladder control and reduced sensation to a noxious stimulus, have a guarded to favorable prognosis following surgical decompression. Any clinical improvement should be seen within 3 to 6 weeks. Paralyzed animals with loss of bladder control and loss of sensation to painful stimuli for greater than 24 - 48 hours have a guarded to poor prognosis [11,96,111]. Prognosis is considered hopeless if sensation to a noxious stimulus is absent in an animal with 100% or greater displacement of the vertebral canal [96]. A functional scoring system for evaluating pelvic limb gait in dogs recovering from acute spinal cord injuries associated with disk protrusions has been developed [112]. This methodology examines the rate and level of functional recovery and may be useful in clinical drug trials aimed at improving the outcome of acute spinal cord injuries.

Nursing care of paraplegic or tetraplegic animals is important to prevent decubital ulcers, urinary tract infections, and disuse muscle atrophy [104]. Many successful surgical procedures have been jeopardized by inadequate supportive care. Adequate alimentation during convalescence is extremely important in short- and long-term therapy. Oral or intravenous fluid therapy should be adequate to correct or prevent dehydration. Paralyzed animals need to be maintained in a sanitary environment, with frequent removal of soiled bedding. Multiple sponge baths are given daily when the animal is frequently soiled with urine or feces. Petroleum jelly applied to the preputial or vulval areas can be used for waterproofing and averting urine scalds. Decubital ulcers can be prevented or delayed by use of waterbeds, air mattresses, or foam rubber pads. Alternating right and left lateral recumbency, at least four times daily, is recommended for paralyzed animals. Post-operative pain can be reduced using opiate analgesics, e.g., morphine, 0.3 mg/kg IM, every 4 hours for the first 12 hours in conjunction with a dermal patch (e.g., Duragesic, Fentanyl Transdermal system) [96].

Active physiotherapy (see rehabilitation in chapter IV) to delay muscle atrophy from disuse is an integral part of the nursing care of paralyzed animals. Through the use of slings, animals should be encouraged to support themselves. Such "standing" exercises can be performed for 10 minutes, 5 or 6 times daily. Additional exercises include supervised swimming for 15 to 20 minutes twice daily, vigorous muscle massaging, and passive manipulation of limbs. Muscle stimulators, used in human physical therapy, actively stimulate muscles to effect active flexion and extension of the limbs. Animals need to be encouraged to stand and walk, initially with assistance, and then unassisted. After an animal has been in the hospital for 1 to 2 weeks, owners should continue physiotherapy at home. Some animals may respond faster in familiar surroundings than in the hospital. Bladder management is of paramount importance in the paralyzed patient. Since most animals lose control of micturition, urine evacuation from the bladder is incomplete and this predisposes the animal to urinary tract infection and bladder overdistension, with subsequent bladder atony. Catheterization, the most effective method for emptying the bladder, should be performed three times a day using soft, sterilized rubber catheters. Indwelling catheters frequently lead to urinary tract infections. However, in female patients, human urinary drainage systems consisting of a Foley catheter and sterile closed system drainage bags are an excellent method of maintaining bladder decompression. It is good practice to instill prophylactic antibiotic solutions, such as nitrofurazone or neomycin, into the bladder following removal of urine. In the event that urinary tract infection occurs, urine should be cultured, and appropriate systemic or intraurinary antibiotic therapy initiated. Manual bladder expression may be possible in some animals, especially females (note: in animals with a lumbosacral syndrome, the bladder is flaccid and easy to evacuate manually). Care should be taken since overzealous manipulation can traumatize the bladder. Contractile function in animals with an upper motor neuron bladder (i.e., with lesions between T3 and L3) may return in several weeks as a sacral reflex ("reflex bladder") but voiding tends to be involuntary, incomplete, and poorly coordinated (detrusor-urethral dyssynergia) [113]. In dogs with resistance to evacuation of the bladder by catheterization or manual expression, the alpha blocker phenoxybenzamine (0.25 - 0.5 mg/kg, PO, sid or bid, in dogs; or 1.25 - 7.5 mg/cat, PO, sid or bid) or a skeletal muscle relaxant, such as diazepam (2 - 10 mg/dog PO, tid; or 1.0 - 2.5 mg/cat, PO, tid or 0.5 mg/kg, IV) may reduce urethral tone [113].

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