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Traumatic head injuries in cats • How I approach... Canine ocular emergencies • Emergency visits to primary care veterinary hospitals • Emergency management of open fractures • Penetrating injuries in dogs • Gastric dilatation and volvulus • Thoracic trauma • Cut-out and keep guide... Pain assessment in the dog: the Glasgow Pain Scale



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Of all the quotes pertinent to those who practice medicine, the Latin phrase *primum non nocere* (“first, do no harm”) is well worthy of note, even if it seems to state the obvious. And it is perhaps especially relevant when it comes to emergency medicine; in the heat of the moment, when we are in the midst of a life-or-death situation, even the most level-headed of clinicians

can succumb to stress, and could potentially do something that will make matters worse, rather than better, for their patient. Logical thinking and common sense are vital partners to the expert knowledge required when we consider the ideal elements necessary for an effective emergency specialist.

However, although we talk of emergency medicine as a specialty, it is perhaps fairer to say that it is several specialties rolled into one. The front-line clinician must be competent as a radiologist, surgeon, pharmacist, ophthalmologist, cardiologist... The list goes on, because the emergency physician, along with the nurse/technician support team, must command a broad field of knowledge and possess many different abilities to allow appropriate treatment of patients on presentation. They must be able to deliver trauma resuscitation, offer advanced cardiorespiratory support and maintain physiological parameters, as well as having the abilities to cope with wounds, stabilize fractures, manage neurologic crises, and relieve pain, discomfort and distress, amongst other problems. Not an easy task, by any means.

However, a counter to the phrase, *primum non nocere*, might be said to be *palma non, sine pulvere* – “there is no reward without effort”. As clinicians we will always try to do the best for every animal we treat, with the aim of *restitutio ad integrum* (“restoration in full”), and if our efforts are successful, the reward – the satisfaction of knowing we have saved the life of our patient – is immense in itself. To this end we believe that this issue of *Veterinary Focus* will offer you *scire quod sciendum* – “knowledge which is worth having”.

Ewan McNeill – Editor-in-chief

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Traumatic head injuries in cats



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■ Introduction

The ability to recognize clinical signs consistent with a declining neurologic status is critical in the management of cats following head trauma (1-4). Trauma significant enough to cause brain injury will have systemic effects, which may be life-threatening. Additionally, systemic injuries and shock will cause continued decline in the head trauma patient, and a complete systemic evaluation and stabilization is therefore required in addition to a thorough neurologic assessment, as summarized in **Table 1**.

KEY POINTS

- A thorough systemic evaluation of any cat which has been involved in a traumatic event is essential prior to focusing on the nervous system.
- The neurologic exam of the head-injured cat can be condensed into assessment of mentation, limb function and pupillary light reflexes.
- Imaging the brain of the head trauma cat can help identify causes of the neurologic dysfunction, but rarely leads to a surgical treatment.
- Fluid therapy is essential for all cats which have suffered a head injury and should focus on restoring systemic blood pressure.
- Flow-by oxygen therapy is recommended as a first-line treatment for cats with traumatic brain injury.

■ Systemic assessment

Initial assessment should involve evaluation of the patient's respiratory and cardiovascular system. A patient airway must be established and maintained, if necessary via endotracheal intubation. Breathing patterns may be affected by thoracic trauma but can also be secondary to brain injury. Auscultation of the thorax may detect pulmonary pathology or cardiac arrhythmias. The cardiovascular system should be evaluated by monitoring heart rate, blood pressure, and electrocardiography. An electrocardiogram may demonstrate cardiac arrhythmias secondary to traumatic myocarditis, systemic shock, or brain injury. Arterial blood analysis and lactate concentrations may provide additional information regarding systemic perfusion and respiratory function (1-4).

Once the patient is stable, radiographs of the chest and abdomen are recommended to evaluate for pulmonary contusions, pneumothorax, and abdominal injuries. Pulmonary contusions are common following trauma and may not be at their most severe until 24 hours after injury. Trauma can also result in injury to abdominal organs; the abdomen should be evaluated via radiography and ultrasonography for the presence of free fluid, such as blood or urine, which may require additional therapy. Radiographs of the cervical vertebrae should also be considered, as head trauma can often be accompanied by fractures and luxations of these bones.

■ Neurologic assessment

Neurologic assessment should be undertaken on all head trauma patients (1-5). Assessment of neurologic status should initially be performed every 30-60 minutes; frequent assessment allows for monitoring efficacy of treatment and early recognition of a deteriorating status.

Table 1. Monitoring parameters for the cat following head trauma.

Parameter	Suggested goal	Suggested treatment
Neurologic examination	Modified Glasgow Coma Scale (MGCS) > 15	Ensure head elevation (30°) Ensure all points below are addressed Consider mannitol (see below) Consider surgery (see text)
Blood pressure	MAP 80-120 mmHg	Adjust fluid therapy Pressor support (dopamine 2-10 µg/kg/min)
Blood gases	PaO ₂ ≥ 90 mmHg PaCO ₂ < 35-40 mmHg	Oxygen supplementation Consider active ventilation
Pulse oximetry	SPO ₂ ≥ 95%	Oxygen supplementation Consider active ventilation
Heart rate & rhythm	Avoid tachy- and bradycardias Avoid arrhythmias	Adjust fluid therapy Treat for pain Address ICP Treat arrhythmias specifically
Central venous pressure	5-12 cm H ₂ O	Adjust fluid therapy
Respiratory rate & rhythm	10-25/min	Ventilate if necessary
Body temperature	37-38.5 °C (98.6-101.3 °F)	Passive warming or cooling
Electrolytes	See individual laboratory normal values	Adjust fluid therapy
Blood glucose	4-6 mmol/L (67-168 mg/dL)	Adjust fluid therapy Consider dextrose administration
Intracranial pressure	5-12 mmHg	As for MGCS abnormalities (see action plan in Figure 4)

A scoring system, the Modified Glasgow Coma Scale (MGCS), has been developed in veterinary patients to provide an objective assessment and allow for rational diagnostic and treatment decisions. The scale evaluates three categories — motor activity, brainstem reflexes, and level of consciousness — enabling initial and serial monitoring in patients following injury (**Table 2**). Each category is evaluated using objective standards and scored between 1 and 6, with lower scores assigned to more severe clinical signs. The score from each category is added together to determine a patient's coma score, ranging from 3 to 18, and may be used to guide treatment decisions and prognosis (5).

Assessment of limb function

The first category describes a patient's motor activity, limb tone, and posture. Voluntary motor activity is characterized as normal, paretic, or recumbent. Patients typically maintain some degree of voluntary motor activity, even in altered states of consciousness, unless comatose. Abnormal motor function usually reflects either brainstem injury or spinal cord injury; the latter may complicate the assessment of head trauma (5).

An animal's posture after head trauma can also provide information about the location and degree of brain injury. Decerebrate rigidity (**Figure 1**) can occur following cerebral trauma and suggests severe brain injury; this posture carries a poor prognosis, as it reflects loss of communication between the cerebrum and the brain

Figure 1. Extensor rigidity in a cat following head trauma.



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Table 2. Modified Glasgow Coma Scale.

Motor activity	Score	Brainstem reflexes	Score	Level of consciousness	Score
Normal gait, normal spinal reflexes	6	Normal pupillary light reflexes and oculocephalic reflexes	6	Occasional periods of alertness and responsive to environment	6
Hemiparesis, tetraparesis or decerebrate activity	5	Slow pupillary light reflexes and normal to reduced oculocephalic reflexes	5	Depression or delirium, capable of responding but response may be inappropriate	5
Recumbent, intermittent extensor rigidity	4	Bilateral unresponsive miosis with normal to reduced oculocephalic reflexes	4	Semicomatose, responsive to visual stimuli	4
Recumbent, constant extensor rigidity	3	Pinpoint pupils with reduced to absent oculocephalic reflexes	3	Semicomatose, responsive to auditory stimuli	3
Recumbent, constant extensor rigidity with opisthotonos	2	Unilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	2	Semicomatose, responsive only to repeated noxious stimuli	2
Recumbent, hypotonia of muscles, depressed or absent spinal reflexes	1	Bilateral, unresponsive mydriasis with reduced to absent oculocephalic reflexes	1	Comatose, unresponsive to repeated noxious stimuli	1

stem. Animals with decerebrate rigidity have opisthotonos with hyperextension of all four limbs and are stuporous or comatose, with abnormal pupillary light reactions. This should be differentiated from decerebellate rigidity, which suggests acute cerebellar damage and may cause either flexion or extension of the pelvic limbs; however, consciousness may be normal.

Assessment of brainstem reflexes

Pupil size, the pupillary light reflex, and the oculocephalic reflex should be immediately evaluated in all head trauma patients. Pupil size, symmetry, and reactivity can provide valuable information about severity of brain injury and prognosis, and these parameters should be frequently re-assessed as they can signal a deteriorating neurologic status. Response of the pupils to a bright light indicates sufficient function of the retina, optic nerves, optic chiasm, and rostral brainstem. Bilateral mydriasis that is unresponsive to light can indicate permanent midbrain damage or brain herniation and suggests a poor prognosis (**Figure 2**). Progression from miosis to mydriasis indicates a deteriorating neurologic status and is an indication for immediate, aggressive therapy. Unilateral changes in pupil size may be seen early in deterioration. Paralysis of cranial nerve (CN) III can lead to mydriasis, loss of direct pupillary light reflex, ptosis, and ventrolateral strabismus. The CN III nucleus is located in the midbrain; therefore, damage to this nucleus can be indicative of midbrain injury or compression secondary to transtentorial herniation (5).

Assessment of consciousness

A patient’s level of consciousness provides information regarding function of the cerebral cortex and the ascending reticular activating system of the brainstem. Consciousness can be described as normal, depressed or obtunded, stuporous, or comatose. An animal in a stupor is partially or completely unconscious, but will respond to noxious stimuli. A patient in a coma is unconscious

Figure 2. Bilateral mydriasis in a cat. This can suggest severe brain pathology after head trauma but in this case it was due to bilateral retinal damage following the trauma, suspected because the cat had a normal level of consciousness.



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and cannot be roused with noxious stimuli. Coma typically indicates severe cerebral injury or brainstem damage, which carries a guarded prognosis.

■ Confirmation of injury and diagnosis

A diagnosis of traumatic brain injury is based primarily on a compatible history and clinical signs of intracranial neurologic dysfunction. However, additional tests can be used to confirm location and extent of injury. It is important to emphasize that advanced imaging of the brain — computed tomography (CT) and magnetic resonance imaging (MRI) — should be reserved for patients that do not respond to initial treatment or for patients who deteriorate despite aggressive therapy. Both imaging modalities require anesthesia, which can destabilize the head trauma patient, unless the patient is comatose on presentation.

Skull radiographs

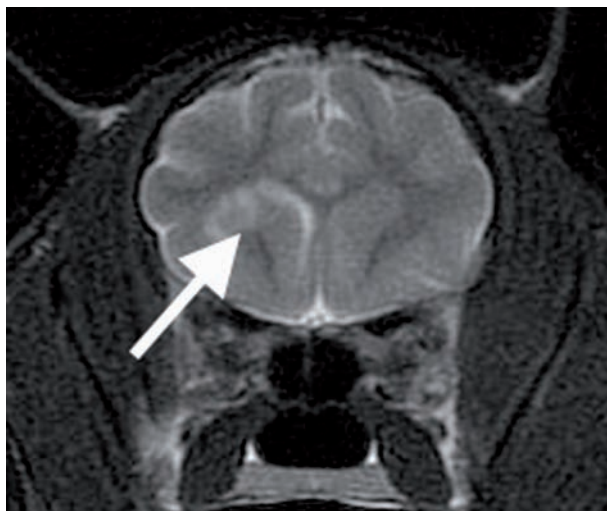
Skull radiography may reveal calvarial fractures, but provides no information regarding the brain parenchyma. Radiographs can be difficult to interpret due to the irregularity of the skull bones and anesthesia is required for accurate positioning, which may be contraindicated in the acutely injured patient. However, radiography should not be limited to the skull following head trauma; images of the vertebral column, thorax, and abdomen are indicated to evaluate for evidence of other injuries.

Computed tomography

CT allows superior evaluation of bony structures and is preferred over conventional radiography, especially considering the 3D reconstruction capabilities (6). Additionally, CT can be used to diagnose intracranial hemorrhage, alterations in ventricular size or shape, midline shift and edema. It does not provide good soft tissue detail of the brain parenchyma, but is frequently the preferred modality for evaluating humans with head trauma requiring surgical intervention because of the speed of image acquisition.

Magnetic resonance imaging

MRI allows superior soft tissue detail and is preferred for evaluation of the brain, especially the caudal fossa which does not image well with CT. MRI can detect subtle parenchymal changes which can be missed on CT and may provide information about prognosis. Hematomas or hemorrhage, parenchymal contusions, and edema are readily apparent on MRI images (**Figure 3**). A recent study correlated MRI findings with prognosis in veterinary



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Figure 3. A focal brain parenchymal lesion (arrowed) was identified post-trauma on this transverse T2-weighted MRI in a cat.

head trauma patients and its use may therefore be of additional benefit (7). Specifically, identification of mass effect and ventricular compression by parenchymal damage can be poor prognostic indicators and in such cases, decompressive surgery should be considered.

■ Treatment

Treatment of head trauma is proposed in a progressive tiered system based on the severity of injury and the success of the initial therapy (**Figure 4**). Tier 1 treatments are administered to all patients; Tier 2 treatments are administered to all patients with a MGCS of < 8 and failure of Tier 1 treatment; Tier 3 treatments are administered to all patients with a MGCS of < 8 and failure of Tier 2 treatments.

Tier 1 Therapy

• Fluid Therapy

The goal of fluid therapy for the head trauma patient is to restore a normovolemic state; it is deleterious to dehydrate an animal in an attempt to reduce cerebral edema. Aggressive fluid therapy and systemic monitoring is required to ensure normovolemia and maintain adequate central perfusion pressure (1-4,8).

Crystalloid, hypertonic, and colloid fluids should be given concurrently to help restore and maintain blood volume following trauma. Crystalloids are usually given initially for the treatment of systemic shock. The shock dose of balanced electrolyte solutions is 60 mL/kg (1-4,8) and it is recommended that the calculated dose is given in fractions, initially administering 25-33% of the total volume

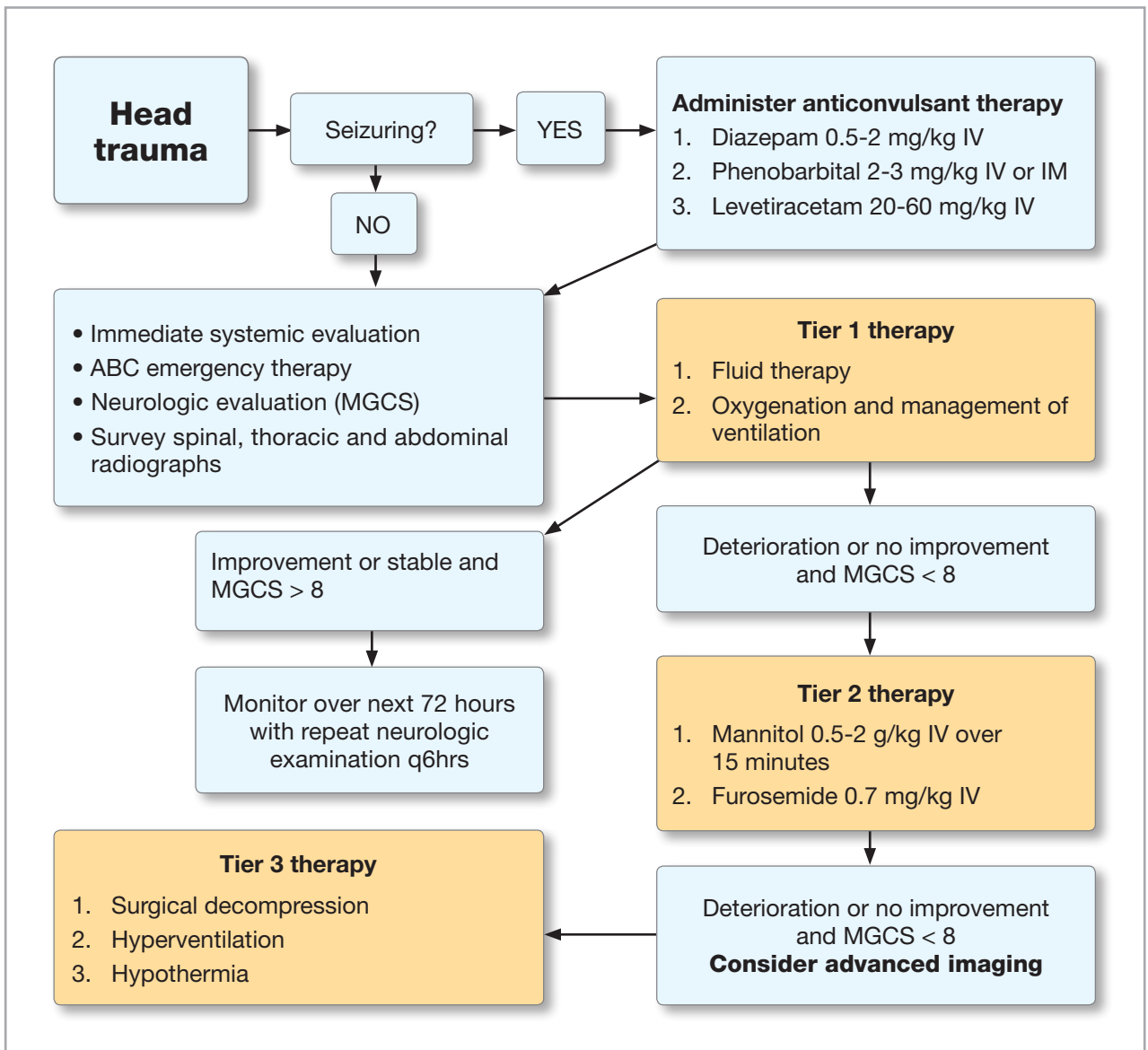


Figure 4. Treatment of head trauma should be via a progressive tiered system based on the severity of injury and the success of the initial therapy, as shown in this algorithm.

with frequent reassessment of the patient for normalization of blood pressure, mentation and central venous pressure (if monitored), with additional fractions given as needed.

Hypertonic and colloid fluid therapy can rapidly restore blood volume using low volume fluid resuscitation; additionally, colloids remain in the vasculature longer than crystalloid fluids. These fluids should be used with caution, as without concurrent administration of crystalloid solutions dehydration can develop. Other benefits of hypertonic fluids include the ability to improve cardiac

output, restore normovolemia, and reduce inflammation after trauma. Hypertonic saline may be preferred in hypovolemic, hypotensive patients with increased intracranial pressure (ICP) as it improves cerebral perfusion pressure and blood flow by rapidly restoring intravascular blood volume. Additionally, the high sodium content draws fluid from the interstitial and intracellular spaces, reducing intracranial pressure. Hypertonic saline is contraindicated with systemic dehydration and hypernatremia. Hypertonic saline only remains within the vasculature for about one hour; therefore, it should be followed by colloids to maximize its effects. A dose of

2-4 mL/kg of 7.5% NaCl should be given over 5-10 minutes in cats (1-3).

Colloids (*i.e.*, Hetastarch, Dextran-70) allow for low volume fluid resuscitation especially if total protein concentrations are below 50 g/L or 5 g/dL. Colloids also draw fluid from the interstitial and intracellular spaces, but have the added benefit of staying within the intravascular space longer than crystalloids. Hetastarch is typically given at 2-4 mL/kg over 5-10 minutes, with frequent patient re-evaluation; a total dose of 20 mL/kg/day may be given. In addition to volume resuscitation, oxygen carrying capacity should be considered, especially if the PCV (packed cell volume) is < 30%.

Head trauma patients should be positioned to maximize arterial circulation to the brain and improve venous drainage; this is best achieved by elevating the animal's head at an angle of 30°. It is important to ensure the jugular veins are not occluded and that no restrictive collars are placed around the neck, which will elevate intracranial pressure.

• **Oxygen therapy and management of ventilation**

Oxygen supplementation is recommended in all patients following head trauma. Control of the arterial partial pressures of oxygen (PaO₂) and carbon dioxide (PaCO₂) is mandatory and will affect both cerebral hemodynamics and ICP. Permissive hypercapnea should be avoided because of its cerebral vasodilatory effect that increases ICP. Hypocapnea can produce cerebral vasoconstriction through serum and CSF alkalosis. Reduction in cerebral blood flow (CBF) and ICP is almost immediate, although maximum ICP reduction may take up to 30 minutes after PaCO₂ has been changed (1-4,8).

The goal of oxygen therapy and management of ventilation is to maintain PaO₂ at least equal to 90 mmHg and the PaCO₂ below 35-40 mmHg. If the patient is able to ventilate spontaneously and effectively, supplemental oxygen should be delivered via "flow-by"; confinement within an oxygen cage prevents frequent monitoring. Face masks and nasal catheters should be avoided if possible, as they can cause anxiety which may contribute to ICP elevation (**Figure 5**).

Cats with severe head injury require mechanical ventilation to maintain arterial blood gas concentrations at their optimal levels. The absolute indications for mechanical ventilation include loss of consciousness,

rising PaCO₂ above 50 mmHg and falling peripheral capillary oxygen saturation (SPO₂) despite appropriate treatment (1-4,8).

Tier 2 therapy

• **Diuretics**

Increased ICP can be aggressively addressed with the administration of osmotic diuretics such as mannitol, but they should not be given to any patient without being certain that the animal has been volume-resuscitated. If not, their use can precipitate acute renal failure, hence they are reserved as tier 2 therapies. After administration, mannitol expands the plasma volume and reduces blood viscosity, which improves cerebral blood flow and delivery of oxygen to the brain, and reduces ICP by decreasing edema. Vasoconstriction occurs as a sequel to the increased PaO₂, which in turn helps to decrease ICP. Additionally, the osmotic effect of mannitol reduces extracellular fluid volume within the brain (1-4,8) and it assists in scavenging free radicals, which contribute to secondary injury processes (9).

Mannitol should be given as a bolus (0.5-2 g/kg) over 15 minutes to optimize the plasma expanding effect; continuous infusions increase the permeability of the blood brain barrier, exacerbating edema. Low doses of mannitol are as effective at decreasing ICP as higher doses, but may not last as long. Mannitol reduces brain edema about 15-30 minutes after administration and has an effect for approximately two to five hours. Repeated dosing can cause diuresis leading to reduced plasma volume, increased osmolarity, intracellular dehydration, hypotension, and ischemia, so adequate isotonic crystalloid and colloid therapy is critical to maintain hydration.

Figure 5. Oxygen delivery via face mask should be avoided after head trauma as intracranial pressure may increase due to the concurrent stress induced by the mask.



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Administration of furosemide (0.7 mg/kg) prior to giving mannitol has a synergistic effect in lowering ICP. The use of mannitol should be reserved for critical patients (MGCS of < 8), a deteriorating patient, or a patient failing to respond to other treatment; there is currently no evidence to support the notion that mannitol is contraindicated if intracranial hemorrhage is present.

• **Seizure therapy**

Seizure activity may occur immediately following trauma or may be delayed in onset, and should be aggressively treated to prevent worsening of the secondary effects in the brain parenchyma due to associated brain hypoxia and subsequent development of edema. The need for prophylactic anti-seizure therapy after severe brain trauma remains controversial. Human patients treated in the first seven days after head trauma with anticonvulsants have a significantly lower risk of post-traumatic seizures within this time period than if not treated, but beyond this period there appears to be no benefit to prophylactic treatment.

Diazepam (0.5-2 mg/kg IV) can be given to treat seizures. Phenobarbital (2-3 mg/kg IV or IM) may be given in addition, and continued parenterally following the loading dose (18-24 mg/kg over a 24-48 hour period) if necessary. Recently, the use of levetiracetam (20-60 mg/kg IV) has been described for emergency seizure treatment, as it may be effective for up to 8 hours without causing excessive sedation and does not require hepatic metabolism. Refractory seizures at the time of head trauma may require additional therapy such as a continuous infusion of diazepam (0.5-1.0 mg/kg/hr) or propofol (4-8 mg/kg bolus to effect followed by 1-5 mg/kg/hr constant rate infusion). Chronic maintenance seizure therapy should be continued for at least 12 months after the last noted seizure following head trauma.

Tier 3 therapy

Failure of fluid therapy, oxygenation and ventilation strategies, and osmotic diuretics to stabilize the patient and/or significantly improve the neurologic status warrants radical therapy and such cases should be considered for advanced imaging such as MRI (6,7). The treatments discussed below have not been evaluated in veterinary medicine in terms of their efficacy and remain controversial or unproven in human head trauma.

• **Hyperventilation**

Hyperventilation has been suggested as a method of quickly lowering ICP. Hypercapnea causes vasodilation

and subsequent increases in intracranial pressure; hypoventilation should therefore be avoided. Mechanical or manual ventilation may be used to lower PaCO₂ to 35-40 mmHg in order to reduce ICP in deteriorating patients unresponsive to other treatment and with no surgical lesions. However, the prolonged use of hyperventilation should be avoided, as a reduction in cerebral PaCO₂ below 30-35 mmHg causes vasoconstriction which ultimately leads to decreased cerebral blood flow and ischemia (1-4).

• **Hypothermia**

Hypothermia is currently an experimental treatment which has not been validated in veterinary medicine and remains controversial in human medicine. Following trauma, the cerebral metabolic rate may increase, leading to exacerbating secondary effects. Hypothermia can be achieved by cooling a patient to a rectal temperature of 32-35°C, which reduces cerebral metabolic rate and oxygen consumption, leading to decreased CBF and ICP. However, reduction of core body temperature carries risks and may lead to the development of cardiac arrhythmias, coagulopathies, electrolyte disturbances, hypovolemia, and insulin resistance. Coma may also be induced using barbiturates, but this prevents neurologic evaluation and requires mechanical ventilation.

• **Surgery**

Surgical intervention is reserved for patients that do not improve, or deteriorate despite aggressive medical therapy. Advanced imaging (CT or MRI) is necessary for surgical planning and is also reserved for similar patients. Surgery may be indicated to remove hematomas, relieve intracranial pressure, or address skull fractures. Ventricular obliteration and mass effect, which can be identified on advanced imaging, should be considered strong indicators for surgical intervention in any animal which does not improve on medical therapy.

■ **Supportive therapy**

Finally, it is important not to neglect general supportive care for all head trauma cases. Urinary catheters should be placed to provide proper bladder management in recumbent patients and to monitor urinary output. Adequate urine output is between 1-2 mL/kg/hr, but should match the volume of fluid given to the patient. Reduced urine output may indicate continued dehydration, hypovolemia, or reduced renal function. Increased urine output may be seen secondary to osmotic diuretic therapy as well as central diabetes insipidus, which can occur as a sequel to intracranial trauma.

Adequate nutrition is critical to the recovery of patients following brain injury; however, hyperglycemia should be avoided as it increases cerebral metabolic rate and promotes anaerobic metabolism leading to cerebral acidosis. Initially, nutrition may be supplemented through a naso-esophageal feeding tube, but in patients with elevated ICP placement may be contraindicated, as this can stimulate sneezing, which causes transient increases in ICP. In patients with proper esophageal function, esophagostomy tubes enable medium to long-term management

of feeding, whilst gastrotomy tubes can offer nutritional support in patients with poor esophageal function, allowing long-term nutritional support.

Recumbent patients require proper bedding and monitoring to prevent the development of decubital ulcers; bedding should be well padded and evaluated frequently to maintain a clean and dry surface. Patients require alternation of recumbency every 4-6 hours and frequent evaluation of pressure points for ulcers.

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HOW I APPROACH...

Canine ocular emergencies



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Dr. Giuliano is a Professor of Ophthalmology at the University of Missouri and Section Head of their comparative ophthalmology service. She has authored over 70 articles and text book chapters and has lectured extensively both in the academic setting and at national and international meetings. She is the recipient of numerous teaching awards, including two Golden Aesculapius Teaching Awards and the Gold Chalk Award. In 2011 she was elected to the ACVO Board of Regents where she served for 3 years; she currently holds the office of ACVO Vice President.

■ Overview

Ophthalmic emergencies are commonly seen by the small animal practitioner and can be said to include any ophthalmic condition that has rapidly developed or is the result of trauma to the eye and/or periocular structures. With proper treatment, most emergencies can be stabilized until consultation with, or referral to, a veterinary ophthalmologist is possible. Most ocular emergencies present due to significant ocular discomfort, loss of vision or compromised globe integrity, and can be classified as being either traumatic or non-traumatic in origin. The first category includes problems such as globe prolapse, conjunctival/corneal foreign body, corneal chemical burn, corneal wound and/or perforation, iris

prolapse, and lens rupture with associated phacoclastic uveitis, whilst the second category includes conditions such as orbital cellulitis/abscesses, acute keratoconjunctivitis sicca (KCS), corneal ulcers, acute congestive glaucoma, uveitis, anterior lens luxation, retinal detachment, SARD (sudden acquired retinal degeneration), optic neuritis, and endophthalmitis. Prompt intervention and proper treatment are essential to preserve vision and restore ocular comfort. This article reviews the initial approach to canine ocular emergencies and discusses some of the more common problems, including conditions affecting the orbit and globe, adnexa, conjunctiva, and cornea. Uveitis, glaucoma, and lenticular diseases are other significant ocular diseases that may present as an emergency but are not discussed in detail in this brief review.

KEY POINTS

- Ophthalmic emergencies are commonly seen in small animal practice, and with proper treatment most cases can be stabilized until consultation with, or referral to, a veterinary ophthalmologist is possible.
- Minimal equipment is necessary to perform a complete ophthalmic exam and the clinician should strive to acquire the “minimum ophthalmic database” to best diagnose and treat ophthalmic patients, although occasionally an aspect of the exam may need to be forfeited due to circumstances.
- Components of the minimum ophthalmic database include: menace response, direct and consensual pupillary light reflex, palpebral reflex, Schirmer tear test, fluorescein stain, and tonometry.

■ Initial approach

I recommend examining all patients initially from a distance. This will help determine if the problem is unilateral or bilateral (if it is an externally visible problem). Observe the relationship of the globe to the orbit and eyelids, and to the other globe, and ask yourself the following questions:

- What is the size of the eye — small, normal or enlarged?
- What is the position of the eye — protruding or sunken into the orbit?
- Is there a difference between the axes of the two eyes?
- Is there any evidence of periorbital swelling?
- Is there any ocular discharge and if so, what is its character (serous, mucoid, or sanguineous)?

Canine ocular emergencies most frequently present as the result of trauma to the eye or periocular structures, or any ophthalmic condition that has developed rapidly. Trauma to the orbit or globe may result from a variety of insults, including concussive forces (vehicle accidents or falling from a height), penetrating foreign bodies, fight

wounds, and chemical and thermal burns, to name just a few. Sudden onset eye problems include retrobulbar processes leading to altered globe position and/or swelling of periorcular tissues, rapidly progressive corneal ulcers with impending perforation, uveitis, or sudden blindness. As with any ophthalmic condition, concern for the patient's vision and ocular comfort should guide your diagnostic and therapeutic plan.

Regardless of the nature of the emergency, a complete ophthalmic examination should be performed to ensure that both the correct diagnosis is reached and that any concurrent ocular disease is recognized and treated appropriately. For example, when presented with a corneal ulcer, if the lids and intraocular structures are not also carefully examined, the underlying cause for the ulcer (e.g., an eyelid abnormality with distichiasis) may be missed. Furthermore, if the intraocular structures are not carefully examined, any reflex uveitis resulting from the corneal ulceration may be missed. If the eyelid abnormality is not addressed, the ulcer will likely not heal and may progress in severity. If the concurrent uveitis is not treated, more serious vision-threatening sequelae may ensue, such as synechia, cataract, or glaucoma. Thorough examination of all external and internal ocular structures of both eyes, even if the patient presents with a unilateral problem, should be attempted on all ophthalmic emergencies. A minimum ophthalmic database, to include menace response (**Figure 1**), pupillary light reflex, tear testing, fluorescein staining (**Figure 2**), and intraocular pressure measurement (**Figure 3**), should be acquired whenever possible (1). Occasionally an aspect of the exam may need to be forfeited due to circumstances, e.g., tonometry should not be performed on an eye with a descemetocoele due to risk of globe rupture.

Orbital conditions

Blunt or penetrating trauma may cause significant orbital damage. Ocular proptosis (**Figure 4**), whereby the equator of the globe advances beyond the margin of the palpebral fissure, is not uncommon, and carries a grave prognosis in dogs with a dolicocephalic (e.g., sight-hound breeds) conformation versus brachycephalic (e.g., Pekingese and Shih Tzu). This is due to the physical force required to proptose a globe that is well-situated in the orbit compared to the relative ease with which brachycephalic canine globes can be extruded.

When presented with a proptosis, assessment and stabilization of the entire patient is paramount. If the dog has



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Figure 1. Menace response in a dog. Note that the contralateral eye is covered and the practitioner uses only one finger to menace the dog, thus preventing air currents or inadvertent touch of the vibrissae.



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Figure 2. A dog after fluorescein stain application revealing a superficial corneal ulcer.



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Figure 3. Proper positioning and use of a tonometer for measurement of intraocular pressure.



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Figure 4. Bilateral proptosis secondary to trauma in a Boston Terrier. The globes could not be salvaged and bilateral enucleation was performed.

sustained a proptosis as a result of a severe concussive injury, always first treat for any signs of shock, cerebral edema or hemorrhage, and respiratory or cardiovascular compromise. Careful examination for facial deformities, epistaxis, crepitus, and subcutaneous edema can help determine the extent of the ocular damage. Traumatic proptosis results in compromise of the globe's vascular supply and rapid, significant peribulbar swelling. Extraocular muscles may be avulsed, resulting in permanent strabismus. The optic nerve will have been stretched, potentially resulting in blindness of the affected eye, but vision in the contralateral eye can also be adversely affected due to traction across the optic chiasm. Immediate therapy should focus on keeping the globe moist, and owners should be advised to lubricate the eye during transport wherever possible; any over-the-counter ocular lubricant is suitable. Negative prognostic indicators for salvage of the globe include rupture of three or more extraocular muscles, lack of a consensual pupillary light reflex to the contralateral eye, corneal laceration that extends past the limbus, and extensive hyphema (2). Providing the patient is stable for general anesthesia and the globe is deemed salvageable, surgery should be undertaken promptly. The eye and periorbital tissues should be cleaned with a dilute (1:50) povidine-iodine solution and sterile saline, and a lateral canthotomy performed to facilitate globe replacement. Once achieved, temporary tarsorrhaphy is performed by placing three or four horizontal mattress sutures of 4-0 or 5-0 silk with stents (e.g., sectioned IV tubing) to prevent eyelid tissue necrosis. A small (2-4 mm) area at the medial canthus may be left open to facilitate application of topical medications. Proper placement of mattress sutures requires

careful attention; the needle should be inserted 4-5 mm from the eyelid margin and exit at, or just external to, the opening of the meibomian glands but inside the cilia. If sutures are placed too far external, entropion will result; however sutures that are placed internal to the opening of the meibomian glands will rub on the cornea and cause severe ulceration. The canthotomy incision should be closed in two layers. I advocate leaving all sutures in place for 10-14 days, as premature removal of the tarsorrhaphy sutures may result in re-proptosis due to significant peri-bulbar edema and hemorrhage. Intravenous broad-spectrum antibiotics and systemic anti-inflammatory corticosteroids are recommended at the time of surgery to prevent secondary infection and reduce both periorbital and intraocular inflammation. Many ophthalmologists also advocate the use of broad-spectrum oral antibiotics and a tapering dose of oral corticosteroids for 7-10 days after surgery. Topical treatment (instilled at the medial canthus) with broad-spectrum antibiotics (4 times daily) and topical atropine (1-3 times daily) for the uveitis is also recommended while the sutures are in place.

Exophthalmos (abnormal protrusion of the eye) may have a sudden onset or be a slowly progressive disease that the owner appreciates as a sudden change in the dog's appearance (**Figure 5**). Exophthalmos is caused by an accumulation of air, fluid (edema, hemorrhage) or cells (inflammatory, neoplastic) within the intraconal or extraconal space (**Figure 6**). The location and nature of the infiltrate will alter the appearance of the eye and may affect the overall health of the animal at presentation (3). Orbital cellulitis and retrobulbar infections are usually associated with severe pain upon opening the mouth or when retropulsion of the globe is attempted. Dogs may

Figure 5. A Greyhound with exophthalmos and lateral strabismus secondary to a retrobulbar tumor; this was an acute presentation.



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be febrile, anorexic, and lethargic. Thorough oral examination is essential in such cases, to look for evidence of tooth root abscessation or fluctuant swelling behind the last molar tooth in the upper arcade. If the latter finding is noted, drainage may be attempted under general anesthesia via a small mucosal stab incision into the pterygopalatine fossa and careful insertion of a closed hemostat into the orbit, with slight opening of the hemostat upon withdrawal. Any obvious foreign body protruding from this space can be gently removed (**Figure 7**). Gentle lavage with sterile saline may promote drainage, and cytology and bacterial culture with sensitivity should be obtained, with appropriate systemic antibiotic therapy for 2-4 weeks.

Note that retrobulbar neoplasia is typically more slowly progressive and not associated with severe acute pain upon opening the mouth. Advanced imaging techniques (e.g., orbital ultrasound, computer tomography scan or magnetic resonance imaging) are often required to effectively delineate the extent of involvement and to aid in surgical planning for biopsy or debulking (4-6), and therapy depends on the type of neoplasia, extent of local involvement, and overall health of the animal. While orbital neoplasia does not usually represent a true emergency *per se*, adverse sequelae from prolonged globe exposure may lead to secondary conditions such as corneal ulceration which can threaten the health of the eye.

Adnexal and conjunctival problems

Ocular emergencies involving the eyelid and conjunctiva are frequently the result of concussive forces (vehicle accident or “high-rise syndrome”) or fighting injuries. While damage to the eyelids is usually obvious, injuries to

Figure 6. Bilateral exophthalmos in a Labrador Retriever with lymphosarcoma.



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Figure 7. A large stick foreign body being removed with a pair of hemostats from the pterygopalatine fossa of a dog presenting with exophthalmos and secondary corneal ulceration.



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Figure 8. Lacerated leading edge of the third eyelid in a dog secondary to a cat-scratch injury. The pupil has been pharmacologically dilated to carefully screen for any intraocular damage.

the third eyelid (**Figure 8**) or deeper ocular structures may be difficult to detect if significant chemosis or conjunctival hemorrhage is present. Careful examination of the intraocular structures is critical, since concurrent globe penetration is potentially more threatening to the long-term health of the eye. Intraocular involvement should be suspected if dyscoria or a shallow anterior chamber is observed, or if intraocular pressure is low. A clear ocular discharge may indicate aqueous humor leakage and can be confirmed by performing a Seidel test (1). This involves applying fluorescein stain to the corneal surface; prior to rinsing the eye with sterile eye-wash, carefully observe for a clear rivulet of fluid emanating from the corneal wound and diluting the fluorescein stain, confirming the presence of corneal perforation.

Untreated eyelid injuries or abnormalities result in a defective lid margin and function. Lacerations should be treated by primary repair and every effort should be made to preserve as much eyelid tissue as possible. I recommend minimal debridement followed by closure using a simple interrupted, double layer method with 7-0 to 5-0 suture (using absorbable material for the subconjunctival layer and non-absorbable in the skin). Closure of the eyelid margin must be meticulous to avoid any long-term “step” irregularities and subsequent corneal abrasion; a modified cruciate or figure-of-eight suture provides good apposition of the lid margin (7,8). If eyelid trauma near the medial canthus damages any part of the nasolacrimal puncta, canaliculi, or duct, reconstruction should be undertaken with microsurgical instrumentation and magnification. Topical and systemic antibiotics for 7-10 days and an Elizabethan collar to prevent further self-trauma are recommended for eyelid wounds, with skin sutures removed after 10-14 days. Prognosis is excellent if proper surgical apposition has been achieved and the wound is not infected.

Conjunctival damage may manifest as chemosis, hemorrhage, and/or localized swelling. As with eyelid trauma, intraocular structures should be critically examined for evidence of involvement. In most cases, treatment requires only protection from corneal desiccation and the prevention of secondary infection; topical broad-spectrum antibiotic (applied 3-4 times daily for 7-10 days) is adequate. A single dose of systemic anti-inflammatory medication may also be considered to help reduce acute swelling.

Corneal ulceration

Disruption of the corneal epithelium with variable loss of corneal stroma defines corneal ulceration (**Figure 2**). Affected dogs frequently present with acute, unilateral blepharospasm and epiphora. Anisocoria, due to reflex uveitis following corneal trigeminal nerve stimulation, results in miosis of the affected eye. Variable degrees of aqueous flare (anterior uveitis) can be detected depending on the ulcer's severity and duration. If corneal perforation — secondary to a penetrating foreign body or cat claw — has occurred, aqueous leakage (as seen by a positive Seidel test), hyphema, or iris prolapse may also be present (1,9) (**Figure 9**). Variable degrees of corneal edema will be evident. Fluorescein stain will adhere to any exposed corneal stroma and is an essential diagnostic tool to fully delineate the ulcer's extent. Ocular ultrasound may be helpful when anterior segment disease

(e.g., severe corneal edema and/or hyphema) preclude adequate intraocular examination (**Figure 10**).

When assessing corneal ulceration, ask yourself the following questions:

- What is the size, shape, depth, and duration of the corneal ulcer?
- What is the underlying cause of the ulcer?
- What is the health of the surrounding cornea (*i.e.*, does this ulcer look infected)?
- What is the proximity of the ulcer to the limbus (from which a neovascular response promoting healing may occur)?

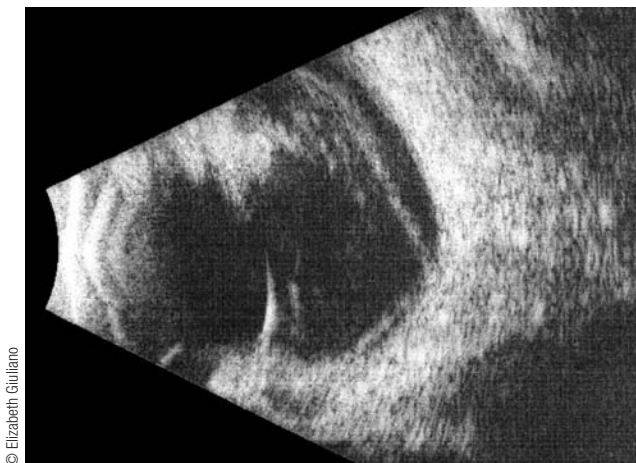
Initial therapy is directed at determining and correcting the underlying cause of ulceration. Prevention of corneal infection and treatment of reflex uveitis should be initiated through broad-spectrum topical antibiotic therapy (4-6 times daily) and mydriatic cycloplegia with atropine to effect in superficial, uncomplicated corneal ulcers. Systemic analgesics will improve comfort in animals in pain; but topical anesthetics should be only used for diagnostic purposes since long-term use adversely affects corneal wound healing. Surgical repair of corneal ulcers is recommended in the following:

- Loss of 50% or more of the corneal stroma
- Rapidly progressing ulcers
- Infected ulcers (as evidenced by yellow/white corneal cellular infiltrate, significant corneal edema, mucopurulent ocular discharge, and moderate to severe uveitis (**Figure 11**))
- Descemetocoeles, or
- Corneal perforations

Figure 9. Focal corneal perforation and iris prolapse in a Boston Terrier secondary to a cat claw injury. Note the obvious anterior synechia.



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Figure 10. B-scan ocular ultrasound of a dog's eye revealing retinal detachment.



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Figure 11. Severe corneal ulcer: there is more than 90% stromal loss in the axial cornea. Note the extensive corneal neovascularization and edema surrounding the ulcer bed.

There are various surgical repair methods including conjunctival grafts, corneal-scleral transposition, cyanoacrylate glue, and penetrating keratoplasty, and these procedures are described in greater detail elsewhere (7, 10, 11). In complicated corneal ulcers, topical and systemic antibiotic therapy should be based on microbial culture and sensitivity results obtained from the ulcer bed. Topical antimicrobials may be administered hourly in infected or rapidly progressive ulcers during the initial stages of treatment. Topical atropine should be administered 2-4 times daily until pupillary dilation is achieved and then given only as needed to affect mydriasis. I recommend topical solutions (rather than ointments) if corneal perforation is imminent, and owners should be educated as to the correct technique to administer drops (**Figure 12**). Topical anti-protease agents (e.g., N-acetylcysteine, fresh serum, EDTA) may also be applied topically (every 2-6 hours) to inhibit progression of corneal malacia. Systemic antibiotic therapy is beneficial if conjunctival grafting has been performed or if corneal perforation has occurred. Systemic nonsteroidal anti-inflammatory drugs will ameliorate uveitis and ocular discomfort, but care must be taken to avoid excessive use in dogs due to their association with gastric ulceration, hemorrhage, vomiting and diarrhea (12). Topical and systemic corticosteroids are contraindicated in complicated or infected corneal ulcers as they delay wound healing and increase collagenase activity (13, 14). An Elizabethan collar is recommended to prevent self-trauma to the compromised globe while healing.

Corneal foreign bodies

Corneal foreign bodies (e.g., plant material, metal



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Figure 12. Proper positioning for administering topical medications to a dog's eye. Note that the head is elevated and the drop is administered from above to avoid any part of the bottle touching the eye or eyelids.

fragments) result in acute blepharospasm and epiphora (**Figure 13**). Following application of topical anesthesia, superficial foreign bodies may be removed by aggressive flushing with sterile eyewash or gentle manipulation with a moistened cotton-tipped applicator. Foreign bodies embedded deeper within the stroma frequently require surgical removal under general anesthesia. Care must be taken to avoid inadvertently pushing the foreign body more deeply into the eye, resulting in corneal perforation. A 25-27 G hypodermic needle may be used to engage the foreign body at 90° to its long axis which can then be removed in a direction retrograde from which it entered the cornea. Following removal, treatment consists of

standard corneal ulcer management. Prognosis for canine corneal foreign bodies is generally good providing the iris and lens have been spared, but note that removal of firmly implanted corneal foreign bodies or those entering the anterior chamber require microsurgery for removal (**Figure 14**) and should be referred to a veterinary ophthalmologist if possible. Foreign body perforation of the lens capsule may cause phacoclastic uveitis, resulting in the demise of the globe.

Uveitis

A thorough review of the clinical findings, diagnosis and treatment of canine uveitis is beyond the scope of this

article, but uveitis and its sequelae (cataract, glaucoma, lens luxation) represent a significant threat to vision and ocular comfort. Clinical findings depend on the cause (endogenous versus exogenous factors) and duration, but the hallmark features of uveitis include pain, episcleral and associated conjunctival vascular congestion, corneal edema, aqueous flare, fibrin and hemorrhage in the anterior chamber, keratic precipitates, rubeosis iridis, miosis, and hypotony (**Figure 15**). Posterior uveitis may result in retinal detachment and blindness. Identification of the underlying cause dictates specific therapy, but symptomatic therapy consists of mydriatic cycloplegics and anti-inflammatory agents.



Figure 13. Plant foreign body in a dog. Note the surrounding corneal ulceration and the reflex uveitis as evidenced by miosis.



Figure 14. A foreign body embedded within the cornea and protruding into the anterior chamber of a dog. Referral to a veterinary ophthalmologist for intraocular surgery is highly recommended in this situation.



Figure 15. A German Shepherd dog with uveitis. Note the elevated third eyelid, clear cornea (as evidenced by the crisp flash artifacts on the corneal epithelium), significant aqueous flare with dependent sero-fibrinous clot in the anterior chamber, and miotic pupil.



Figure 16. A mixed breed dog with chronic glaucoma; the intraocular pressure was 42 mmHg (normal value: 15-25 mmHg). The globe is mildly buphthalmic, and scleral injection and intraocular hypohemia are evident.

■ Other problems

Glaucoma can occur as a primary disease in dogs, but may also be secondary to uveitis, hyphema, or neoplasia (**Figure 16**). Similarly, lens luxation occurs as a primary

disease in dogs, especially in terrier breeds, or secondary to chronic uveitis, and readers are referred to more complete discussions of glaucoma and lenticular diseases elsewhere (15).

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Emergency visits to primary care veterinary hospitals



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■ Introduction

This paper will present some basic epidemiology of dogs seen for emergency care at a network of primary veterinary clinics in the United States of America. The findings presented in this paper are only intended to provide a bird's eye view of the common causes of emergency visits to most primary care veterinary practices, and the progress and clinical outcomes for each category are beyond the scope of this paper.

■ Methods of analysis

The health records of all dogs presented at Banfield Pet Hospital during 2014 were screened to identify those that came in as an "emergency" visit. To be included, a case had to meet at least one of the following criteria: the reason for the visit was recorded as "emergency", the owner was invoiced for "emergency medical treatment" (e.g., emergency/urgent/after hours care), or there was a diagnosis of "hit-by-car" (HBC). A count of the diagnosis entered at each visit was made; from this list, any condition likely to be unrelated to the reason for the emergency visit (e.g., dental calculus, nuclear sclerosis) was removed, and a list of the top 10 "emergency diagnoses" created. These were then grouped into 10 categories: dermatologic conditions (e.g., abrasion/wound/trauma, abscess, bites); HBC or bone fracture; respiratory disease (e.g., bronchitis, tracheal collapse,

asthma, coughing, dyspnea); toxin exposure (e.g., plant, chemical or other medicinal toxicity); allergic reaction (e.g., anaphylaxis, urticaria); neurologic disease (e.g., seizures, anisocoria, vestibular disease); gastrointestinal disease (e.g., vomiting, diarrhea, hepatic disorder, pancreatitis); endocrine/metabolic disease (e.g., diabetes mellitus, diabetic ketoacidosis, adrenal disorder); urogenital disease (e.g., dystocia, pyometra, eclampsia, kidney disease, urinary tract obstruction); or nonspecific (e.g., malaise, anorexia, fever). Co-occurrence of certain problems (bone fracture, skin wound, respiratory disease or neurologic disease) related to a hit-by-car diagnosis was also investigated.

■ Results

Nearly 2.4 million dogs were seen in almost 7 million pet visits to Banfield Pet Hospital in 2014. This included 21,840 dogs (0.9%) seen for 22,625 "emergency" examinations, and of these, approximately 57.7% (13,056) of the visits had an exact diagnosis entered into the appropriate field of the health record. The top 10 breeds presented as an emergency are shown in **Table 1**; Chihuahuas and Labrador Retrievers were the most common breeds seen. Top diagnoses and the frequency of each diagnosis are shown in **Tables 2a and b**; HBC was the most common cause of an emergency, at 22.8% of all visits. Within the ten "emergency categories", conditions within the "dermatologic" sector were

Table 1. Top ten breeds of dogs seen as an “emergency” visit in 2014*.

Dogs	Number of unique pets seen	Percentage of emergency pets
Chihuahua	2,114	9.7%
Labrador Retriever	1,932	8.8%
Pit Bull	1,292	5.9%
Yorkshire Terrier	1,247	5.7%
Shih Tzu	1,060	4.9%
Dachshund	795	3.6%
Mixed breed	742	3.4%
German Shepherd	720	3.3%
Boxer	691	3.2%
Maltese	676	3.1%

*The list of the top affected breeds is very similar to their breed representation in the overall Banfield pet population seen during the course of the year.

most commonly encountered (25.4% of all cases), followed by the “HBC/bone fracture” category (24.5%). With regard to animals hit by a car, concurrent injuries were not uncommon, as shown in **Table 3**; for example 27.8% of dogs had related dermatologic wounds and 11.5% had bone fractures.

■ Discussion

The list of diagnoses from emergency visits will not be a surprise to the general practice veterinarian. It is tempting to suspect there might be a breed predisposition to having an injury or condition resulting in an emergency visit, but this is unlikely as the list of the top affected breeds is very similar to their breed representation in the overall Banfield pet population seen during the course of the year. It is possible that HBC as the most common diagnosis was biased, as the definition of an “emergency visit” for this study included all pets involved in a vehicle incident, irrespective of whether or not the animal was deemed to require an “emergency” visit. In addition, there is potential bias because over 40% of the cases did not have an exact diagnosis entered in the appropriate field of the health record; however, a random review of these cases found that their actual problems could be categorized with frequencies similar to those cases where a diagnosis had been entered. It is therefore unlikely that HBC cases (and indeed the other diagnoses) in **Tables 2a and b** are either over- or under-represented, and it is therefore reasonable to assume that the percentages for the co-occurrence of certain conditions related to car accidents, as shown in **Table 3**, are fairly accurate.

Table 2a. Most common presentations for “emergency” visits.

Specific diagnosis	Number of emergency visits with this diagnosis	% of emergency visits with diagnosis
HBC (hit by car)	2,975	22.8%
Seizures	1,362	10.4%
Poisoning/toxicity	942	7.2%
Malaise	836	6.4%
Laceration	733	5.6%
Abrasion	717	5.5%
Wound from animal bite	590	4.5%
Allergic reaction**	501	3.8%
Allergic reaction (acute)**	406	3.1%
Hepatopathy	356	2.7%

Table 2b. The frequency of each diagnosis group for “emergency” visits.

Diagnosis group	Number of emergency visits	% of emergency visits
Dermatologic problems	3,322	25.4%
HBC/bone fracture	3,197	24.5%
Gastrointestinal	2,032	15.6%
Neurologic	1,694	13.0%
Toxin/poison	1,565	12.0%
Nonspecific	1,117	8.6%
Allergy/allergic reaction	1,077	8.3%
Respiratory	660	5.1%
Urogenital	319	2.4%
Endocrine/metabolic	242	1.9%

Table 3. Select comorbidities in dogs seen as hit-by-car “emergency” cases.

Number of HBC cases***	2,453
% with bone fracture	11.5%
% with dermatologic wound	27.8%
% with respiratory diagnosis	3.8%
% with neurologic diagnosis	1.9%

***“Allergy” covers problems such as skin reactions whilst “acute allergy” was used for life-threatening or severe conditions.

***Note that the number of HBC cases are slightly less than the number of HBC visits given in **Table 2a**; this is because some animals had more than one visit for a HBC incident.

Emergency management of open fractures



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■ Introduction

An open fracture is any fractured bone that is exposed to environmental contamination due to disruption of soft tissues surrounding the bone. By extension, if there is a skin wound in a limb or body segment that has a fracture, this should be considered as an open fracture, regardless of whether or not the fracture is assumed to communicate with the wound. One study reported that

open fractures occurred in 16.7% of all traumatic fractures in dogs and cats, and vehicular trauma, younger age, larger body weight, and fracture comminution were associated with a greater likelihood of open fracture (1). Proper treatment of open fractures requires recognition of two central facts:

1. The degree of future morbidity resulting from open fracture repairs is often directly related to the initial emergency management of the fracture.
2. Open fractures present unique management challenges to the practicing surgeon in terms of additional considerations for fracture repair and in wound care and closure.

KEY POINTS

- Any fracture with a skin wound anywhere in that body segment should be considered an open fracture and to be at increased risk for later infection.
- Open fracture wounds should be treated on an emergency basis, but immediate rigid stabilization of the fracture itself is not an emergency.
- Every patient with vehicular trauma should receive a minimum of chest and abdominal radiographs, CBC, serum chemistry, ECG, pulse oximetry and blood pressure measurements to assess potential co-morbidities.
- Initial sterile wound dressings should be applied while the patient is assessed and stabilized to protect against infections from nosocomial organisms, and systemic broad-spectrum antimicrobials should be given as soon as possible.
- External skeletal fixators allow open wound access while providing rigid fixation, preserving bone blood supply and minimizing soft tissue disruption.

Open fractures are often the results of vehicular trauma or other high-energy events, and such trauma can result in significant co-morbidity that must be addressed. In addition to limiting future morbidity, initial management of patients with open fractures is critically important to minimizing cost and healing time, and to the functional outcome of the patient. Especially when treating open fractures, veterinarians are best advised to strictly adhere to established principles and not attempt to cut corners to save time, cost or effort. Post-operative osteomyelitis or non-union nearly always results from compromises made in initial wound and fracture care. **Figure 1** provides a useful algorithm for management of patients with open fractures.

■ Patient assessment

Open fractures should always be appropriately treated on an emergency basis, but the fractured bone itself does not need emergency repair. Certainly, recognition

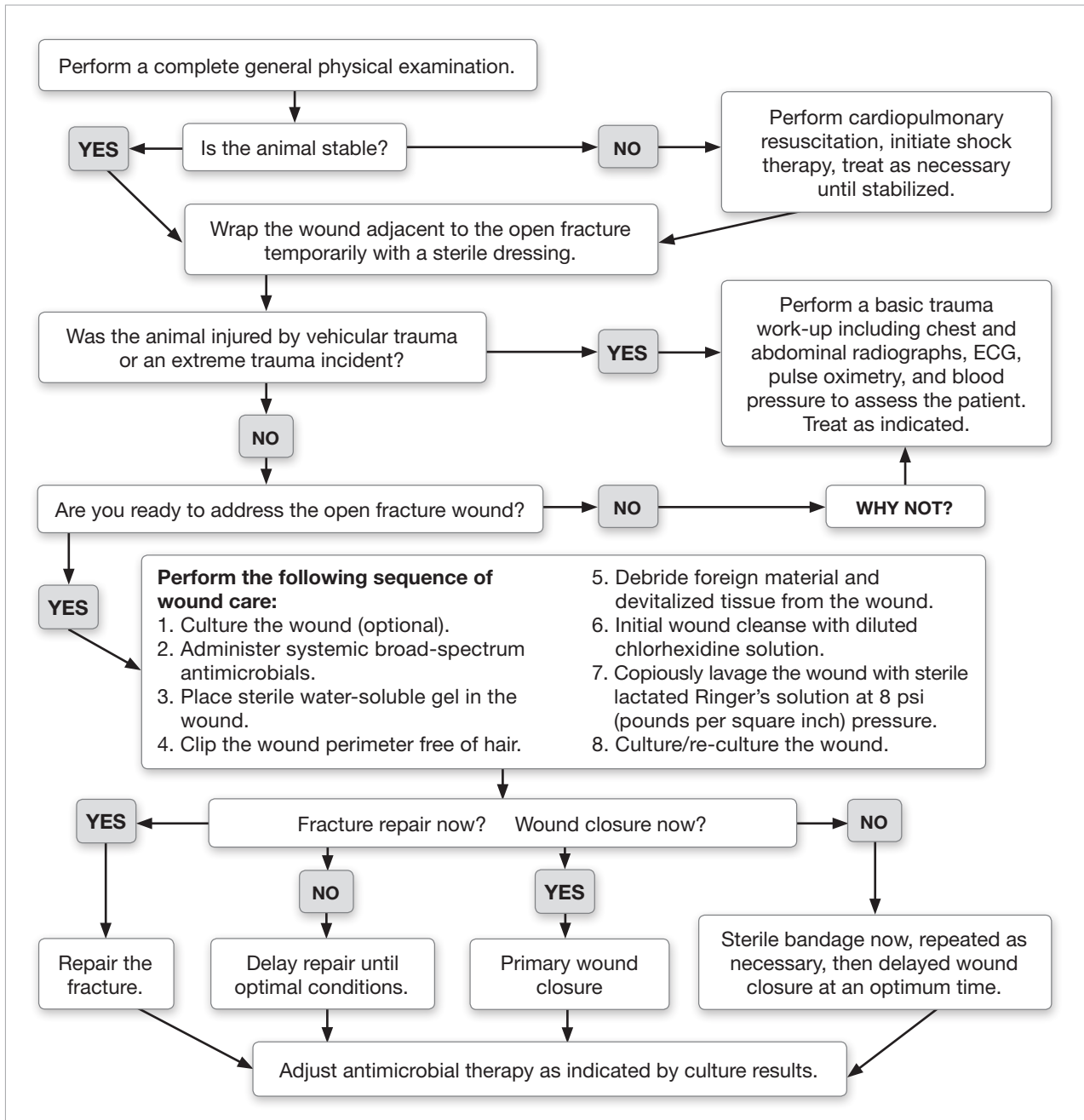


Figure 1. Emergency management of open fractures.

of the open nature of a fracture does not supersede the care of more life-threatening injuries or co-morbidities. The first and most important action in open fracture diagnosis and management is to thoroughly assess the patient for additional systemic abnormalities. All animals with open fractures due to trauma should be assessed for occult injuries to the thorax and abdomen, and should have a complete neurologic examination in order to rule out pre-existing neurologic dysfunction or additional

neurologic injury. One study reported that 57% of dogs with skeletal injury had radiographic, electrocardiographic, or other evidence of thoracic trauma, including pulmonary contusion, myocardial contusion, pneumothorax, or diaphragmatic hernia (2); however, only 21% of the dogs had associated clinical signs of thoracic injury. Every animal sustaining vehicular or other trauma sufficient to result in long bone fracture should receive a minimum database of routine chest and abdominal

radiographs, complete blood count (CBC), serum chemistry, blood pressure assessment, pulse oximetry and a diagnostic electrocardiogram (ECG). Post-traumatic cardiac arrhythmias may not appear for 48-72 hours after the event, so ECG's that are initially normal should be repeated at 12-hour intervals for 72 hours post-trauma. Patients with initial or subsequent cardiac arrhythmias, or with other systemic trauma, should be treated appropriately for any life-threatening injuries, and fracture repair delayed until the physical condition stabilizes. The neurologic status of each animal must be assessed to rule out central nervous system injuries and peripheral fracture-associated injuries. Urologic injuries are common in patients with pelvic or femoral fractures, and may result in hyperkalemia and uremia before the injury is detected. Urine output should be carefully monitored, especially in recumbent animals.

■ Initial open fracture management

Two factors are considered paramount in proper initial management of the fracture itself. The first is the “grade” of the fracture. Open fractures in veterinary patients are commonly classified from Grades I through III (**Table 1**) in an effort to better predict any potential for increased morbidity or post-operative infection, but evidence for the efficacy of grading open fractures in veterinary medicine is scant. Grade I open fractures in the past have been erroneously described in the veterinary literature as fractures where the bone “penetrated from within”, a distinction that implies a pattern of displacement during trauma that cannot be determined simply by viewing the fracture and wound after injury. This assumption of the sequence of trauma should be avoided by veterinarians and in future veterinary literature. Some authors subdivide Grade III open fractures into three subtypes (3), but subclassification for management purposes is not supported by improved fracture outcomes in the available literature.

The second and more important factor in open fracture management is consideration of the nature and duration of the microbial contamination, with a “golden period” for wound closure often described as being a period of 6 to as long as 12 hours from the initial wound trauma. In reality, the “golden period” is not strictly limited to duration, but is more appropriately viewed as the perceived degree of wound contamination or infection that has occurred up to the time of debridement and closure. Within the first 6-12 hours, contaminated wounds, including wounds that communicate with fractures, may be converted to clean wounds by effective surgical debridement and lavage, and then closed in primary fashion, decreasing the time to

Table 1. Open fracture grade definitions.

Grade I	An open fracture with an associated wound < 1 cm in diameter. Grade I fractures are often simple, two-piece fractures and are associated with minimal soft tissue trauma.
Grade II	An open fracture with a skin wound > 1 cm diameter but without extensive soft tissue trauma or comminution.
Grade III	An extensively comminuted open fracture with severe soft tissue trauma and a skin wound of > 1 cm diameter. All fractures caused by projectiles are considered Grade III.

complete wound healing and wound care costs. After 12 hours, most wounds, regardless of any assumption concerning the degree of contamination, should be similarly debrided and lavaged, but should be closed over surgical drains or left open for delayed closure techniques. Decisions regarding wound closure or non-closure should be ideally based on peri-operative examination of a Gram-stained smear of the wound taken before debridement or lavage. The presence of visible bacteria in the smear indicates likely infection of $> 1 \times 10^5$ bacteria/mm² in the wound and therefore a recommendation that the wound should be managed as an open wound until uncomplicated healing with delayed closure techniques is likely to be achieved.

For all patients, a temporary sterile bandage or dressing should be placed over the wound as soon as possible during initial assessment of the patient. Ideally, samples for aerobic and anaerobic culture are collected at the level of the fractured bone at the time of presentation, although a prospective, randomized study found that only 18% of open fracture infections were caused by organisms found on initial culture (4). In a study of bacterial contamination in 110 canine fractures, 72.7% of dogs with open fractures had a positive culture for aerobic and/or anaerobic micro-organisms (5). Appropriate doses of broad-spectrum antimicrobials (see below) should be given systemically immediately after wound culture. During wound care, the animal should be placed in an aseptic environment such as a surgical suite, and all personnel should follow aseptic protocols to minimize iatrogenic contamination. Regardless of the assessed open fracture grade, and as soon as the patient is otherwise determined to be systemically stable, the open

wound should be clipped free of hair to a wide margin, cleaned of gross debris by surgical antiseptic soaps, and all damaged or necrotic soft tissue debrided (**Figure 2**). To decrease iatrogenic contamination of the wound, the wound can be filled with sterile, water-soluble lubricant prior to clipping. Isolated bone fragments without soft tissue attachments should be removed. After debridement an initial cleansing with diluted chlorhexidine gluconate solution is recommended (3).

Debrided and cleansed wounds should then be copiously lavaged with sterile lactated Ringer's solution or similar sterile isotonic solution; lavage volumes of 3-5 liters of isotonic solution in a 1 cm diameter wound are not excessive. It is important that lavage is achieved by a pressure of 7-8 psi (pounds per square inch) to assure adequate disruption of bacterial adherence from the tissue while causing minimal damage to healthy tissues adjacent to the wound; this is achievable with either a commercial surgical lavage device or by using a 19 G needle placed on a 60 cc syringe and vigorously sprayed over the wound. These methods assure a fluid pressure of approximately 8 psi, which is the strength of bacterial adherence to the wound surfaces. Higher pressure lavage is detrimental to healthy tissue and is not recommended. The use of antibiotics or antiseptic compounds in the lavage solution is not necessary and may be detrimental to the normal cellular elements of the tissue, but a solution of 0.05% chlorhexidine has been determined to provide antibacterial activity without causing tissue reaction (6).

Figure 2. This open Grade II radius and ulna fracture has been clipped of hair, but the wound site has not yet been debrided or lavaged.



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Cleansing, debridement, and lavage should include the entire depth of the wound down to the fracture. Following copious lavage, the wound should again be aerobically and anaerobically cultured to assess and properly treat the microbial population left in the wound at the time of closure. At this stage the clinician should assess the tissue viability and degree of contamination and choose one of three options; repair the wound by primary means; repair the wound using a closed, sterilely maintained surgical drain; or manage the open wound with sterile dressing materials until closure can be performed at a later date, or until secondary intention healing occurs.

■ Broad-spectrum antimicrobial therapy

Broad-spectrum antimicrobials should be administered after the initial aerobic and anaerobic wound culture is performed. The combination of a first or second generation cephalosporin and a fluoroquinolone has been recommended for coverage of both Gram-positive and Gram-negative organisms (3,4); e.g., cefazolin (22 mg/kg IV Q6hr) and enrofloxacin (5 mg/kg IM Q12hr) is a common initial choice until definitive culture and sensitivity results are known. Because hospital-specific nosocomial organisms are common causes of open fracture infections, broad-spectrum antimicrobial regimens should be chosen and modified based on the nosocomial surveillance within an individual hospital. Antimicrobial choices should be adjusted based on culture and sensitivity results and therapy should continue for a minimum of 28 days post fracture, and even animals with negative wound cultures should receive broad-spectrum antimicrobials for this minimum period before prophylactic therapy is terminated. While the earliest possible antimicrobial administration in traumatically induced open fractures has been advocated, recent reports suggest that the timing of administration is not as critical in infection rates after open fractures (7).

In general, Grade I open fractures can be cleaned and the wound closed primarily if the trauma occurred less than 6-12 hours prior to closure. Grade II fractures are often more contaminated and have increased potential for infection, but wounds in these fractures may also be converted to clean wounds by proper debridement and lavage and subsequently closed primarily. Grade III fractures, which include all projectile wounds, should never be closed, but should be managed as open wounds until delayed primary or secondary closure is indicated, or until second intention healing occurs. If the surgeon chooses to manage the wound as an open wound post-operatively, the area should initially receive daily or

twice-daily debridement and lavage, followed each time by placement of fresh, sterile, wet-to-dry bandages until granulation tissue forms and then non-adherent dressings applied until surgical closure or healing occurs. Debridement and bandaging frequency can be decreased as wound exudate and appearance allow. Closure at the earliest appropriate time for the individual wound is always in the best interests of decreased patient morbidity.

■ Temporary and definitive fracture stabilization

Open fractures do not require definitive stabilization immediately if proper emergency wound care has occurred. Definitive rigid fracture stabilization should only occur when the patient is appropriately stabilized, an experienced surgeon is to hand, and with all anticipated fixation devices and equipment immediately available.

Temporary stabilization of open fractures is performed to increase patient comfort and to minimize local soft tissue swelling and further soft tissue injury. Fractures of the lower extremities have less soft tissue coverage, and closed fractures may become open fractures or undergo additional comminution if unsupported. Analgesics (preferably opioid agonists such as morphine) should be administered to the patient to improve comfort.

Fractures proximal to the elbow or stifle are difficult to stabilize with external coaptation alone, and the patient should be cage-confined without splinting and treated with analgesics until definitive repair. Fractures distal to the elbow or stifle should be stabilized with external coaptation until definitive fixation or while awaiting transport to a referral center. External coaptation should consist of either a Robert-Jones bandage, or a modified Robert-Jones incorporating a molded lateral fiberglass splint. For fractures with wounds left open after debridement, all bandage materials should be sterile and applied in aseptic fashion. External coaptation should always immobilize the joint immediately proximal to the fracture and extend distally to the toes.

■ Definitive open fracture repair

The high and increasing standard of care created by the rising expectations of the client and the increased regional availability of surgical specialists leaves fewer general practitioners with either the time or economic incentive to maintain expert knowledge, experience, and equipment for proper open fracture repair. Such fractures are often demanding on the time and resources of

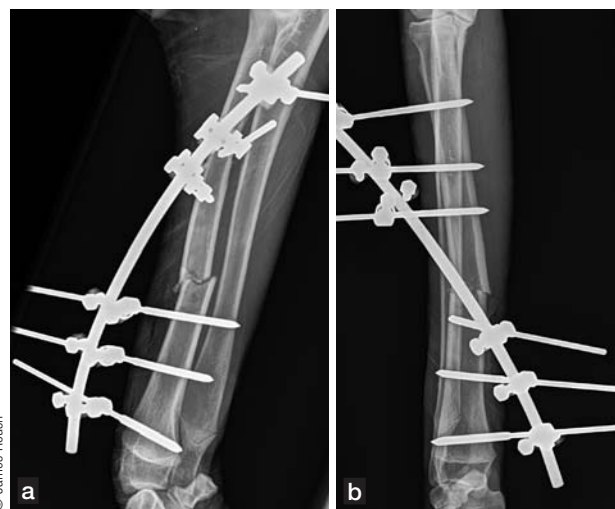
general practitioners and appropriate consideration should always be given for referral to a specialist.

Open fractures with an exposed wound should not be managed with external coaptation for long periods because of the expense, discomfort, and contamination potential from the frequent cast changes necessary to provide for wound care. Definitive and rigid fixation of open fractures should be based, as all fracture repairs:

- On careful pre-operative planning, including assessment of the fracture on orthogonal radiographs or by computed tomography
- On local availability of the surgical expertise, experience and equipment indicated for the particular fracture
- On individual patient considerations such as temperament, patient confinement opportunities, and likely owner compliance

Other unique considerations for the repair of open fractures, in addition to the timing of initial wound cleansing or definitive surgical intervention, are determined primarily by the continued presence of an open wound that requires care. Fractures with wounds that are to be managed in open fashion are particularly amenable to fixation by rigid or circular ring external skeletal fixators because these devices allow frequent bandaging, debridement, lavage and daily wound care without disruption of the fixation, and because the “closed” placement

Figure 3. Post-operative mediolateral (a) and craniocaudal (b) radiographs of an open radius fracture with an applied external skeletal fixator. Such devices are excellent choices for open fracture fixation because they allow care of an open wound while preserving bone blood supply and minimizing soft tissue disruption.



of such devices minimizes further soft tissue trauma and maximizes bone viability (**Figure 3a and b**). Surgeons should not, however, necessarily eliminate bone plating as an option in fractures with open wounds, but if bone plates are applied and exposed to the open wound, the surgeon should anticipate a need to remove the plate after the fracture has healed as it will serve as a nidus for future infection. Bone plates may, in extreme circumstances such as extensive loss of soft tissues over the bone, be left exposed so that the wound and soft tissues may granulate over the plate during healing. Fractured bone will heal in the presence of contamination or infection if the fracture repair remains rigidly stable, and thus bone infection is not a cause for immediate repair revisions. If bone fragments have been debrided and the fracture gap requires a bone graft, delayed autogenous bone grafting is recommended approximately two weeks after wound closure or after existing infections have cleared.

■ Conclusion

Potential complications for open fractures include superficial wound infection, wound dehiscence, acute or chronic osteomyelitis, and delayed union or non-union. Despite a literature search for retrospective or prospective studies of infection rates in dogs with open fractures, the author could not find a report of a large case series within the past two decades. Large, broad case series infection rates after open fractures in people are also not often reported today in lieu of smaller geographically- or bone-specific reports, however a review of infection rates after open tibial fracture in people in the last decade listed infection rates between 0-25% (8), and a recent retrospective study of 296 open radius or ulna fractures had an overall incidence of deep infection of 5% (9). Careful and sterile wound cleansing, debridement, copious lavage, administration of early broad-spectrum antimicrobials, and rigid fracture stabilization are best practices to decrease the incidence of complications in open fractures.

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Penetrating injuries in dogs



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■ Introduction

Penetrating wounds are often deceiving! An innocuous-looking skin puncture may overlie tissue that has been significantly compromised by strong forces, vascular damage, and/or inoculation of bacteria or foreign material. Even if the animal appears stable initially, continuing deterioration of damaged tissue can lead to necrosis, infection, inflammation, sepsis and death. Effective management of penetrating wounds requires first and foremost that the clinician recognizes small wounds can hide severe damage.

■ Forces and tissue damage

A dog bite can generate over 450 psi (pounds per square inch) of force (1), causing both direct and collateral damage to tissues. When the attacker's canine teeth penetrate the skin and the attacker shakes its head, the elasticity of skin allows it to move along with the teeth, so only puncture holes are made in the skin. Subdermally, however, the teeth shear through a wide area of less mobile tissue, avulsing skin from muscle, tearing soft tissue and neurovascular structures, creating dead space, and inoculating bacteria and foreign material. All of this injury is further compounded by the crushing forces exerted by the premolars and molars.

KEY POINTS

- When presented with a bite or bullet wound case, think “iceberg”: a small amount of surface damage often belies a large amount of damage in the deeper tissues!
- Endoscopy allows early detection of esophageal perforations before clinical signs appear.
- Penetrating wounds should be opened, explored, debrided, and lavaged; they are usually then best managed as open wounds. If wounds require closure, they should be closed over a drain.
- If there is a penetrating wound (or suspicion thereof) or significant crush injury to the abdomen, exploratory celiotomy is indicated.
- Foreign objects lodged in the body are best removed via a surgical approach in an operating theatre with an anesthetized, fully prepared patient.

Like bites, bullets cause both direct and collateral damage (**Figure 1**), imparting energy proportional to their mass and velocity [Kinetic Energy = $\frac{1}{2} \times \text{mass} \times \text{velocity}^2$]. Dense tissues (e.g., liver, spleen, bone) absorb more energy than less dense, more elastic tissues (e.g., muscle and lung), which explains why cortical bone hit by a bullet may shatter into multiple pieces (each of which becomes a new projectile) while an identical bullet with the same energy can pass cleanly through a lung lobe. Cavitation – the pressure wave created by a projectile – can mean that a bullet may fracture bones, tear vessels, rupture bowel, and contuse organs that the missile never contacts directly.

The term “iceberg effect” can be used to describe bite and bullet wounds, because the small amount of damage seen on the skin often belies a large amount of damage underneath. In subdermal tissues, necrosis, hematomas, compromised vasculature, dead space, inoculated bacteria, and foreign material stimulate local

inflammatory, immunologic, coagulation, and fibrinolytic cascades. With insufficient treatment, these cascades may overwhelm the body's controls, resulting in systemic inflammatory response syndrome (SIRS) or sepsis (SIRS + infection) (2-4). Patients can appear stable even as the body is ramping up to SIRS, and then acutely decompensate several days after injury. The clinician needs to think about the iceberg effect from the start and be proactive to stop progression to SIRS.

Other penetrating injuries can occur from sticks (e.g., when playing "fetch", running into a stick in the field) or other environmental objects. The amount of energy imparted depends on mass and velocity (of the object or the dog, whichever is moving), and the iceberg effect occurs due to the blunt trauma associated with objects that are not aerodynamic.

■ Patient assessment

Immediately life-threatening injuries such as bleeding and respiratory compromise should be managed first. Wounds over the chest should be covered immediately with a sterile dressing in case they penetrate into the thorax. Ultimately, a full physical exam is performed, including orthopedic and neurologic assessments, and all wounds are examined. This may require extensive shaving; dogs that have been bitten typically have wounds in multiple locations (5,6).

Diagnostic work-up is tailored to the patient's injuries. Hematology and serum chemistry panels provide baseline values as well as evidence of organ compromise due to the injury itself, SIRS, or sepsis. Elevations in lactate and creatine kinase reflect the degree of tissue damage. Orthogonal radiographs, ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) can help determine the path of the penetrating injury, locate foreign material, and define orthopedic damage and internal injuries, although damage to soft tissues, including viscera, cannot be ruled out with imaging alone (3,4,7,8). If the number of intact bullets seen with imaging cannot be reconciled with the number of entry and exit holes seen on the patient, look for stray bullets with additional images or shave further to find additional wounds.

A number of key structures are at risk with penetrating wounds to the neck (9). Severe hemorrhage may indicate laceration of a carotid artery or jugular vein; if necessary, both carotids and/or both jugulars can be ligated at the same time in dogs, assuming normal collateral circulation is intact. Tracheal perforation should be suspected

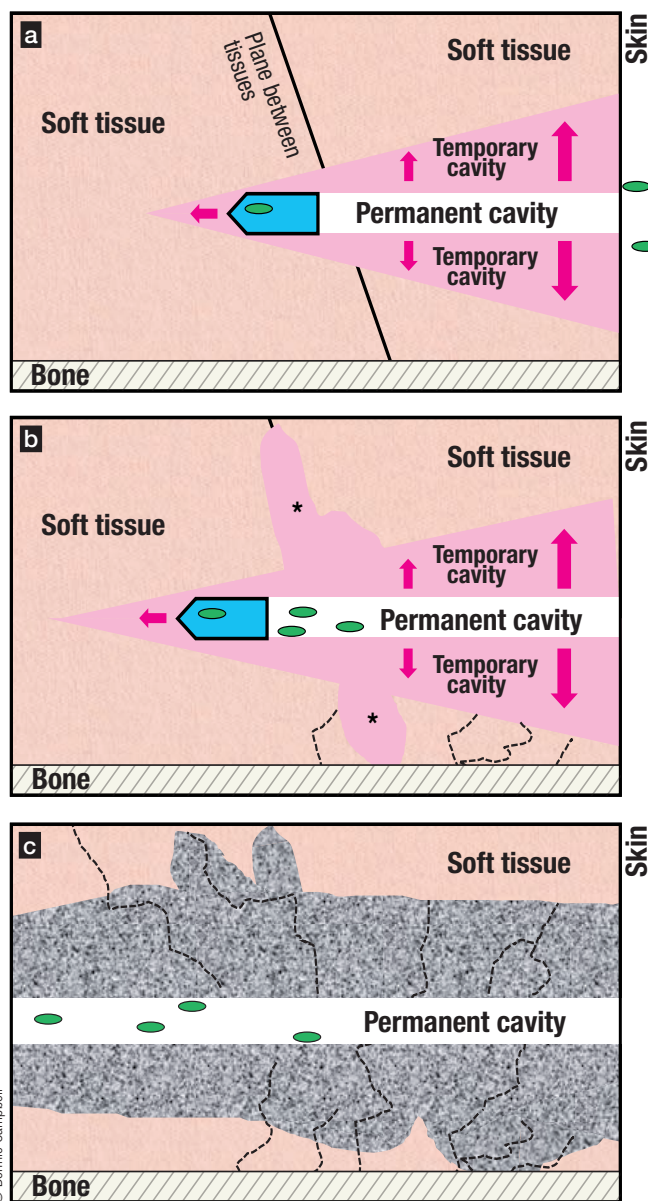


Figure 1.

(a) A bullet enters the body, carrying bacteria and debris (green) from the skin surface. The permanent cavity (white) is created as the bullet moves through tissue directly in its path. The temporary cavity (pink) is created as the cavitation energy moves ahead and perpendicular to the bullet (pink arrows), damaging tissue via compression.

(b) Cavitation energy expands along paths of lesser resistance, such as the fascial plane between muscles (asterisks). Tissue that is not flexible or that is compressed against bone by cavitation can fracture (dashed lines), as can tissues that rebound back together after the cavitation energy dissipates. The bullet's passage creates a vacuum that draws in more bacteria and debris.

(c) Tissues can be damaged (mottled grey, dashed lines) by cavitation although untouched by the bullet.

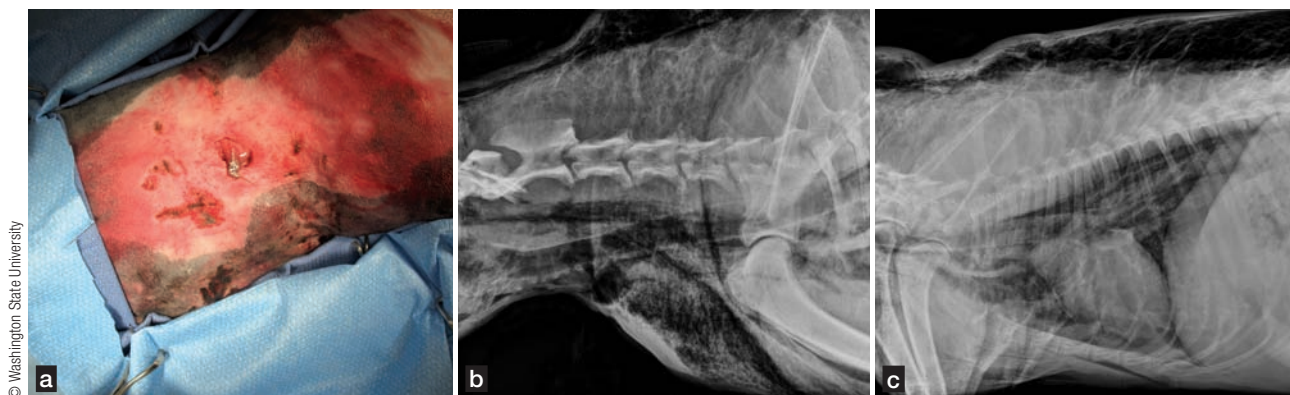


Figure 2. A nine-year-old Border Collie which had been attacked by another dog. (a) Shaving revealed multiple cervical bite wounds (dorsal recumbency, cranial to the left). Cervical (b) and thoracic (c) radiographs show severe subcutaneous emphysema and pneumomediastinum. A 1 cm diameter puncture was found in the trachea at surgery.

in patients with neck wounds and extreme subcutaneous emphysema or pneumomediastinum (**Figure 2**). The esophagus is also at risk of puncture, but clinical signs may not be apparent for several days, during which time ingested food or water accumulates in the cervical tissues. It is thus prudent to scope the esophagus when there are deep wounds to the neck; tracheal damage can also be assessed while scoping.

■ Surgical management

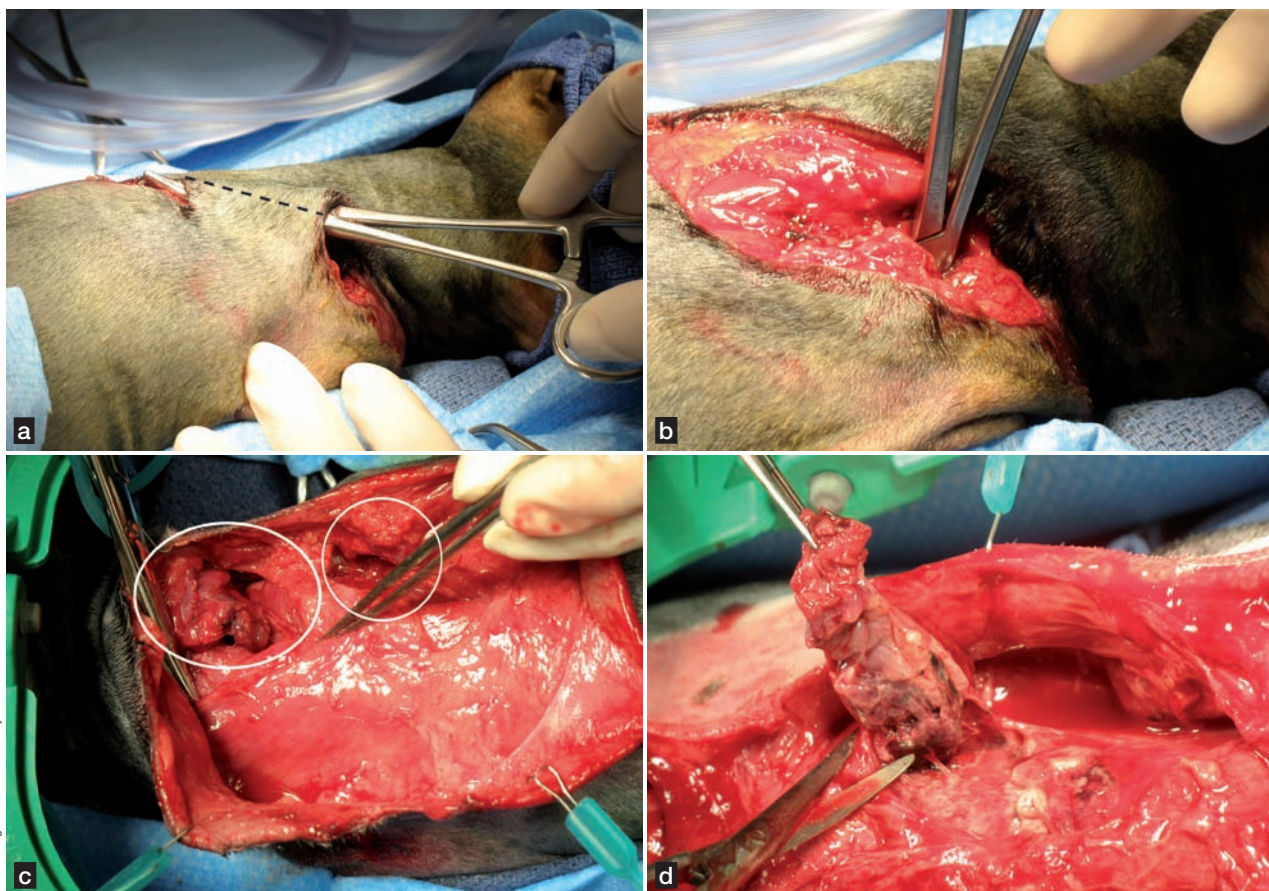
Surgical exploration is needed to fully assess the extent of trauma caused by penetrating injuries (2,3,7). Furthermore, thorough debridement of devitalized, contaminated tissue is the only effective way to prevent or treat SIRS or sepsis. Thus, penetrating wounds should be opened, explored, debrided, and lavaged early on (2,3). If the damage ended directly below the skin, the surgery has been minor. If the damage continued into deeper tissue and/or if foreign material was lodged inside, surgery can prevent considerable morbidity and even mortality.

A large area should be prepped for surgery, since the path(s) of penetration may deviate in the deeper tissues. The surgeon should be prepared to enter the abdomen or chest if necessary. Entry and exit wounds are opened, the underlying tissue is visualized, and path of injury is followed to its deepest extent, debriding damaged tissue along the way (**Figure 3**) (2). In bite wound victims, one can commonly insert a hemostat into one wound and exit it out a number of others due to avulsion of skin that has occurred (**Figure 3a**). When there are multiple bite wounds in an area, one longer incision can be made to access the tissue deep to all of these bite wounds at once.

An instrument or rubber tube can be inserted into the wound tract to aid dissection. It is common to see increasing tissue damage as one follows the tract into deeper tissue (**Figure 3**). Walls separating areas of dead space should be broken down and tissue that is clearly necrotic excised – no matter how much the clinician may wish to save it – as leaving it perpetuates inflammation, blocks granulation, and increases the risk of infection. Signs of necrosis include abnormal color and consistency (dry necrotic tissue is dark/black and leathery; moist necrotic tissue is yellow/gray/white and slimy) and lack of bleeding when cut (assuming the patient is not hypothermic or hypovolemic). Debridement should be continued until viable tissue is reached. Guidelines for debridement of tissue with uncertain viability are in **Table 1**.

Debridement is followed by copious lavage at 7-8 psi, which maximizes removal of debris and bacteria while minimizing tissue damage (**Figure 4**). Avoid pressurized lavage on fragile organs. Lavage of abdominal and thoracic cavities should be with sterile saline alone, but antiseptic solutions (not scrubs) can be used in subcutaneous tissues and muscle. Appropriate concentrations are 0.05% chlorhexidine solution (e.g., 25 mL of 2% chlorhexidine + 975 mL diluent) or 0.1%-1% povidone-iodine solution (e.g., 10 mL of 10% P-I + 990 mL diluent for 0.1%; 100 mL of 10% P-I + 900 mL diluent for 1%).

The debrided wound is left open and managed with moist wound healing (10) and serial debridement and lavage as needed. The wound is closed once the veterinarian is confident it is free of contaminants, necrotic tissue, and unhealthy tissue that might necrose later. If



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Figure 3. A four-year-old Yorkshire Terrier bitten over the cranial thorax by another dog. (a) Hemostats were easily passed from one bite wound to the next due to the disruption of underlying tissue. The skin overlying the hemostats was incised along the dotted line. (b) Unhealthy tissue and a deep tract (probed with an instrument) were present below the incision. (c) As the tract was opened up, additional tissue damage and multiple other puncture tracts (circled) were found. These tracts were followed, unhealthy tissue was debrided, and the surgery site closed over a suction drain after copious lavage. (d) In this photo of a different dog, unhealthy muscle is being excised using a similar technique.

a wound must be closed before that point, a drain (preferably a closed, active suction drain) should be placed and covered with a bandage (11). Post-operative care also includes fluid support as needed, analgesics, and good nutrition with a recovery diet to support the healing process. In highly compromised patients, consider placing a feeding tube during anesthesia to ensure adequate nutrition during recovery.

More conservative debridement and lavage may be considered for superficial and/or low severity non-abdominal penetrating injuries (12,13). For example, damage caused by a single, non-tumbling, non-deforming bullet passing only through skin and muscle may be limited to the permanent cavity since these elastic tissues can handle a lot of cavitation energy. A similar effect may be created by penetration with a sharp, smooth, clean foreign body.

■ Wounds to the abdominal or thoracic cavity

Without surgery It can be difficult to determine if penetration of a body cavity has occurred. Penetrating wounds can be probed to assess their extent, but they may not follow a straight path and thus the probe may not be able to follow the tract to its end. Abdomino- or thoracocentesis may reveal air, blood, urine, bile, ingesta, or purulence indicative of body cavity penetration, but a negative tap does not rule this out. Imaging may show free air/fluid, foreign material, or damaged tissues consistent with cavity penetration, but normal images do not rule out internal injury (3,4,7,8,14).

If there is a penetrating abdominal wound (or suspicion thereof) or significant abdominal crush injury, exploratory celiotomy is indicated at the time of presentation because:

Table 1. Guidelines for debridement of tissue with uncertain viability*.

“When in doubt, cut it out” if:	“When in doubt, leave it in” if:
removal is compatible with life	removal is incompatible with life
And	Or
there is only one opportunity to access and assess the tissue	there will be multiple opportunities to access and assess the tissue
And/or	And
there is plenty of residual tissue so it will not be missed	the tissue will be valuable for later wound closure
Examples – damaged muscle deep in a wound; damaged spleen, jejunum, liver lobe, or lung lobe	Examples – damage to the one working kidney; damaged skin on a distal limb, where there is limited skin available for repair

*Uncertain viability *i.e.*, it is unclear whether the tissue will survive; it has some signs of viability and some signs that it is dying; clearly necrotic tissue should be removed.

- There is a high risk of intestinal damage
- Untreated intestinal perforation is life-threatening, and clinical signs may not be apparent until there is full-blown septic peritonitis and septicemia
- Normal test results do not exclude internal injury (see above)
- Intestines are constantly moving, so the damage cannot be reliably found just by following the wound tract through the body wall

While this “default celiotomy” approach will result in some negative abdominal explores, the risk-benefit ratio is squarely on the side of surgery even if penetration is unproven (2,5,13,15).

Penetrating wounds over the thorax are opened, debrided, lavaged, and explored as for any wound; this may take the surgeon into the thoracic cavity. However, unlike an abdominal penetration, full exploration of the thoracic cavity is not the default, as:

- The rib cage makes it challenging for penetrating objects that are not correctly aligned to enter the chest
- The elasticity of the lung makes it less susceptible to

- penetration and associated collateral damage
- The lungs are not laden with bacteria

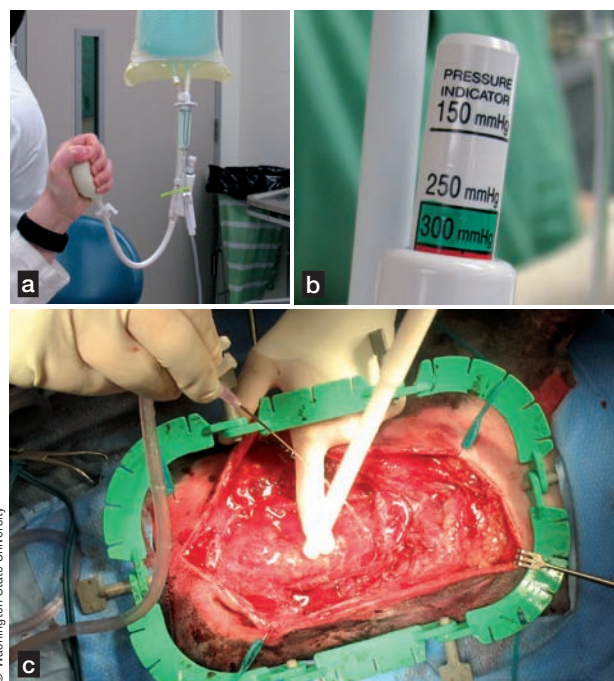
Exploratory thoracotomy is indicated when hemothorax or pneumothorax is not responsive to stabilization measures.

Penetrating wounds into internal organs are debrided and lavaged. The small diameter of intestine makes adequate debridement difficult, so affected areas are treated with resection and anastomosis. Liver lobectomy, splenectomy, and lung lobectomy are usually the most efficacious ways to manage wounds in these tissues. More complex procedures may be required to resect damaged tissue in non-redundant organs.

■ Removal of penetrating objects

There are risks associated with removing a foreign body lodged in tissues; these include bleeding from holes in major vessels previously plugged by the foreign body, additional tissue damage caused by barb-like projections on the foreign body, and/or leaving behind fragments of foreign material (e.g., pieces of bark from a

Figure 4. (a and b) The desired lavage pressure of 7-8 psi is best achieved via a needle (16-22 G) on a standard intravenous drip set attached to a bag of fluids pressurized to 300 mmHg with an emergency pressure sleeve (22). **(c)** The debrided wound from the dog in Figure 2 is held open with a ring retractor (green) for lavage with 0.05% chlorhexidine solution.



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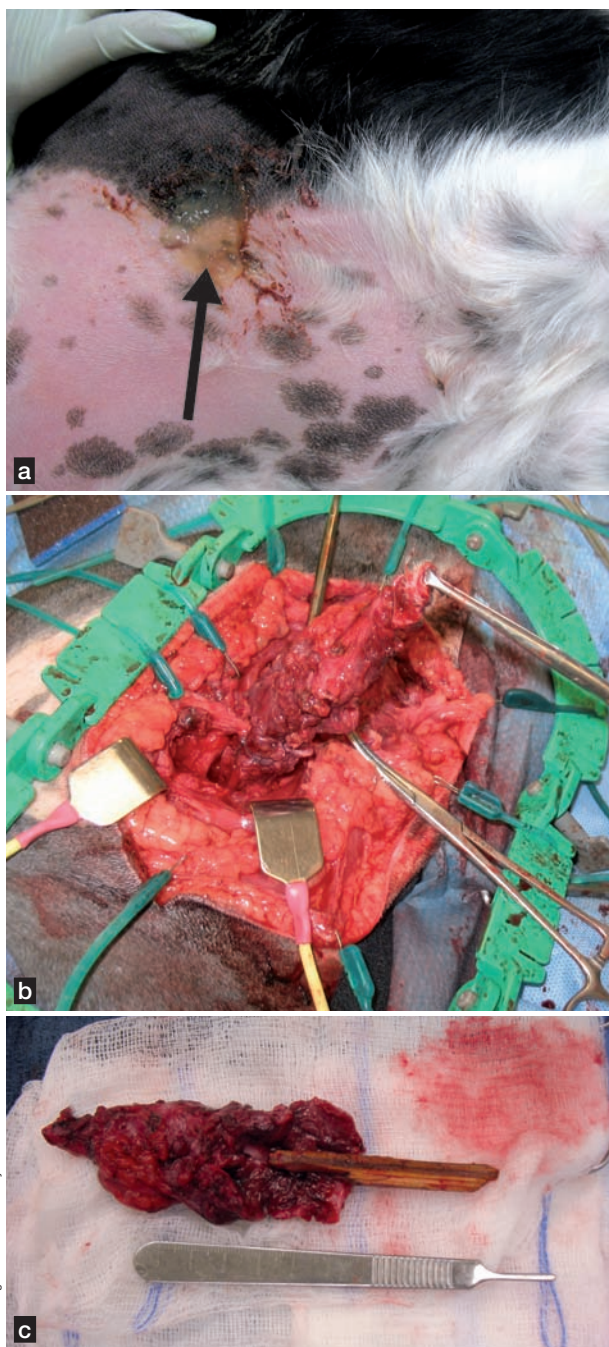
stick). It is therefore best to remove foreign material via a surgical approach in an operating theatre with an anesthetized, fully prepped patient. Because foreign material can migrate due to body motion or gravity, images used to guide surgery should be current.

Retained organic or non-organic material can cause inflammation, infection, and/or chronic draining tracts, so removal is indicated if the reaction is causing significant clinical signs or if vital structures are at risk should the object migrate. The inflammation associated with steel shot (which is 99% iron) tends to be self-limiting after two to eight weeks in dogs, so it may not need to be removed. Lead bullets in soft tissues are typically walled off by fibrous tissue and do not pose a poisoning risk (12,16,17). However, lead in the gastrointestinal tract or in contact with cerebrospinal fluid can cause toxicosis, and lead within a joint can cause destructive synovitis, so removal of bullets in these areas may be prudent (17-19).

Two dissection techniques can be used to remove a penetrating object. In the first, an incision is made alongside the foreign body or down its tract until the material can be removed without resistance. In the second technique, the entire tract and foreign body are removed as a unit, similar to removing a neoplasm with margins (**Figure 5**). This technique maximizes the chance of removing all foreign material and associated unhealthy or contaminated tissue. After removing the foreign body with either technique, surrounding tissue is debrided further as needed, lavaged, and the tract is left open to heal on its own or closed over a drain (11).

■ Use of antibiotics

The question can be asked: are antibiotics indicated for all penetrating wounds? Such wounds are contaminated with bacteria and debris, and the risk of infection increases with the amount of tissue damage and vascular compromise. While antibiotics are typically given during surgery, proper debridement and lavage are pivotal to minimizing the risk of contamination turning into infection; antibiotics do not replace the need for local wound care (3,20)! Antibiotics can be stopped post-operatively for shallow, minimally contaminated wounds surgically converted to clean (3,19). Post-operative antibiotics are clearly indicated in patients with extensive tissue damage, open joint or fracture, sheared bone, SIRS, immunocompromise, and actual infection (1,2,19,21). In between these two groups, the decision is less clear cut and must be tailored to the individual,



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Figure 5. (a) A 4-year-old castrated male Border Collie crossbreed presented for a recurrent draining tract cranial to the left shoulder that had been treated with antibiotics, surgical exploration, and drainage. Five months earlier, the dog had surgery for a hard palate injury from playing with a stick. Serial radiographs around the draining tract had not shown any foreign body, but the stick, which must have entered the neck after penetrating the oropharynx, was seen on MRI. **(b)** The stick and surrounding tract were dissected out *en bloc* from the neck. **(c)** The stick can be seen emerging from the excised tract after removal.

factoring in the need to avoid unnecessary use of antibiotics due to multi-resistant bacteria. For patients with infected wounds, antibiotic choice is ultimately based on aerobic and anaerobic cultures. Culture of a piece of tissue cut from deep in the wound is the most reliable, followed by culture of purulent material; culture of the wound surface is least desirable due to surface contaminants.

■ Conclusion

Recognition of the iceberg effect is important for thorough treatment of penetrating wounds. Early, pre-emptive debridement and lavage of penetrating wounds prevents the development of SIRS or sepsis several days after injury. If penetration of the abdominal cavity cannot be ruled out, the abdomen should be explored due to the high risk for intestinal perforation.

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Gastric dilatation and volvulus



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■ Introduction

Gastric dilatation and volvulus (GDV) is a life-threatening condition which requires prompt diagnosis and treatment. Although cases have been reported in cats it is more commonly dogs that present with the condition, which involves gaseous distension of the stomach in association with rotation along the longitudinal axis. Historically the mortality rate from the condition has been regarded as

high; different studies put it between 15%-68% (1-5), but with improved understanding of the condition it is hoped that morbidity and mortality will be reduced in future.

■ Predisposing factors

Predisposing factors for GDV include genetic and environmental factors. The condition is most commonly seen in large or giant breeds of dogs, but it can and does occur in smaller breeds. There is a recognized breed predisposition to GDV; at-risk animals include the Great Dane, German Shepherd, Standard Poodle and Irish Setter, although this is by no means an exhaustive list. One study suggested the condition was most prevalent in the Grand Bleu de Gascogne (6), although this is not a breed commonly encountered in practice. The risk of developing the condition increases with age, regardless of breed; one study (which focused on Irish Setters) found the risk was elevated by 33% with each year of age (7). It has been suggested that stretching of the hepato-gastric ligament allows greater stomach mobility in older dogs (8,9). Any dog with an increased thoracic depth-to-width ratio is at greater risk of GDV; this may be due to the different anatomical relationship between the stomach and the esophagus in these animals, which may restrict their ability to remove air from the stomach (7). An anxious temperament has also been perceived as a predisposing factor, and is commonly reported by owners of dogs who have developed GDV (8), whilst animals described by their owners as “happy” seem less

KEY POINTS

- Gastric dilatation and volvulus is a life-threatening condition; prompt diagnosis and treatment are vital to reduce morbidity and mortality.
- The pathophysiology of gastric dilatation is a complex subject and incompletely elucidated.
- Gastric decompression is of the utmost importance when treating this condition, either by placement of a stomach tube or by percutaneous trocharization.
- Serum lactate levels are a useful indicator of response to treatment; it is thought to be a positive indicator of survival if the level decreases by > 50% within the first twelve hours of therapy.

likely to develop the condition (7). Any dog that has a “first degree” relative (e.g., parents, offspring or siblings) with a history of GDV is also considered to be at higher risk (7). All of the aforementioned points are likely to indicate genetic factors which predispose to the condition.

Risk of GDV has been linked to a number of environmental factors including dietary factors such as feeding one large meal a day, feeding from a height, and various ingredients within a diet, but cause-and-effect relationships have not been fully elucidated (10).

An increased risk of GDV following splenectomy has been described (11). The study concerned does not suggest an association between the cause of splenectomy and GDV; rather it is postulated that removal of the spleen increases space within the abdomen, which may allow the stomach to have more mobility than previously and hence an increased risk of GDV (12). Splenic torsion is a complication that can be encountered in patients presenting with GDV, likely to be caused by the spleen being pulled with the stomach as it rotates.

■ Pathophysiology of GDV

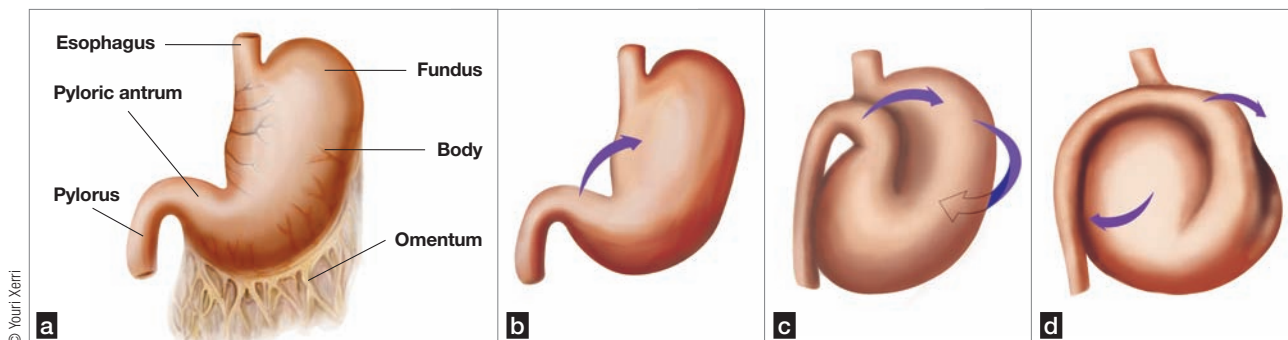
The pathophysiology of gastric dilatation is a complex subject and incompletely elucidated. It is not known if dilatation occurs prior to or after volvulus, as dilatation can occur without the presence of torsion of the stomach. One study which analyzed the components of stomach gas in dogs presenting with GDV suggested that the gas was produced by bacterial fermentation and was not related to aerophagia as previously accepted (13). However, there is still a degree of uncertainty regarding aerophagia and gas build-up in GDV, and it is possible that both may be present simultaneously. It

remains a common belief among owners that “gulping air” predisposes to GDV, and many people feed their dog from a height for this reason. However, the raising of food bowls has been suggested to increase the risk of GDV in larger breeds of dogs (14).

In the normal dog the pylorus is located cranially on the right-hand side of the abdomen. In GDV the pylorus and duodenum move ventral to the stomach, towards the left-hand side of the abdomen; the pylorus then becomes displaced dorsally to sit above the cardia of the stomach (**Figure 1**). Increasing gastric distension is the precipitating factor for several complications of GDV. Distension leads to increased intragastric pressure, which in turn places pressure on the smaller blood vessels within the stomach wall, compressing them and decreasing perfusion to the stomach tissues, which can lead to the development of necrotic areas. As the duodenum becomes effectively trapped between the distended stomach and the left body wall, pyloric function is affected and duodenal obstruction can occur. However, if obstruction is present, an underlying disease should also be considered, as outflow obstruction may also arise from a foreign body, intestinal ileus, or a dysfunctional pyloric outflow.

Dogs with GDV often present in shock and can experience more than one type of shock simultaneously, e.g., obstructive, distributive, cardiogenic and hypovolemic. The distended stomach can obstruct blood flow within the caudal vena cava, resulting in reduced blood volume returning to the heart. This results in a reduced preload and stroke volume, which subsequently affects cardiac output; this is often described as obstructive shock. Patients will frequently be tachycardic on admission, a compensatory response in an attempt to maintain cardiac

Figure 1. This diagram illustrates the movement of the pylorus during GDV. In the normal dog the pylorus is located cranially on the right-hand side of the abdomen (a); with a GDV it moves underneath the stomach (b) and is then displaced dorsally to lie in the left cranial abdomen (c). The fundus moves ventrally to sit in the ventral abdomen (d).



output. Stress and pain should also be considered as factors in animals that present with tachycardia (15).

An increase in venous pressure within the abdomen results in sequestration of blood in the splanchnic and portal veins. This, together with the up-regulation of inducible nitrous oxide synthase (iNOS) and the release of vasoactive cytokines as a result of the gastric inflammation, leads to peripheral vasodilation which can cause further pooling of blood, worsening the condition (15). These factors combine to cause mal-distributive shock.

Hypovolemic shock is not a major contributor in these patients, although as noted splenic torsion can occur with GDV, and if there is tearing of the short gastric arteries and veins hemoabdomen can result. A decrease in oral fluid intake can contribute to dehydration in these patients, but is unlikely to be substantial enough to affect blood pressure (15).

Cardiac compromise is closely connected with morbidity and mortality in dogs. Myocardial ischemia can develop as a consequence of global hypoperfusion; in addition some of the pro-inflammatory cytokines released as a result of the condition have a direct depressant effect upon the myocardium. ECG monitoring can give some indication of the presence of damage to the myocardium (16), as changes in electrical activity may be identified. Monitoring serum troponin levels can allow assessment of heart damage; increases in this biomarker indicate a worsening prognosis, but if the sample has to be sent to an external laboratory for analysis this limits its usefulness.

It has been suggested that cardiac arrhythmia and a degree of myocardial dysfunction is seen in 40% of GDV patients (1,2,15). Life-threatening arrhythmias do not necessarily present prior to surgical correction of the condition; they can arise up to 72 hours later. One study suggested that dogs diagnosed with a cardiac arrhythmia prior to surgery had a 25-38% increase in mortality rate (2). The presence of an arrhythmia is not necessarily a deciding factor on whether the patient should have surgical intervention or euthanasia, but it is a factor to be considered alongside a full clinical examination and history to enable a more accurate prognosis for the patient.

Patients may show signs of respiratory distress, such as increased respiratory rate and effort. This may be as a result of increased stomach size. Normally, the diaphragm moves caudally during inspiration, but this is prevented if the stomach is enlarged (17) and the

decrease in intrathoracic volume can reduce tidal volume and lead to a ventilation/perfusion mismatch. Aspiration pneumonia is also a risk; this can be present prior to surgery or develop as a post-operative complication, and can be associated with a less favorable outcome. If pulmonary function is severely affected by either of these problems then hypoxemia can result, to the further detriment of the patient.

■ Diagnosis

Diagnosis is generally based on signalment, history and clinical examination. Owners commonly report unproductive retching and hypersalivation, although abdominal distension and collapse can be the first signs noticed by an owner. On clinical examination there may be evidence of hypoperfusion: tachycardia, weak peripheral pulses, and pale mucous membranes with a capillary refill time (CRT) greater than two seconds. However, as the patient may be displaying signs indicative of distributive shock, the mucous membranes may be injected with a rapid CRT. Abdominal distension may be obvious on visual assessment and tympany may be noted on abdominal percussion. However, in deep-chested dogs it can be difficult at times to discern if the stomach is tympanitic, as it may lie within the costal arch (**Figure 2**).

Diagnostic imaging will confirm if torsion is present or if the patient has gastric dilatation alone; this is important as dilatation may not require immediate surgery. A single right lateral abdominal radiograph is usually diagnostic; with classic GDV there will be two gas-filled structures visible in the cranial abdomen (**Figure 3**). The larger one is the gastric fundus, with the pylorus appearing as a smaller gas-filled structure dorsally. The two gas-filled areas may be separated by a band of soft tissue. Thoracic radiographs are also useful as they may give an early indication of aspiration pneumonia and allow prompt use of antimicrobials (18).

■ Initial therapy

It is important to record the initial clinical findings to assess response to treatment: heart rate, respiratory rate, pulse quality, mucous membrane color, capillary refill time and, if possible, blood pressure. A large-bore intravenous catheter should be placed (normally into the cephalic vein) as soon as possible and fluid therapy commenced. In large dogs it can be difficult to give an adequate fluid volume rapidly enough to increase preload with one catheter, therefore placing two catheters, one in each cephalic vein, can be beneficial. The rate of the fluid therapy will depend on the clinical findings and



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Figure 2. A Great Dane on initial presentation with a confirmed GDV. Note that abdominal distension cannot be visualized in this patient due to the deep-chested anatomy of the breed.



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Figure 3. A right lateral abdominal radiograph confirming GDV. The pylorus is the smaller gas-filled structure (a) with a band of soft tissue (b) between that and the fundus of the stomach. Here a soft tissue band does not completely separate the two gas-filled structures.

any other underlying health conditions, but in general crystalloids at 90 mL/kg/hour (shock rate) is recommended, with vital parameters assessed every 15 minutes. However it can be more appropriate to administer smaller volumes as a bolus (e.g., 20 mL/kg of fluid given over 15 minutes) with frequent reassessment, adjusting the dose as necessary. A full opioid agonist (e.g., methadone at 0.2-0.3 mg/kg IV) should be given as early as possible (19).

Rapid gastric decompression is of the utmost importance because the risks of hypoxemia are so great. Placement of a stomach tube or decompression by percutaneous trocharization are both suitable options. One study comparing the two techniques concluded that neither had a high complication rate and both were found generally to be successful (20). Surgical intervention should proceed once the patient is stable, which will be determined by clinical examination, such as a resolution of tachycardia and normalization of cardiovascular parameters.

■ Laboratory findings

Baseline bloodwork should be acquired as soon as possible: a minimum database should include packed cell volume, total solids, blood urea nitrogen (BUN), blood glucose and a blood smear. A serum lactate level, electrolytes and blood gases can also be useful. Hematology and biochemistry may also be run at this time, but they are unlikely to change the emergency treatment required. They can however provide a baseline; if there are abnormal findings the values should be reassessed as appropriate.

Measuring serum lactate is a useful indicator of response to treatment, but care must be taken when interpreting the initial lactate level. An increased serum lactate has been shown not to be indicative of gastric necrosis or prognosis, but if the level decreases by at least 50% in the first twelve hours of therapy it is thought to be a positive indicator for survival (21).

Increased levels of BUN and creatinine may indicate renal impairment, but it is hard to distinguish between pre-renal and renal azotemia at this time. Any increase in these parameters should be rechecked after hypoperfusion is corrected, as acute kidney injury can occur with GDV. One explanation is that a reduction in circulating volume leads to decreased perfusion and tissue ischemia; once perfusion is restored, reactive oxygen and nitrogen species are generated, leading to ischemia-reperfusion injury (IRI). The risk of renal injury is a compelling reason to avoid the use of COX-1 or COX-2 inhibitors during GDV treatment.

Electrolyte abnormalities, most commonly hypokalemia and hypochloremia, can also be detected at this stage. Acid-base changes are often complex; the presence of electrolyte and perfusion abnormalities usually creates a moderate to severe metabolic acidosis, and the animal's ability to compensate by generating a respiratory alkalosis through hyperventilation is often hampered by the effect of gastric distension. This can result in a profound mixed respiratory and metabolic acidosis. Employment of the previously mentioned therapies, gastric

trocharization and aggressive intravenous fluid therapy, should help alleviate these problems.

■ Surgical procedure

Premedication and anesthetic induction agent are at the discretion of the clinician. Generally methadone should be adequate for initial pain relief. Anesthesia should be maintained with either isoflurane or sevoflurane; nitrous oxide should not be given to a GDV patient, as it will accumulate in gas-filled spaces and can therefore worsen the gastric dilatation. Since the dog may regurgitate when the stomach position is corrected, it can be useful to have suction equipment ready for use. Given that gastro-esophageal reflux is a known risk factor for esophagitis and esophageal stricture formation post-operatively, the use of omeprazole (1 mg/kg IV) prior to induction may reduce the risk of this occurring (17).

The aim of surgery is to de-rotate the stomach. The torsion is typically clockwise (when viewed from behind the animal) and on incision the omentum will usually be covering the stomach. Gaseous distension noted at this point can be easily drained using a needle or cannula attached to the surgical suction tubing. The surgeon should locate the pylorus (often found dorsally on the left-hand side of the abdomen), and grasp it with one hand whilst the other hand holds the fundus of the stomach. The fundus should then be pushed towards the operating table whilst the pylorus is manipulated up towards the incision and over to the right side of the abdomen. This simultaneous pushing and pulling should correct the stomach position (**Figure 4**).

After correcting the stomach position it should be fixed to the body wall to prevent recurrence. Various gastropexy techniques have been described; the most frequently used is known as “incisional gastropexy”. With this technique an incision is made in the seromuscular layer of the gastric wall, parallel to the long axis of the stomach, at the level of the pyloric antrum. Another incision of the same length is made in the right transversus abdominus muscle and the incisions are sutured together using an absorbable monofilament suture, with the aim of forming an adhesion which prevents repeat torsion in future (22).

■ Complications that affect prognosis

At surgery the stomach should be assessed for color, wall thickness and blood supply, and the viability of the tissue assessed; any areas of necrosis or impaired viability



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Figure 4. The surgeon can be seen correcting the gastric torsion.

should be resected. The spleen should also be examined; if the splenic artery or vein are damaged, or there is active bleeding, a splenectomy should be performed. Post-operative mortality is significantly increased in patients that require gastric resection or splenectomy; one study found that partial gastrectomy was not associated with an increased risk of death, but the risk of post-operative complications was increased (3). In patients where there is marked gastric necrosis, such that resection is not possible, euthanasia will be required. It should be emphasized that euthanasia may be the preferred option for some patients, either because of the financial outlay involved or because there is severe concurrent disease. The clinician should discuss fully the risks, costs and potential outcomes with the owner before embarking on surgery.

■ Post-operative care

Patients which are severely hypoperfused prior to anesthesia may be slow to recover from surgery and can require intensive supportive care. Once perfusion is restored patients can develop post-operative complications related to IRI. This is associated with increased mortality, as myocardial damage can result in the development of arrhythmias. ECG monitoring should be continued in the post-operative period as cardiac arrhythmias are common following GDV; the most frequent are ventricular in source, although supra-ventricular arrhythmias can also occur. If these are considered significant, the treatment of choice is lidocaine administered as a bolus of 2 mg/kg (repeated up to a total of 8 mg/kg) followed by a continuous rate infusion (at 25-75 µg/kg/min). At least one study has evaluated the pre-emptive use of lidocaine treatment for dogs with GDV (23); given as a bolus prior to decompression and initiation of fluid

therapy, lidocaine reduced damage from IRI, lessened the risks of complications developing, and resulted in a decreased mortality rate.

In patients requiring extensive gastric resection placement of an esophageal feeding tube may be of benefit. Nausea post-operatively can be treated with medication such as maropitant. Pain relief will be dependent on the individual patient; opioids such as methadone can be given in the initial post-operative phase, with transition to buprenorphine when possible. NSAIDs should be avoided due to the risks of gastric wall compromise and renal dysfunction. Fluid therapy should be continued until the patient is eating and drinking.

Conclusion

Post-operatively, it is important the owners understand that whilst gastropexy reduces the risks of repeat volvulus it does not negate the risks altogether. Patients may

develop gastric distension without volvulus following gastropexy, and some animals may require medication to manage this. Treatment with metoclopramide may be beneficial in these patients, however the evidence for this is equivocal at present. Note that whilst the risk of a repeated episode of GDV in dogs following gastropexy has been reported as less than 5% (5), dogs that have had a GDV surgically corrected without a gastropexy being performed have a recurrence risk of up to 80% (24).

Long term, any predisposing environmental factors (e.g., feeding one large meal per day) should be addressed to limit recurrence, but as the condition can be multifactorial it may not be possible to eliminate all predisposing factors, and assessment should be on a case-by-case basis. Finally, dogs with a high, breed-related risk of developing GDV may benefit from prophylactic gastropexy, which can be performed laparoscopically or via celiotomy.

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Thoracic trauma



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KEY POINTS

- Thoracic trauma cases are frequently seen in small animal clinics and should always be treated as an emergency.
- Pneumothorax must be considered a possibility for all thoracic trauma patients until proven otherwise.
- Bilateral thoracocentesis can be both diagnostic and therapeutic in pneumothorax cases, and must be carried out before radiography is attempted.
- Hemothorax is rare in animals, but significant blood loss into the pleural cavity can occur following trauma.
- Most cases of hemothorax secondary to closed trauma do not need surgery and can be treated conservatively.
- Bites to the thorax can be very problematic; even if there is no skin perforation, some bites can cause massive internal damage. Surgical exploration of all bite wounds is mandatory.
- The intense pain from fractured ribs, along with any lung damage, contributes to hypoventilation; good pain management is vital in these patients.

■ Introduction

Animals with thoracic trauma frequently present in small animal clinics and should always be treated as an emergency. Trauma is often as a result of traffic accidents (11-40% of cases (1)) and many patients will also have fractures (20-60% of cases (2,3)). Other than traffic accidents and other blunt trauma such as kicks, the most common etiologies are animal bites and penetrating injuries from impaling, knives, firearms, etc. However many patients with thoracic injuries may not show signs and/or lesions at the time of admission, although signs can appear and progress 24-48 hours following the incident. Thoracic trauma can be classified as being open (e.g., from knives, bites or bullets (**Figure 1**)) or closed (e.g., following falls, traffic accidents) in nature.

In the event of severe or multiple injuries, it may be necessary to obtain the full medical history after or whilst the patient is being stabilized. A detailed history, including the time between the incident and presentation at the clinic, can offer important information. A complete and exhaustive physical examination is essential, paying special attention to the respiratory and cardiovascular systems. A detailed inspection of the thorax is mandatory, and it should be palpated, percussed and auscultated; if necessary the entire area should be shaved — especially when dealing with open injuries (**Figure 2**). The mucous membranes, capillary refill time, arterial pressure and mental state should all be assessed, and any neurologic/posture alterations noted. Initial supportive therapy should follow the ABC rule:

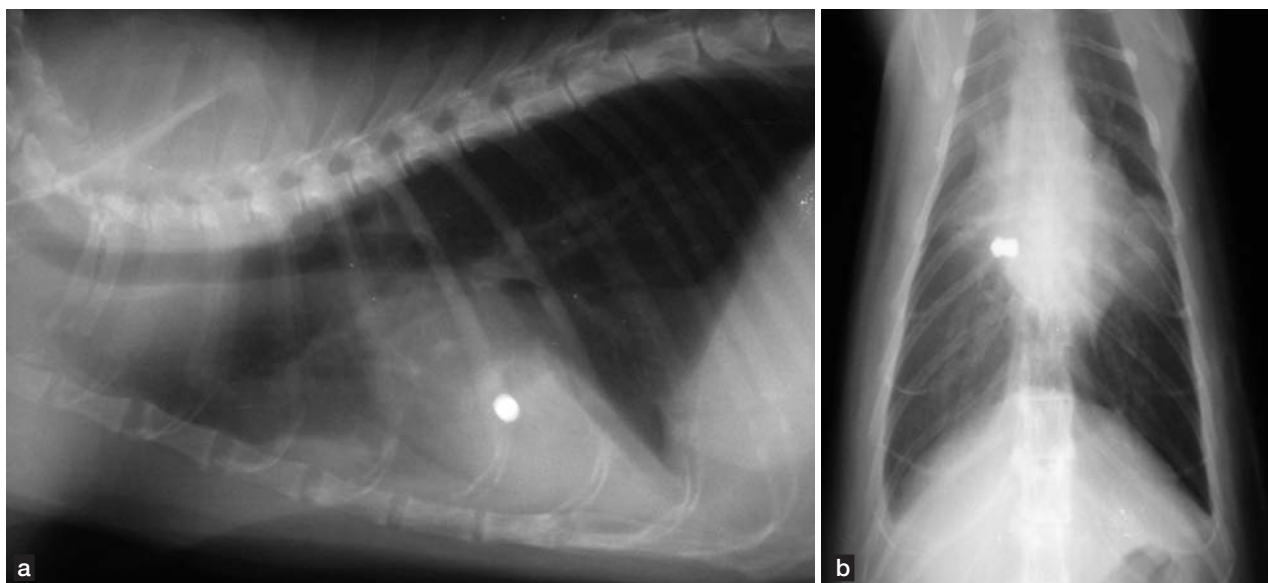


Figure 1. Lateral (a) and ventro-dorsal (b) radiographs illustrating a lead buckshot pellet in the myocardium of a cat.

- Airway maintenance and oxygen therapy
- Breathing (respiratory/thoracic wall) support
- Cardiovascular and circulatory support

Hypoxia and hemorrhage are two of the main causes of death in the polytraumatized patient. If a patient presents in shock and there is no evidence of external bleeding, internal hemorrhage should be considered, and the abdomen and thorax should be thoroughly checked (4).

Figure 2. A Yorkshire Terrier with multiple thoracic injuries following a fight with another dog. The patient has been stabilized and the thorax shaved to allow cleaning and assessment of the lesions. Prompt and correct action by the veterinarian resulted in a full recovery.



■ Initial handling and stabilization

This article will focus on traumatic pneumothorax, hemothorax and lesions of the chest wall, but five key points to consider when initially assessing any patient subjected to thoracic trauma are as follows:

1. Oxygen supplementation: this requires careful handling to minimize stress, and can be done either by mask (only on initial admission), chamber or nasal catheter.
2. Re-establish negative intrathoracic pressure: if an animal is dyspneic, bilateral thoracocentesis should be performed and any free air or fluid removed. Chest radiographs should then be obtained. If there is a penetrating wound the area should be shaved, cleaned and protected with a padded, non-compressive, hermetic dressing.
3. Hemodynamic stabilization: one (or two) intravenous catheters should be placed, a blood sample taken for analysis, and fluid therapy commenced. If necessary (e.g., severe hypotension) fluids should be administered by the intra-osseous route.
4. Multimodal pain management: pain control is very important and opioids are often first-choice on admission; a continuous IV infusion of morphine, lidocaine and ketamine (MLK) can also be very effective.
5. Broad-spectrum antibiotic therapy: drugs such as cefazolin or potentiated amoxicillin should be given, preferably by the intravenous route for open trauma (5).

Surgery should normally only be performed when the animal is stable (or as stable as possible). The most common indications for surgical intervention include the following (6-9):

- All penetrating lesions to the thorax
- Progressive emphysema of the neck and thorax
- If there is internal organ damage or uncontrollable hemorrhage
- Progressive pneumothorax which cannot be controlled with thoracocentesis or thoracic drain
- Pulmonary contusions that worsen despite treatment/mechanical ventilation
- If there is communication between the pleural cavity and the peritoneal cavity

With respect to the last point, it can be said that diaphragmatic rupture usually results from abdominal, rather than thoracic, trauma; although they can undoubtedly cause significant secondary thoracic pathology, the treatment of diaphragmatic hernias is outwith the scope of this paper.

■ Traumatic pneumothorax

Pneumothorax can be classified as open or closed (**Figure 3**) (8,10,11). An open pneumothorax is a lesion in the chest wall that allows communication between the pleural cavity and the environment. A closed pneumothorax occurs when there is air within the pleural cavity from a pulmonary or mediastinal lesion but there is no communication with the outside. In some cases the lesion may act as a unidirectional

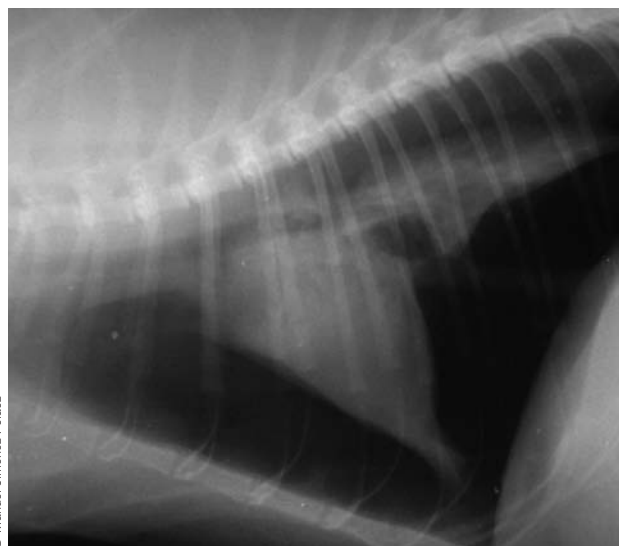
valve, so that air enters the pleural cavity but cannot leave, creating a tension pneumothorax. In all cases the accumulation of air increases pressure within the pleural cavity, limiting lung expansion and venous return, severely compromising both respiratory and cardiovascular systems (6,7). Affected animals present with a superficial, restrictive respiratory pattern and may be dyspneic.

For all trauma patients, pneumothorax must be considered a possibility until proven otherwise (6). Bilateral thoracocentesis – usually best done with the animal in sternal recumbency – can be both diagnostic and therapeutic, and must be carried out before radiography (**Figure 4**). It is better to have a negative thoracocentesis than a dead animal on the X-ray table.

A closed pneumothorax generally does not require surgical intervention; such cases are often self-limiting and can be managed with thoracocentesis repeated as necessary – clinical evaluation of the patient should guide treatment (6,7). However, a drain tube should be considered if signs persist and the pneumothorax recurs despite repeated thoracocenteses (*e.g.*, more than 2-3 times daily and/or for more than two days) or if excessive fluid (> 2 mL/kg/day) is present.

When a pneumothorax requires surgical intervention, the surgical approach depends on the location of the lesion. If unilateral, a lateral thoracotomy offers the best approach. If bilateral, or the exact location of the

Figure 3. Lateral thoracic radiograph of a cat with a severe pneumothorax following a fall from a height.

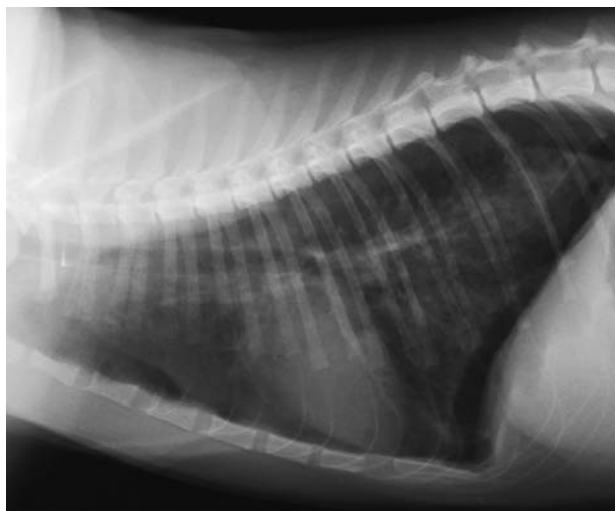


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Figure 4. Thoracocentesis using a butterfly needle and a three-way tap on a dog which had been hit by a vehicle.



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Figure 5. Lateral thoracic radiograph of a cat with a pneumothorax and pulmonary contusions following a fall.



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Figure 6. Drainage of a traumatic hemothorax using a large-caliber catheter and a three-way tap.

lesion is unknown, a medial sternotomy is required (7). Note that pulmonary contusions (**Figure 5**) and mediastinal bleeding are also seen frequently after closed thoracic trauma, whether concomitant with pneumothorax or not.

■ Traumatic hemothorax

Hemothorax is rare in animals (in contrast to the human situation) but significant blood loss into the pleural cavity can occur following trauma. Blood may be lost from damaged lung tissue or from laceration of the large pulmonary vessels, intercostal vessels or internal thoracic arteries. Thoracocentesis serves as both a diagnostic and therapeutic procedure, although ultrasound can also be useful to evaluate the amount of blood present, and repeat scans allow reassessment as required. If there is a considerable volume of blood, fluid therapy (crystalloids, colloids and blood) should be given (6).

Treatment of a traumatic hemothorax will depend on several factors, including the amount of blood present and the rate of blood loss into the pleural cavity, the type of trauma (open or closed), and the stability of the patient. Most cases secondary to closed trauma do not need surgery. A minor hemothorax with minimal respiratory distress should be treated conservatively, although the free blood should be removed if the animal develops labored breathing. When undertaking drainage of a hemothorax, it is not necessary to drain the thorax entirely, but sufficient blood should be removed to stabilize the patient; the drainage procedure should be done slowly

whilst carefully monitoring the patient. Thoracocentesis may need to be repeated as necessary (**Figure 6**). If hemorrhage persists, or there has been severe blood loss into the pleural cavity, the patient may require a blood transfusion in addition to conventional fluid therapy. Autotransfusion is a quick and readily available method, but the blood must be collected aseptically and filtered blood bags should be used. If necessary consider placing an indwelling drain tube, and in extreme cases (e.g., if the bleeding does not resolve), exploratory thoracotomy may be necessary. However these cases have an increased risk of mortality. Remember that all penetrating thoracic lesions must be surgically explored, whether a hemothorax is present or not.

■ Thoracic trauma

Closed trauma

For closed trauma cases, opinions vary as to which cases require exploratory surgery (5,6). Some clinicians recommend exploratory surgery for all cases where there are fractured ribs or flail chest, pulmonary contusions or pneumothorax, but the optimal time for surgery on these potentially unstable patients is unknown (12,13). The authors prefer to treat most closed trauma lesions conservatively and in general obtain good results.

An exception to this is thoracic trauma from bites. In some cases these may be considered to be “closed trauma”, as there may be minimal or no perforation of the skin; however, even if there is no visible break in the skin, all cases should be explored surgically, as a bite

can frequently cause severe damage to the underlying tissues, including the intercostal muscles, ribs, intrathoracic blood vessels and internal organs (**Figure 7**).

Penetrating wounds and open trauma

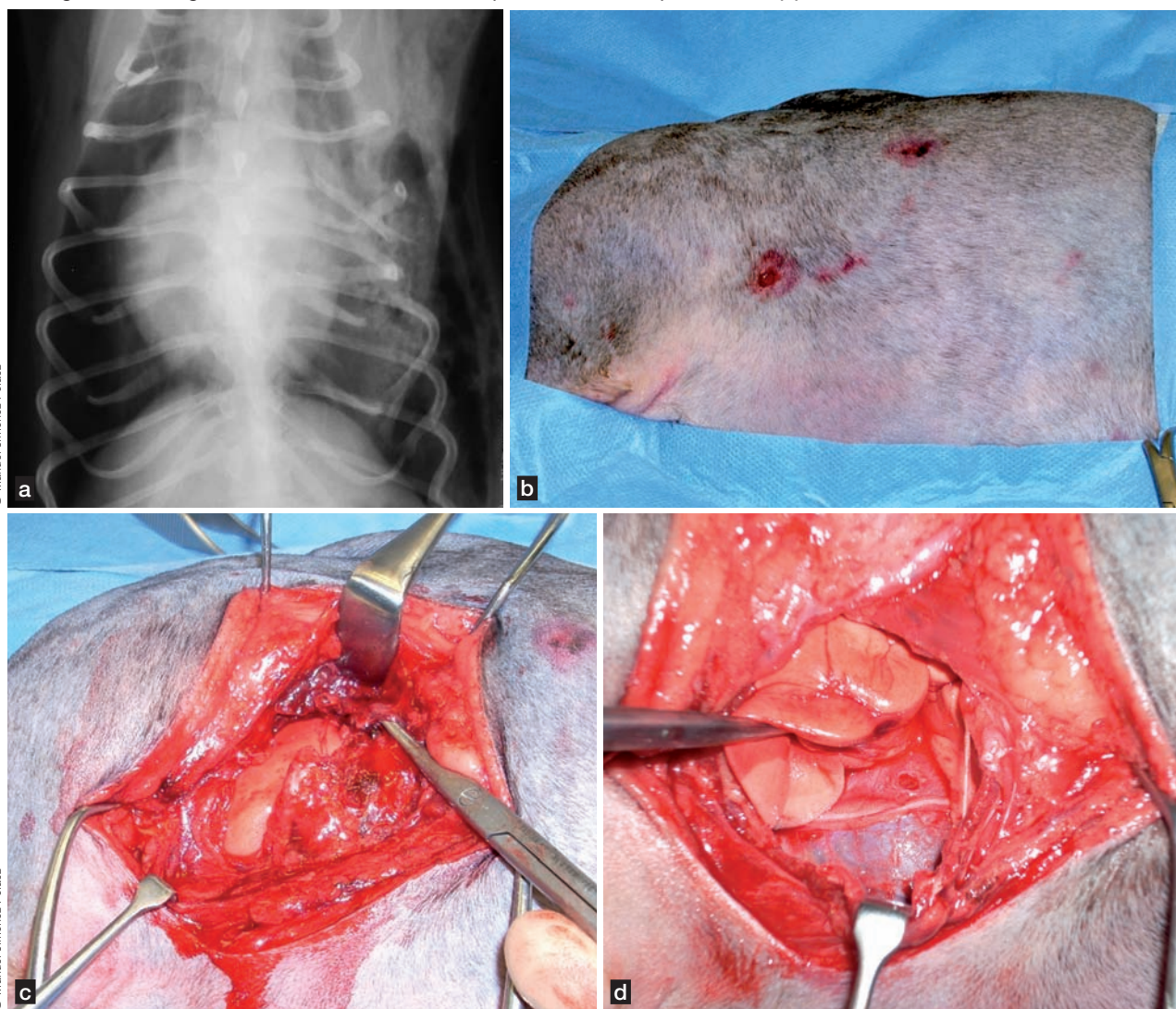
Any penetrating thoracic wound is a surgical emergency and the patient must be moved to the operating theatre as soon as possible. While the patient is being stabilized (oxygen, analgesics, fluids, etc.) the wound should be shaved, washed and covered so that the thorax is airtight and the pneumothorax resolved by thoracocentesis or drain tube (7, 14). In general the degree of damage will be under-estimated on initial examination and even

on radiography, especially when dealing with trauma secondary to bites (12,15); the full extent of the injury is often apparent only at surgery (**Figure 8**).

When dealing with bite wounds, all abnormal and damaged bone and soft tissue must be debrided and the entire area flushed copiously with sterile saline (7). Repair should be with absorbable, monofilament suture material and thoracic and subcutaneous drainage tubes should be placed as necessary (5, 15).

In all cases wound closure must employ healthy, well-vascularized tissue, using muscle and omentum as

Figure 7. A Dachshund with bites to the thorax. Radiography (**a**) showed significant lesions (pneumothorax, rib fractures, subcutaneous emphysema, pulmonary contusion), despite minimal skin damage (**b**). At surgery (**c**) there was severe internal damage with tearing of the intercostal muscles and perforation of the pericardium (**d**).



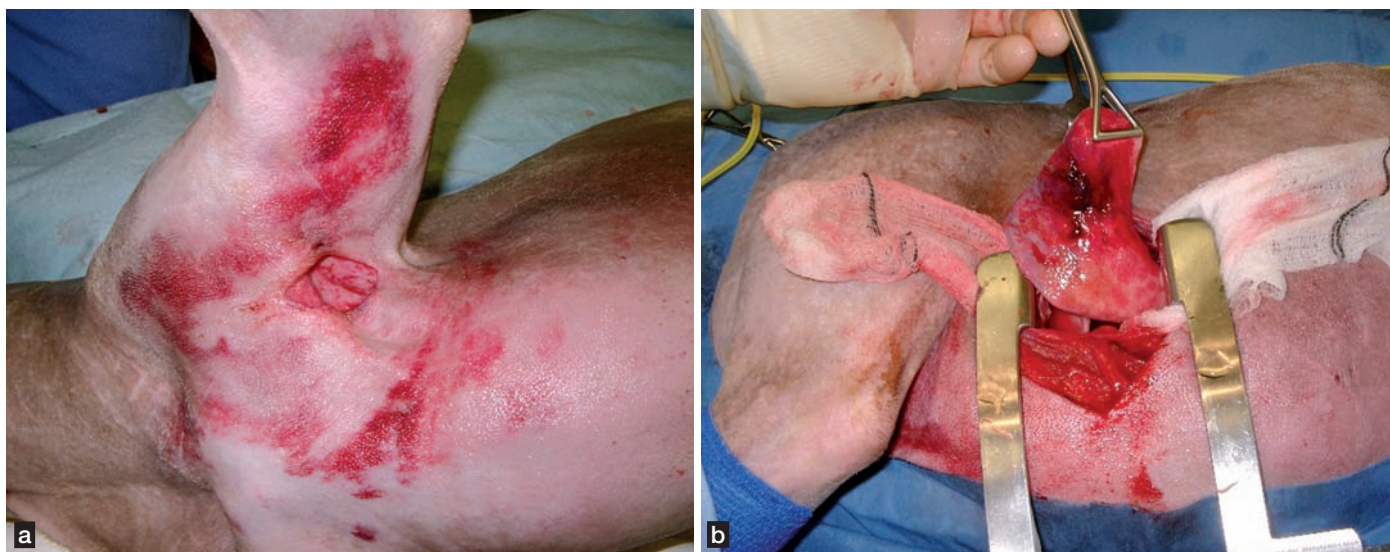


Figure 8. This dog was impaled and presented with an axillary wound (a); however the trauma resulted in penetration of the thorax and a perforated lung lobe had to be removed (b).

necessary. Rarely, large wounds may require reconstruction using synthetic implants; note however that implants are contraindicated if the trauma has been caused by a bite, because of the risk of infection.

If the thoracic wall requires reconstruction, the options depend on the exact anatomical location. The diaphragmatic advancement technique may be useful, transposing healthy local tissue such as the external oblique abdominal muscle and/or *latissimus dorsi* muscle, and the omentum (**Figure 9**). If there is no damage to the pulmonary parenchyma, it is currently unclear if absolute rigidity of the thoracic wall is essential. Skin reconstruction, if necessary, may be achieved using a simple advancement flap, a rotation flap (utilizing the deep (subdermal) plexus) and/or an axial pattern flap (e.g., using the cranial superficial epigastric artery) (7).

Good analgesia for these cases is paramount, and infiltration of the area with local anesthetic (or using a field block around the intercostal muscles) can allow better control of pain and thus improved ventilation.

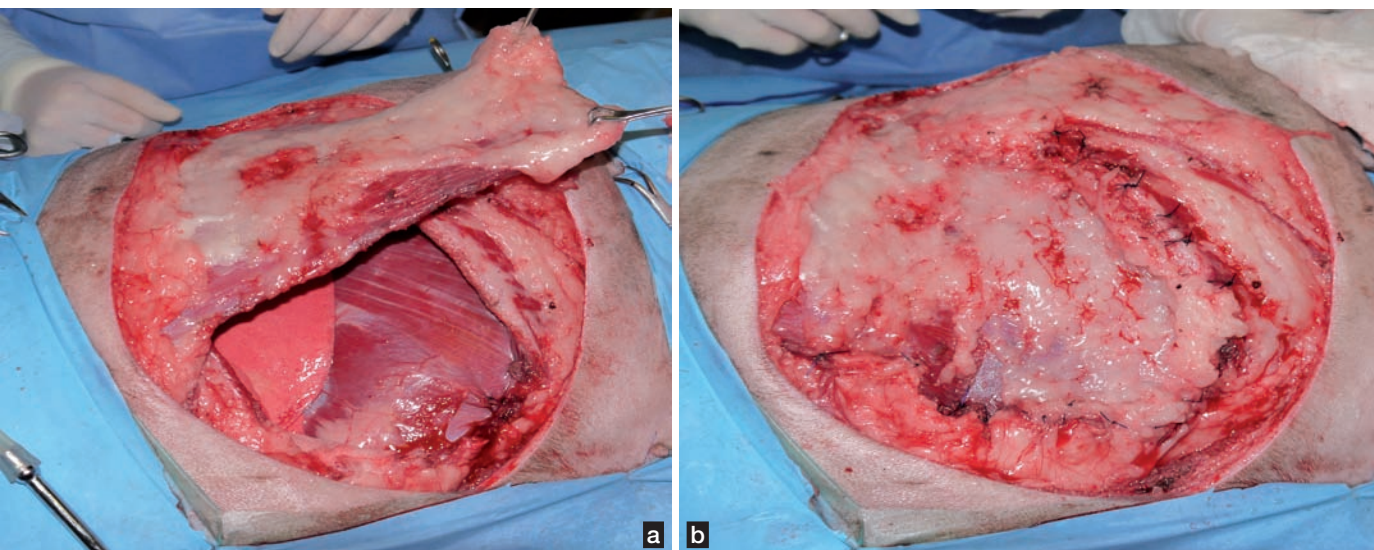
At the end of the surgery, always verify if there is any leakage of air from the thorax by filling the area with sterile saline and gently inflating the lungs (5,15). A thoracotomy tube allows negative pleural pressure to be re-established as necessary and also allows aspiration of any intrapleural fluid, which should always be subjected to cytological evaluation. Any concurrent lesions

(e.g., limb fractures) must be treated in a second surgical intervention once the animal is stable.

Rib fractures

Fractured ribs are intensely painful, which can result in hypoventilation. This may be exacerbated if a broken rib has caused lung damage (16-18). However, simple rib fractures from a closed trauma situation can generally be treated conservatively with pain control. Multiple rib fractures can lead to a flail chest developing; this may be suspected on physical examination if a paradoxical respiratory pattern is noted. For a flail chest to occur, at least two adjacent ribs must be fractured at two levels (ventrally and dorsally); the paradoxical respiratory movement results from the change in intrapleural pressure, such that the damaged section moves inwards during inspiration and outwards on expiration. The combination of abnormal airflow, underlying pulmonary trauma and pain predisposes to hypoxemia and hypoventilation. Stabilization of the fractured ribs is rarely performed but, along with pain relief, may improve ventilation in the polytraumatized patient. A return to full pulmonary function will require further treatment of the other associated pathologies.

Note that pulmonary contusions can be progressive in nature, and in severe cases mechanical ventilation for 24-48 hours, along with supportive medical therapy, may be beneficial until a definitive repair can be effected. If there is extensive damage, a flail chest may be



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Figure 9. Reconstruction of the thoracic wall following a dog bite using a flap of *latissimus dorsi* muscle. **Figure 9a** shows the thoracic wall defect after resection of all necrotic and devitalized tissues. **Figure 9b** shows the thoracic wall defect fully closed using the *latissimus dorsi* muscle flap.

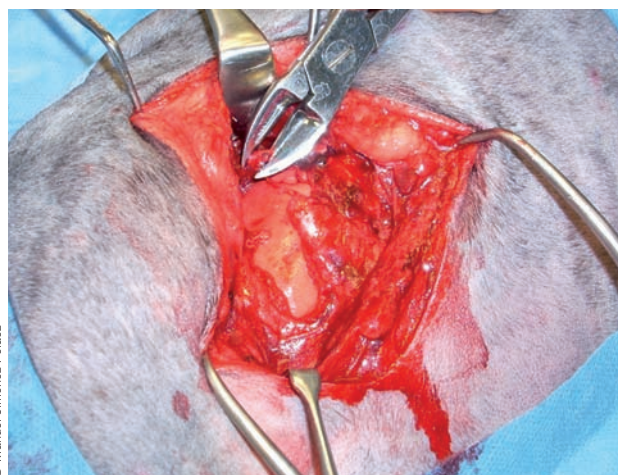
immobilized using percutaneous circumcostal sutures and an external splint, although this is rarely necessary. Otherwise fractured ribs can be stabilized by suturing them to the adjacent ribs; if the damage is severe, or secondary to a bite, it is usually preferable to resect them (**Figure 10**). In general, studies suggest that there is no difference in the prognosis whether or not a flail chest is stabilized surgically or treated medically (13), and the majority of flail chests caused by closed trauma do not require surgical intervention for stabilization and repair. However, it is worth re-emphasizing that surgical

exploration is essential for all thoracic wall lesions caused by open trauma and for all thorax bites, even where the skin is not breached (8-10).

Post-surgical monitoring and treatment

Following any surgery to the chest wall, respiratory and cardiovascular parameters must always be monitored post-operatively. This will include assessing the color of the mucosa and the capillary refill time, and measurement of arterial pressure and oxygen saturation. Hypothermia, hypotension and hypoventilation are the main potential complications. Oxygen therapy may be helpful since recovery may be slowed by pain, residual free air or fluid, dressings or secondary lung pathology. The intense pain from fractured ribs, along with any lung damage, contributes to hypoventilation (16-18) and hence pain management is vital in these patients. Optimal analgesia can be attained with systemic administration of suitable drugs (by bolus and continuous rate infusion as necessary) as well as transdermal patches, and/or local analgesia via intercostal and intrapleural infiltration using the thoracic tube. Blood gas analysis is often helpful, especially if there is hypoventilation. If necessary a drain tube may be placed to allow removal of any free air or fluid from the thorax. If there is free pleural fluid, it should be monitored carefully during the post-operative period; the fluid should be analyzed for bacterial growth and cellularity, and it is vital to determine the volume of fluid production and the trend (*i.e.*, is it increasing or decreasing on a daily basis?) – ideally

Figure 10. Resection of fractured ribs following a bite; note the large area of contused tissue affected by the bite.



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there should be less than 2 mL/kg/day, although reaching this figure is not mandatory.

■ Conclusion

Animals that have experienced thoracic trauma are often polytraumatized, and it is essential that the emergency veterinarian is able to accurately assess and prioritize such cases on presentation. Rapid diagnosis and appropriate

treatment (e.g., immediate thoracocentesis) can make the difference between life and death for many of these patients and the clinician must be able to respond as necessary in these situations; it is important to remember that the initial thoracic pathology can sometimes worsen within the first 24-48 hours after trauma, and it is vital to ensure that the patient is carefully monitored and frequently reassessed during the post-trauma period.

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Pain assessment in the dog: the Glasgow Pain Scale

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Pain is an unpleasant personal emotional experience. It has 3 dimensions (1):

- Sensory – discriminative (location, intensity, quality, duration).
- Motivational – affective (describes the unpleasantness – how the pain makes us feel).
- Cognitive – evaluative (influence of cognitive activities on the pain experience).

The conscious perception of pain is the final product of a complex neurologic information-processing system, resulting from the interplay of facilitatory and inhibitory pathways throughout the peripheral and central nervous systems. Adaptive “physiological” pain (*e.g.*, stubbing a toe) serves the vital purpose of rapidly altering behavior in order to avoid damage or minimize further damage, but maladaptive “clinical” pain represents malfunction of neurologic transmission and serves no physiological purpose. Poorly controlled acute pain leads to discomfort and suffering, as well as other unwanted consequences that can delay or impair recovery. Uncontrolled post-operative pain can lead to delayed healing, increased morbidity and the risk of developing chronic persistent pain which is very difficult to treat, so “prevention is better than cure”. Effective pain management must include properly conducted pain assessment carried out routinely and regularly throughout the post-operative period, as well as before and after analgesic administration to assess its effect.

The short form of the Glasgow Composite Measure Pain Scale (CMPS-SF) was designed as a practical decision-making tool for dogs in acute pain, and can be applied quickly and reliably in a clinical setting. There are 30 descriptor options within 6 behavioral categories, including mobility. Within each category, the descriptors are ranked numerically according to their associated pain severity; the person carrying out the assessment chooses the descriptor which best fits the dog’s behavior/condition. It is important to carry out the assessment procedure as detailed overleaf.

The pain score is the sum of the rank scores, with a maximum score of 24 (20 if mobility is impossible to assess). The total score is a useful indicator of analgesic requirement; the recommended analgesic intervention level is 6/24 (or 5/20). Note that the scale should be used only once dogs are fully conscious and ambulatory without assistance (except where mobility is contraindicated), so typically wait 2 hours after endotracheal extubation before assessment, but each case should be treated according to its individual circumstances. Because the protocol involves palpation around the surgical wound and a mobility assessment (unless contraindicated) it is recommended that scoring is not carried out more frequently than hourly in the early post-operative period to avoid unnecessary stress to the animal and to limit the deleterious effect frequent disturbance might have on subsequent measurements.

The suggested protocol for dogs in post-operative care using the CMPS-SF is as follows:

- Evaluate the dog once it has recovered sufficiently from anesthesia (as scores can be affected by the hangover effect of sedative and anesthetic drugs).
- If pain scores are greater than 5/20 or 6/24, consider giving analgesia.
- Allow the analgesic to take effect and reassess after an hour; if the score has decreased to below the intervention level, reassess in 2 hours. If not consider additional analgesia.
- Thereafter assess every 3-4 hours or earlier as appropriate (depending on the severity of the surgical procedure and the class/route of administration/expected duration of analgesic administered) and after each analgesic administration.
- The scale is intended to be an adjunct to clinical judgment, and no animal should be denied analgesia on the basis of the scores alone.

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SHORT FORM OF THE GLASGOW COMPOSITE MEASURE PAIN SCALE

Dog's name _____ **Date** / / **Time** _____
Patient ID _____
Procedure or condition _____

In the sections below, please circle the appropriate score in each list and sum these to give the total score

A. Look at dog in kennel

(I) Is the dog?

- Quiet 0
- Crying or whimpering 1
- Groaning 2
- Screaming 3

(II) Is the dog?

- Ignoring any wound or painful area 0
- Looking at wound or painful area 1
- Licking wound or painful area 2
- Rubbing wound or painful area 3
- Chewing wound or painful area 4

In the case of spinal, pelvic or multiple limb fractures, or where assistance is required to aid locomotion, do not carry out section B and proceed to C.

Please tick if this is the case

B. Put lead on dog and walk animal out of the kennel

(III) When the dog rises/walks, is it?

- Normal 0
- Lame 1
- Slow or reluctant 2
- Stiff 3
- It refuses to move 4

C. If the dog has a wound or painful area including abdomen, apply gentle pressure 2 inches (5 cm) around the site

(IV) Does it?

- Do nothing 0
- Look round 1
- Flinch 2
- Growl or guard area 3
- Snap 4
- Cry 5

D. Overall

(V) Is the dog?

- Happy and content or happy and bouncy 0
- Quiet 1
- Indifferent or non-responsive to surroundings 2
- Nervous or anxious or fearful 3
- Depressed or non-responsive to stimulation 4

(VI) Is the dog?

- Comfortable 0
- Unsettled 1
- Restless 2
- Hunched or tense 3
- Rigid 4

Total score (I + II + III + IV + V + VI) = _____

The pain score is the sum of the rank scores, with a maximum score of 24 (20 if mobility is impossible to assess). The total score is a useful indicator of analgesic requirement; the recommended analgesic intervention level is 6/24 (or 5/20).

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