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Emergency Room Crash Course Online 'Mini Series'

Session 2: How to Assess and Stabilise Your Patient Following Motor Vehicle Trauma

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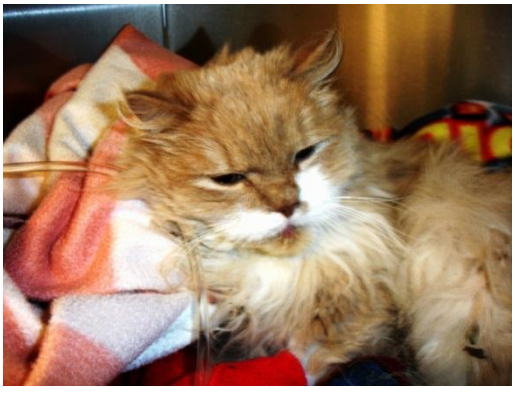

OVERVIEW

1. General Approach
2. The Abdomen in Trauma
3. Thoracic Injuries
4. Traumatic Brain Injury
5. Feline Sacrocaudal/Tail Pull Injury

GENERAL APPROACH

Trauma is relatively common in companion animals; cats hit by vehicles are probably the most common examples but other types of blunt trauma as well as for example injuries following fights are also seen relatively commonly.

Some injuries following motor vehicle accidents:

- Head
  - Traumatic brain injury
  - Ocular injury
  - Skull fracture
  - Dental injury
- Thorax
  - Pulmonary contusions
  - Pneumothorax
  - Diaphragmatic rupture
  - Haemothorax
  - Rib fractures
  - (Flail chest)
- Abdomen
  - Abdominal wall rupture
  - Haemoabdomen
  - Uroabdomen – non-abdominal urinary tract rupture possible
  - Bile peritonitis
  - Septic peritonitis from gastrointestinal rupture
- Spine (vertebral column)/spinal cord
  - Vertebral fracture
  - Vertebral luxation/subluxation

    - Intervertebral disc extrusion/herniation/prolapse
- Pelvic fractures
- Limbs
    - Long bone fracture
    - Joint luxation/subluxation
    - Tendon/ligament injury
    - Degloving injury
    - Wounds

A **general approach to the emergency patient** involves the application of certain core principles or treatments before providing specific therapies or interventions for the particular problems present, and this approach applies equally to trauma cases.

It is essential to realise that the early management of the trauma patient can have significant consequences on both subsequent morbidity and mortality. A rational approach to the trauma patient starts with a major body system examination – cardiovascular system, respiratory system, and the central nervous system (mentation, gait). The two most important problems to look for are:

- Poor systemic *perfusion* due to *hypovolaemia*: does the patient need immediate intravenous fluid therapy to correct hypovolaemia?
- *Respiratory compromise*: does the patient need oxygen supplementation and perhaps thoracocentesis for dyspnoea?

Next consider what level of *analgesia* the patient requires. Then go on to perform a **more thorough assessment**, ideally including an abdominal free fluid scan and some sort of emergency database bloodwork, and provide specific interventions for injuries sustained as indicated. A complete physical examination should be undertaken to search for any soft tissue, skeletal or neurological injuries; first aid should be applied to soft tissue and orthopaedic injuries identified in the initial stabilisation period. A thorough history should be obtained from the owner, including previous medical/surgical diseases and concurrent medications.

Bear in mind that any interventions should only be performed at the appropriate time based on patient stability always thinking about the risk-benefit assessment: how beneficial is the proposed intervention to the patient? Is it informative? Essential? Will it impact on-going management or prognosis? Does it need to be performed at all? At that moment in time? What are the associated risks? Etc. A lot of emergency medicine – in fact medicine in general – revolves around making risk-benefit assessments.

Trauma is relatively common in companion animals; cats hit by vehicles are probably the most common examples but other types of blunt trauma as well as for example injuries following fights are also seen relatively commonly.

Most common causes:

- Motor vehicle accidents
- Animal (especially dog) bite injuries
- Tail pull injuries
- Falls from a height (esp. feline high-rise syndrome)
- Puppies and kittens trodden on
- Dogs kicked by horses

Cranial, thoracic and pelvic injuries especially common in cats

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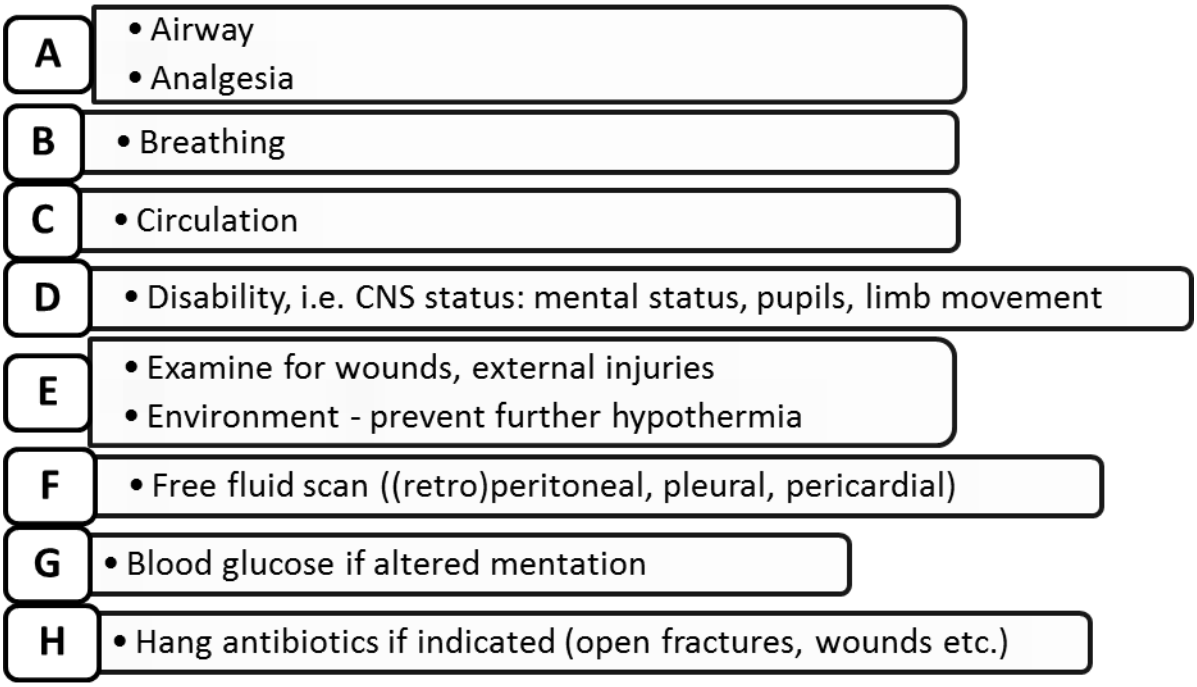
Mnemonic for basic approach – performed sequentially or concurrently depending on your circumstances:



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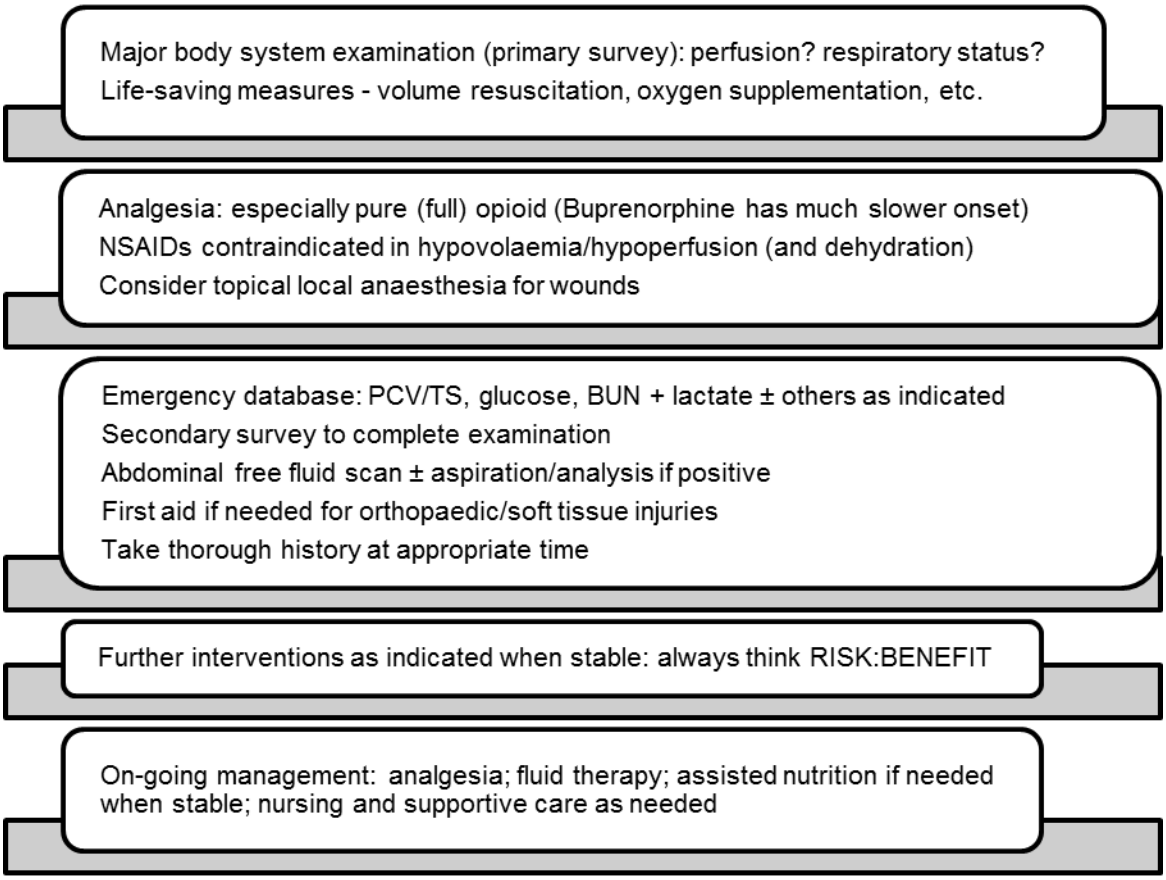


Cardiovascular examination

Evaluation involves examination of the following:

- Heart rate and rhythm
- Heart sounds
- Pulse quality and rate
- Mucous membrane colour
- Capillary refill time
- (Mentation; temperature of the extremities)

The most important questions to answer are:
Is systemic perfusion normal? Perfusion is a key concept in emergency medicine.
Is there a murmur or gallop sound suggesting possible primary cardiac disease?

Heart rate and rhythm

Normal adult range in **dogs**: 70-120 bpm

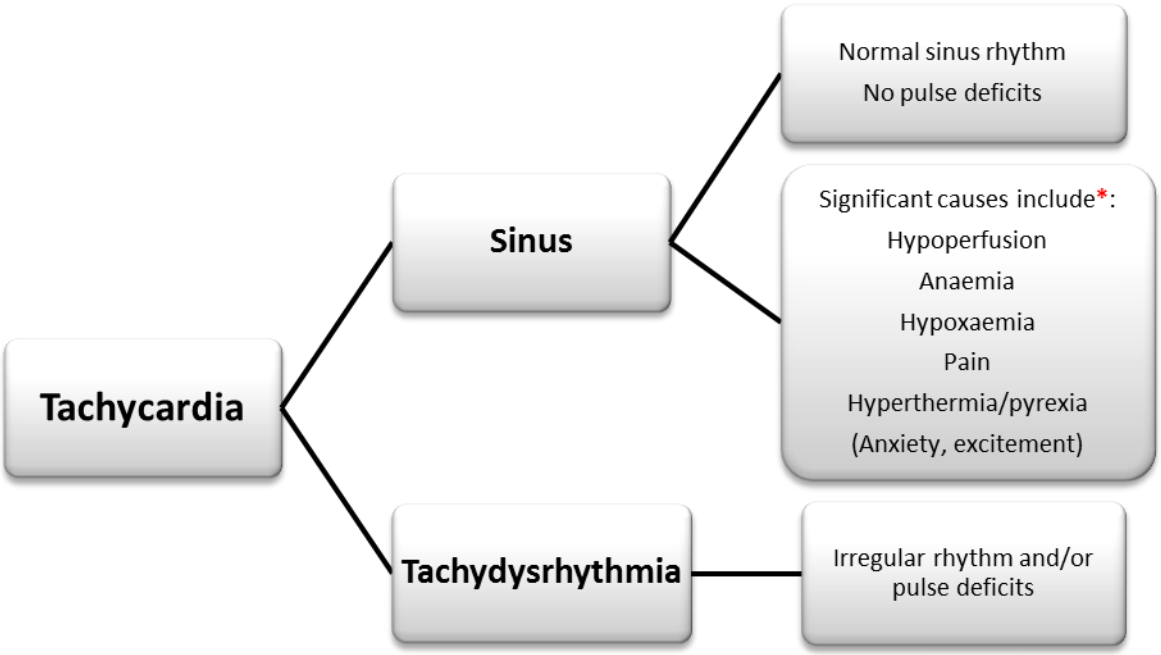
- Larger dogs usually have slower rates than smaller dogs
- Potentially affected by various other factors
- Interpret the measured HR in the context of each individual patient
-

In general when interpreting physical examination findings it is essential to ask not just if the measured value falls within quoted 'normal ranges', but whether it is *appropriate for the individual patient in question.*

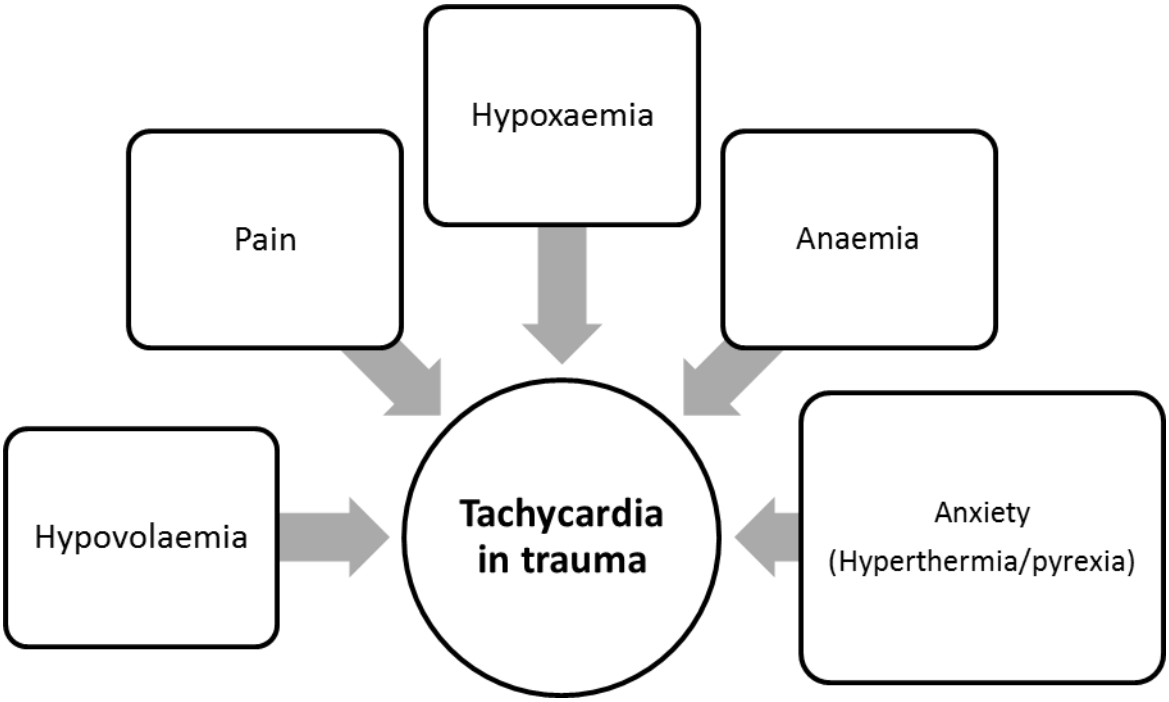
Normal adult range in **cats**: 160-200 bpm (at initial consultation)

- More susceptible to 'stress' and much higher rates (e.g. 220-240 bpm) may be found in healthy cats

Tachycardia



* Most or indeed all of these may contribute to tachycardia in trauma patients.



Bradycardia

Both sinus bradycardia and bradydysrhythmias considered significant. Causes include:

- High vagal tone: many causes, e.g. respiratory disease, intra-abdominal disorders
- Drug side effect: especially anaesthetic agents and (pure) opioids
- Cardiac conduction disturbances (e.g. atrioventricular block)
- Brain disease (secondary to raised intracranial pressure)
- Hyperkalaemia (e.g. urethral obstruction, Addison's disease)
- Hypoglycaemia (probably only if moderate-to-severe)

Absolute, or indeed relative (i.e. inappropriately low rate for the patient's perfusion/pain status), bradycardia in trauma patients may be due to raised intracranial pressure in traumatic brain injury, high vagal tone due to pain in/injury to a variety of anatomical sites, or potentially hyperkalaemia secondary to urinary tract rupture. (Although considered rare it may also occur as part of neurogenic shock secondary to damage to the cervical or upper thoracic spinal cord (see below).)

Bradycardia in haemorrhagic shock:

A phenomenon whereby patients with hypotension secondary to haemorrhage, in particular intra-abdominal haemorrhage, have a relative bradycardia has been identified. The mechanism(s) involved remain unclear with several having been suggested, e.g. a parasympathetic reflex (e.g. via vagus nerve), a vagal-vagal reflex arch.

In general any patient presenting with shock with what would be considered an inappropriately low heart rate that cannot be explained by other causes may be experiencing autonomic dysfunction with sympathetic-parasympathetic imbalance.

Heart sounds

Most dogs with clinically significant heart disease have a readily audible murmur.
Many cats with clinically significant heart disease have a readily audible gallop sound (third heart sound) or murmur.
Murmurs are more likely to be audible at higher rates or in the presence of greater sympathetic stimulation of the heart.
Quiet heart sounds may be due to muffling (especially from pericardial effusion; also e.g. pleural effusion, mass/organs between stethoscope and heart) but possibly also hypovolaemia.

The true incidence of traumatic pericardial effusion in dogs and cats is still unknown but is currently thought to be rare, or at least it is rare for it to occur to such an extent as to cause muffling of heart sounds. Traumatic haemothorax is more common but again typically is not so severe as to muffle heart sounds. Diaphragmatic rupture with displacement of abdominal viscera into the thoracic cavity may potentially cause heart sounds to be quiet – often this is unilateral with cardiac displacement towards and readily audible heart sounds on the other side. Trauma patients with severe hypovolaemia may also have quiet heart sounds until perfusion has been resuscitated.

Normal cardiac auscultation does not exclude the presence of clinically significant heart disease.

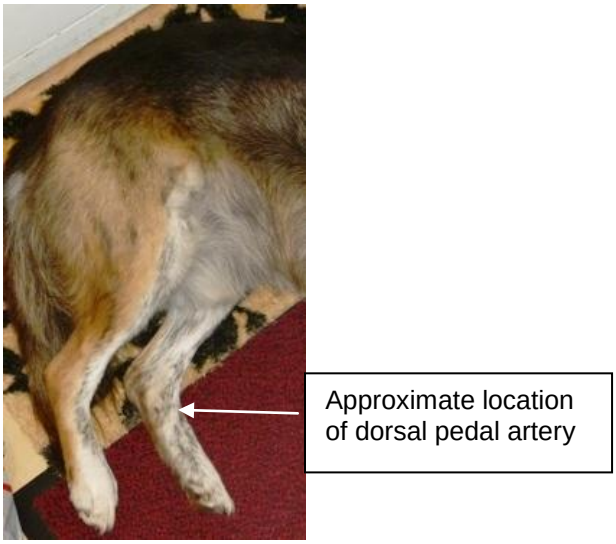
Pulse quality and rate

Arterial sites: especially *femoral* (medial aspect of proximal hindlimb) and *dorsal pedal* (metatarsal; distal to hock on craniomedial aspect).

Normal arterial pulse: readily palpable and strong
Stronger or easier than normal to feel: 'bounding', 'snappy', 'hyperdynamic'

- Typically tachycardic
- Causes e.g. early shock, anaemia

Weak, very weak/'thready' or absent: e.g. later stages of shock; low output (forward) heart failure



Pulse deficit: pulse rate lower than concurrent heart rate

- Due to dysrhythmia with some heart beats unable to generate palpable peripheral arterial pulse
- Perform ECG to evaluate further
- Relatively rare in trauma patients but may occur for example with myocardial bruising

Mucous membrane colour

Normal salmon pink appearance requires there to be enough oxyhaemoglobin in enough red blood cells in the capillaries of the mucous membrane site being examined. Abnormalities are:

Pallor
Injection/congestion
Cyanosis
Other abnormalities

Pallor

Causes of:

- Poor blood flow due to vasoconstriction (e.g. common in shock; also hypothermia)
- Overall lack of circulating red cells in anaemia
- Both mechanisms may be present in trauma patients

Injection/congestion

Increased blood flow (hyperaemia) gives membranes that range from being pinker than normal right through to bright red
Mild cases, e.g. due to tachycardia and increased cardiac contractility in early shock
More severe cases are usually due to vasodilation and tachycardia, e.g. systemic inflammation, vasodilatory anaphylaxis, hyperthermia.
Mild congestion may be seen in trauma patients in early shock but more severe injection is unusual.

Cyanosis

Bluish or purplish discoloration of the mucous membranes or skin due to a severe decrease in the amount of oxygen dissolved in the perfusing blood.
Central cyanosis typically indicates life-threatening systemic hypoxaemia due to circulatory and/or ventilatory failure.
Cyanosis in trauma is most likely to be central in origin.
It may be difficult to detect if perfusion is very poor or the patient has a packed cell volume (PCV) < 15% (as > 5 g/dL of deoxyhaemoglobin is required for cyanosis to be visible).

All cyanotic patients require urgent intervention with oxygen supplementation and addressing the underlying cause if/as possible.

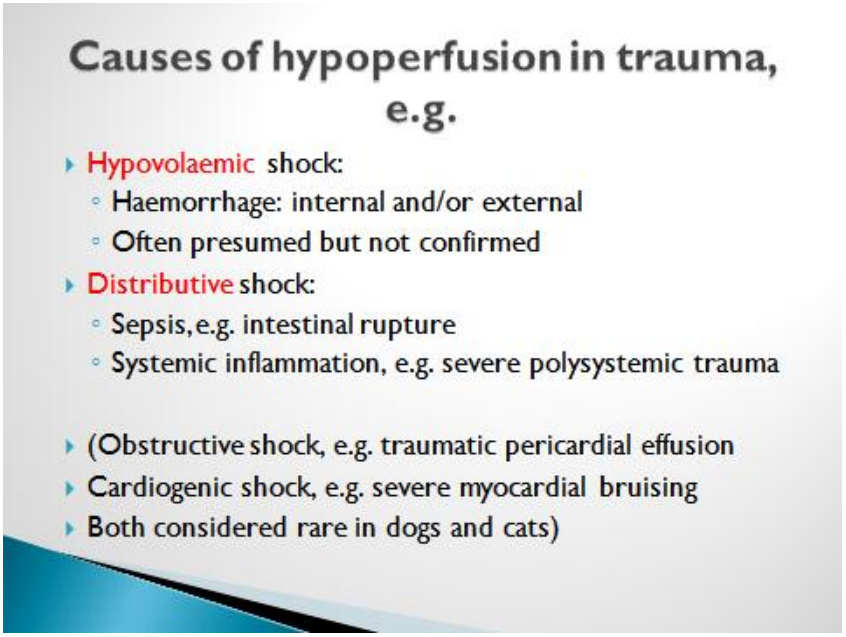
Capillary refill time

Affected by blood flow to the site and therefore varies predominantly according to whether the underlying vessels are constricted or dilated:

- Normal: 1-2 seconds
- Slow (> 2 seconds): especially in peripheral vasoconstriction (e.g. shock, hypothermia)
- Fast (< 1 second): fastest times especially seen in vasodilatory states (e.g. systemic inflammation, hyperthermia) – often accompanied by tachycardia and congestion.

- CRT may also be fast in mild (early compensatory) hypovolaemic shock.

Hypoperfusion in trauma patients may be the result of hypovolaemia secondary to blood loss. Other factors may also be involved, for example sepsis secondary to intestinal rupture or systemic inflammation due to severe polysystemic trauma. There does appear to be a proportion of patients where a focus of significant haemorrhage cannot be identified and blood work does not support significant blood loss. A specific reason for the patient's hypoperfusion may not be forthcoming but nevertheless they respond favourably to fluid resuscitation.



[Neurogenic shock: although considered a comparatively rare presentation, animals that suffer cervical or upper thoracic spinal cord damage may present with neurogenic shock. Peripheral sympathetic denervation results in untempered parasympathetic output that can cause bradycardia as well as vasodilation from decreased vascular tone. This should be considered a diagnosis of exclusion made after excluding other causes of shock and only in patients with compatible spinal cord injuries.]

The hypovolaemic dog

In dogs with 'uncomplicated' hypovolaemia physical examination perfusion parameters tend to change in a predictable way both as hypovolaemia progresses and as normovolaemia is restored.

Perfusion parameter	Mild hypovolaemia Compensatory	Moderate hypovolaemia Early decompensatory	Severe hypovolaemia Late decompensatory
Heart rate	120-150 bpm	150-170 bpm	170-220 bpm
Femoral pulse	Bounding, snappy	Weak	Very weak or thready
Dorsal pedal pulse	Readily palpable	Just palpable	Not palpable
Mucous membrane colour	Normal, pinker	Pale pink	Very pale/white
Capillary refill time	≤ 1 second	1-2 seconds	> 2 seconds or not detectable
Mentation	Usually normal	Depressed	Severely depressed
Extremities	Usually normal	Cool (or normal)	Cold

Compensatory phase/mild hypovolaemia:

- Sympathetic nervous system activation
- Increase in heart rate and cardiac contractility
- Peripheral vasoconstriction
- Hyperdynamic clinical picture

Early decompensatory phase/moderate hypovolaemia:

- Progressive failure of compensatory mechanisms results in reduced tissue perfusion

Late decompensatory phase/severe hypovolaemia:

- Systemic perfusion is severely compromised; hypodynamic clinical picture
- Usually tachycardic but some dogs may have an inappropriately 'normal' or slow heart rate – this is due to failure to sustain the compensatory chronotropic response

Note that physiological sinus tachycardia is unlikely to exceed 220-240 bpm in a dog (300 bpm in a cat). Faster heart rates are likely to be due to tachydysrhythmia.

The hypovolaemic cat

Given that the typical hypovolaemic cat has a hypodynamic clinical picture, a cat presenting like this should be seen as having a life-threatening problem but the prognosis is not necessarily grave if appropriate treatment is provided.

'Cats are not small dogs' and the classic presentation for the hypoperfused cat is a hypodynamic picture with a hyperdynamic picture (tachycardia, hyperaemic membranes, fast CRT) being rarely identified.

Typical hypovolaemic cat:

- 'Inappropriate' bradycardia – heart rate 120-160 bpm
- Weak or absent pulses
- Pale mucous membranes
- Prolonged (> 2.5 seconds) or undetectable CRT
- Depression
- Hypothermia

Should hypothermic hypovolaemic cats be warmed aggressively?

Hypothermia significantly decreases the cardiovascular response to fluid resuscitation. However it is possible that re-warming the patient too quickly before administering sufficient fluid therapy may worsen their perfusion status as the increase in body temperature may cause peripheral vasodilatation thereby increasing the intravascular space. Not everyone agrees with this – some people believe that non-sweating animals like dogs and cats do not vasodilate (significantly) with warming especially when core temperature is low and there is even some suggestion that cats in shock may already be relatively vasodilated peripherally compared to dogs. These individuals therefore argue for more aggressive warming early on. However the most widely accepted recommendation is to limit on-going heat loss with passive warming (e.g. wrap in blankets, place in incubator) during initial fluid resuscitation. In many cases as the patient's perfusion improves their rectal temperature will often increase notably; however more aggressive warming can be performed following initial fluid resuscitation if still thought necessary.

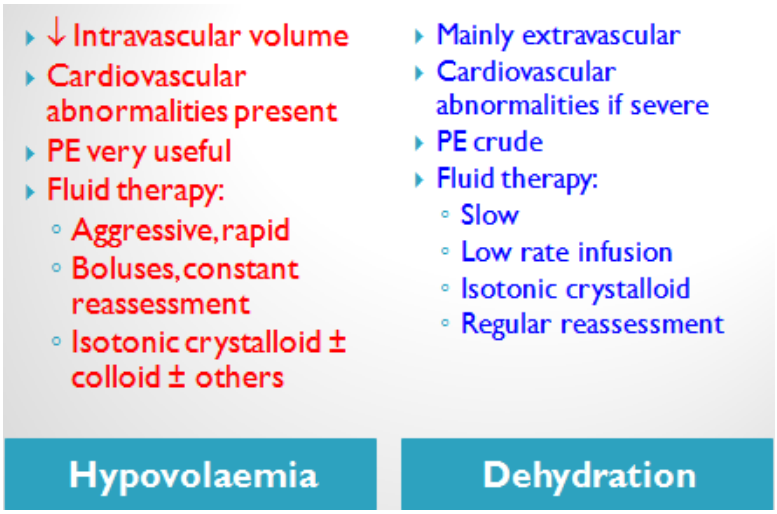
‘Complicated’ hypovolaemia

Hypovolaemia is not always ‘uncomplicated’. A proportion of hypovolaemic patients have other abnormalities that may affect one or more of the physical perfusion parameters described. Common examples include:

- Mucous membranes may remain pale despite appropriate fluid therapy in an animal with severe anaemia
- Mentation may remain depressed in an animal with head trauma despite appropriate fluid therapy as a result of raised intracranial pressure

Fluid therapy in blunt trauma

Patients hit by motor vehicles may present with hypovolaemia as described above. Depending on the timeframe elapsed since the accident some degree of dehydration may also have developed – although this is of less concern until hypovolaemia has been corrected and perfusion restored. It may also be that on occasion a sick dehydrated animal is hit by a vehicle because he/she is sick and therefore less able to avoid the vehicle, i.e. occasionally pre-existing dehydration may be present. Assessment of perfusion versus hydration and the appropriate use of intravenous fluid therapy for these indications were discussed in a previous set of notes and will not be covered in detail here. Some summary information is as follows:



Special considerations

The priority in treating hypovolaemic patients is to restore an effective circulating volume. Nevertheless there are some patient populations in which a less aggressive approach is warranted with smaller fluid boluses being used and/or fluid boluses being given over a longer period of time. Occasionally it is tolerated that a patient remains borderline hypovolaemic rather than risk fluid overload.

Scenarios relating to the motor vehicle trauma patient that warrant specific consideration include:

Recent or active haemorrhage
Lung pathology
Raised intracranial pressure

[Others not related to trauma include:

Heart disease
Chronic anaemia
Renal insufficiency]

Recent or active haemorrhage

Much has been written about the 'best' approach to the resuscitation of haemorrhagic hypovolaemia. Most of this has been based on work done in human patients and in experimental animals. Essentially fluid therapy in haemorrhagic shock aims to:

- Restore intravascular volume
- Restore blood-oxygen carrying capacity
- Normalise coagulation status

Which fluid to use?

In terms of isotonic crystalloid versus synthetic colloid, there is no evidence-based consensus that one is 'better' than the other here and both have pros and cons. The majority of clinicians use a replacement isotonic crystalloid in the first instance and the potential for synthetic colloids to cause dose-dependent coagulopathy must not be overlooked in patients already bleeding.

Haemostatic resuscitation

As well as the potential for anaemia, bleeding patients have various reasons to be coagulopathic, both primarily, i.e. if a coagulopathy is the cause of bleeding in the first place and secondarily – for example due to inflammation from trauma; hypothermia ('lethal triad' of hypothermia, acidaemia and coagulopathy); clotting factor consumption; and dilutional coagulopathy from resuscitative fluid already administered. 'Haemostatic resuscitation' describes the aggressive early use of packed red blood cells and clotting products, a practice which is increasingly preferred in human patients as a way of restoring, or at least minimising impact on, oxygen-carrying capacity and coagulation status while improving intravascular volume. However lack of resources means that this approach is unlikely to be realistic in most veterinary environments in which avoiding both synthetic colloid use if possible and overzealous crystalloid administration may be sensible.

Hypotensive resuscitation

Haemorrhagic hypovolaemia is one example in which the concept of 'permissive hypotension' or 'hypotensive resuscitation' has been put forward as opposed to more standard 'normotensive resuscitation'. This involves titrating volume resuscitation to a sub-normal end-point that should nevertheless improve tissue oxygen delivery enough to maintain organ viability until such time as bleeding can be controlled. So for example in patients in which systemic arterial blood pressure can be reliably monitored, one may usually aim to resuscitate to a mean arterial pressure of 80 mmHg (or a systolic blood pressure of 100 mmHg); in permissive hypotension the end-point might be MAP 65 mmHg (or systolic 80 mmHg).

By avoiding overzealous fluid resuscitation which targets normotension, the hope is to preserve the first and often best clot at any sites of bleeding. The need to control bleeding as soon as possible by either temporary (e.g. tourniquet, haemostatic agent) or permanent (i.e. surgical intervention) measures is important to stress although in some patients bleeding will be self-limiting. More aggressive fluid resuscitation is then used as required once haemorrhage is controlled.

In bleeding patients with head trauma, normotensive resuscitation would seem most rational to ensure adequate cerebral perfusion pressure and thereby try to minimise secondary brain injury; the importance of CPP in brain injury has a strong evidence base at least in human patients if not yet in clinical dogs and cats. This may also be true for spinal cord perfusion pressure in spinal cord injury but more evidence is needed.

Note that permissive hypotension is usually only considered in patients with on-going haemorrhage in which it is planned to intervene to control the bleeding and this technique is used to maintain organ viability in the meantime. This is a patient population that is encountered much less frequently in veterinary medicine than in human medicine so care is needed not to over-apply this resuscitative technique the evidence base for which is still scarce in humans, even more so in dogs and cats.

Lung pathology

The lungs have extensive capacity to cope with fluid but pathological lungs may be more prone to oedema than healthy lungs. This scenario is encountered most commonly in the form of pulmonary contusions following thoracic trauma. Animals suffering from severe trauma (thoracic or non-thoracic) or from other causes of shock may also develop acute lung injury (ALI) or acute respiratory distress syndrome (ARDS); this is especially true for cats.

In hypovolaemic animals with lung pathology that do not respond adequately to conservative crystalloid boluses, a synthetic colloid may prove effective. However in some types of lung pathology, the pulmonary vasculature can become leakier to colloid molecules despite their large size. Once the colloid molecules move into the pulmonary interstitium they may worsen pulmonary oedema and may persist there for some time. Nevertheless it is not possible to predict in advance whether this will occur and it is therefore recommended to assess the patient's response to a colloid bolus and discontinue this therapy if respiratory status worsens as a result.

Raised intracranial pressure

This will be discussed in the notes on Traumatic Brain Injury.

Respiratory system

It is very important not to stress animals in respiratory distress; these animals – especially cats – can be very unstable and may decompensate easily. In some cases even a brief major body system examination will not be tolerated. Observation from a distance, gentle handling, oxygen supplementation and empirical interventions are potentially important components of their management.

Evaluation involves examination of the following:

- Respiratory rate
- Respiratory effort
- Respiratory pattern/noise

Respiratory rate

Normal adult range in **dogs** and **cats**: 15-30 breaths per minute

- Higher rates are relatively common, often associated with shallow breathing (tachypnoea) and the key question to consider is whether this is the result of a primary respiratory abnormality or secondary for example to pain, anxiety, excitement amongst other causes.

Beware of (excessive) panting: don't automatically dismiss panting as being of minimal concern as occasionally it is the result of early compensation for respiratory embarrassment.

Respiratory effort

Abnormal respiratory rate and effort may be seen due to primary abnormalities in both the respiratory system and in other systems (neurological, metabolic). In general respiratory abnormalities secondary to non-respiratory factors are less severe.

Postural adaptations to alleviate air hunger in dyspnoeic patients include:

- Open-mouth breathing
- Neck extension
- Repeated changing of position
- Abduction of elbows (dogs)
- Sternal recumbency (cats)
- Anxious expression

Increased abdominal effort:

- Increased contraction of abdominal muscles
- Assists with expiration only

Paradoxical abdominal breathing:

- During normal inspiration both the thoracic (actively) and abdominal (passively) walls move outwards;
- In paradoxical abdominal breathing, they move in opposite directions
- Generally associated with severe respiratory distress; may also be more common with diaphragmatic dysfunction and pleural space diseases

Respiratory pattern/noise

Ideally the aim is to try to localise the cause of the patient's respiratory abnormality to one or more of the following:

<table>
<tr><th>Respiratory findings</th><th colspan="2">Localisation</th></tr>
<tr><td>Prolonged inspiration

Inspiratory stridor: harsh high pitched noise; especially from partial laryngeal obstruction (e.g. laryngeal paralysis)

Stertor: 'snoring'; usually due to partial obstruction of airflow through the nasal passages or nasopharynx (e.g. brachycephalic airway obstruction syndrome)</td><td>Dynamic obstruction</td><td rowspan="2">Upper respiratory tract</td></tr>
<tr><td>Inspiratory and expiratory stridor</td><td>Fixed obstruction (e.g. mass lesion)</td></tr>
<tr><td>Prolonged expiration with increased abdominal effort</td><td colspan="2">Lower airway disease</td></tr>
<tr><td>Lung sounds louder than normal: harshness, crackles**, wheezes</td><td colspan="2">Small airway or lung parenchymal disease (e.g. pulmonary contusions*)</td></tr>
<tr><td>Lung sounds muffled ventrally</td><td colspan="2">Pleural space: effusion (e.g. traumatic haemothorax*)</td></tr>
<tr><td>Lung sounds quiet dorsally</td><td colspan="2">Pleural space: pneumothorax*</td></tr>
<tr><td></td><td colspan="2">Thoracic wall and diaphragm</td></tr>
<tr><td>Often rapid shallow breathing (tachypnoea)</td><td colspan="2">*Primary neurological, metabolic or cardiovascular influences</td></tr>
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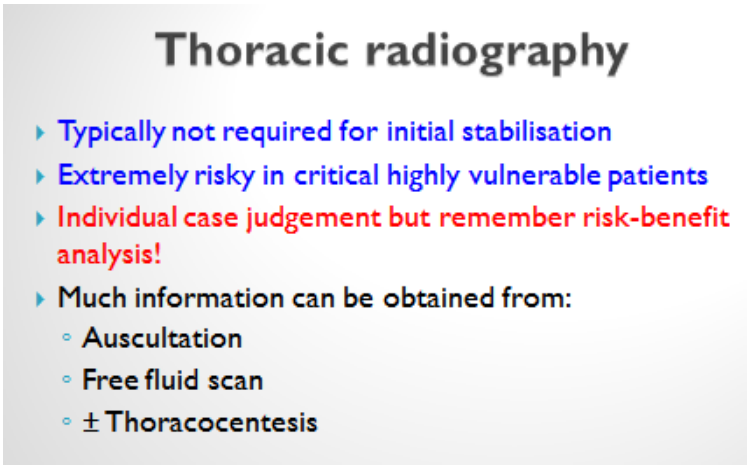
* As discussed later, these are some of the variably common causes of respiratory abnormalities in trauma patients.

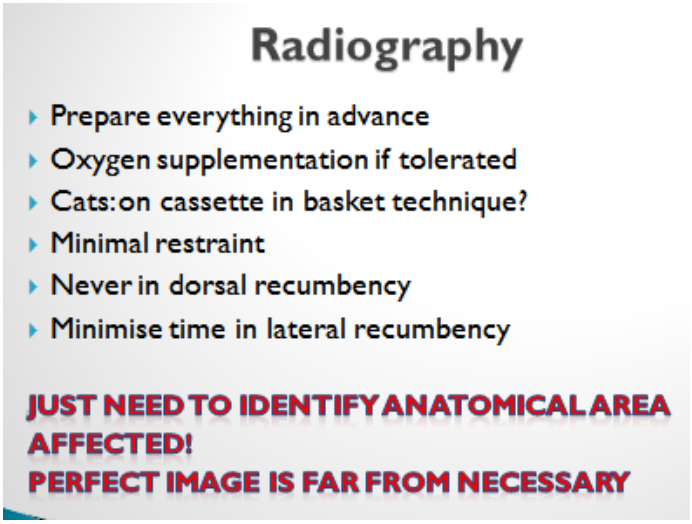
** It can be difficult to hear crackles in animals with a low tidal volume, e.g. during panting or tachypnoea.

Causes of decreased lung sounds:

- Pleural fluid (ventrally)
- Pneumothorax (dorsally)
- Intra-thoracic masses (heart sounds displaced? reduced rib cage compliance?)
- Diaphragmatic hernia (bowel sounds in thorax?)
- Rarely decreased ventilation

Lung sounds must be related to the degree of respiratory effort the patient is showing – apparently normal lung sounds may be inappropriately quiet in a patient with a marked increase in effort.





Central nervous system

Mentation

Normal mentation (awareness, consciousness) is alert with normal response to environmental stimuli. Types of abnormal mentation are:

Depressed/obtunded: Decreased functional activity; mentally dulled; drowsy; less responsive to environmental stimuli
Stuporous: Unresponsive to normal environmental stimuli but responsive to painful stimuli
Comatose: Unresponsive to environmental or painful stimuli
Hyperaesthetic: Increased sensitivity to a normal level of stimulation
Confused/disorientated (delirious): Responding to environmental stimuli in an inappropriate manner

Abnormal mentation may be due to one or both of:

- Primary CNS disorder: concurrent cranial ± peripheral nerve abnormalities more likely
- Primary abnormalities in other systems, e.g. hypoperfusion, hypothermia, hypoglycaemia: concurrent cranial ± peripheral nerve abnormalities less likely

When assessing mentation, consider whether the patient's consciousness may be affected by any other concurrent abnormalities. Abnormal mentation that is not explained by abnormalities in other systems gives a high index of suspicion for a primary central nervous system disorder.

Remember it may not be possible to reliably interpret mentation until other concurrent abnormalities that can affect consciousness have been adequately addressed (e.g. fluid therapy for hypovolaemia, warming for hypothermia). This is an important consideration in traumatic brain injury.

Gait

Gait abnormalities are most often related to central or peripheral neurological disorders.
Multiple limb involvement usually means more serious disease.
Recumbency may be due to a primary CNS disorder but also e.g. cardiovascular compromise; respiratory embarrassment; severe pain. Most animals with primary central neurological recumbency do not have cardiovascular instability.

Pain assessment

A full discussion of pain assessment and treatment is beyond the scope of these notes and was discussed in an earlier webinar in this series. A few summary points only will be made here.

If in doubt, treat for pain! As long as they are used rationally, analgesic agents, opioids in particular, carry little risk but offer potentially great benefit, i.e. the risk-benefit assessment lands heavily on the side of benefit. Give a low testing dose and assess the patient for a positive response confirming that pain has been alleviated.

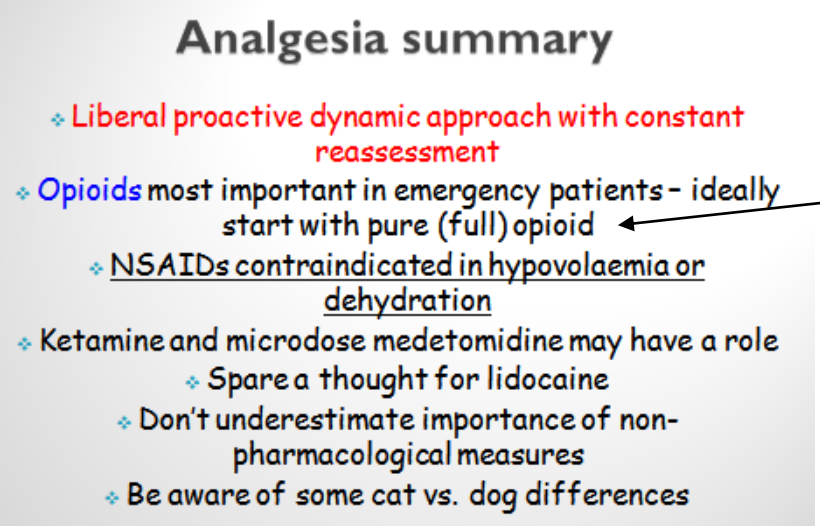
Untreated pain is clearly bad for patient welfare.
Pain may also affect physiological parameters thereby confusing clinical assessment; however it is essential to realise that physiological parameters such as heart rate and respiratory rate may be normal in a painful animal – normal parameters do not exclude the possibility of pain!
Detecting pain can be difficult, especially in cats. Sick animals may also be unable to exhibit behavioural signs of pain.

Sensitisation or 'wind up' pain:

'Wind up' refers to changes in nervous system processing that occur following noxious stimulation and result in greater conscious pain perception with continued pain that is harder to control. A proactive preventative approach to pain management is therefore essential.

Emergency trauma patients are often already painful at presentation. To minimise wind up:

- Administer analgesia as soon as possible to reach a pain-free state
- Maintain the patient at this pain-free plateau
- Ensure adequate analgesia prior to any surgical or other potentially noxious procedures



Buprenorphine may be an effective analgesic for mild-to-moderate pain but it has a longer onset of action (e.g. up to 45 minutes) than pure opioids and is therefore not ideal as a first-line analgesic.

NSAIDs can prove very useful in trauma patients once there is no longer any concern for on-going cardiovascular instability or hydration abnormalities, and as long as no other contraindications exist.

Emergency database

A minimum database consisting of manual packed cell volume (PCV), plasma total solids/protein (TS), blood glucose and blood urea nitrogen should be performed in the trauma patient. If available, blood lactate concentration is also extremely useful as a marker of hypoperfusion and successful resuscitation.

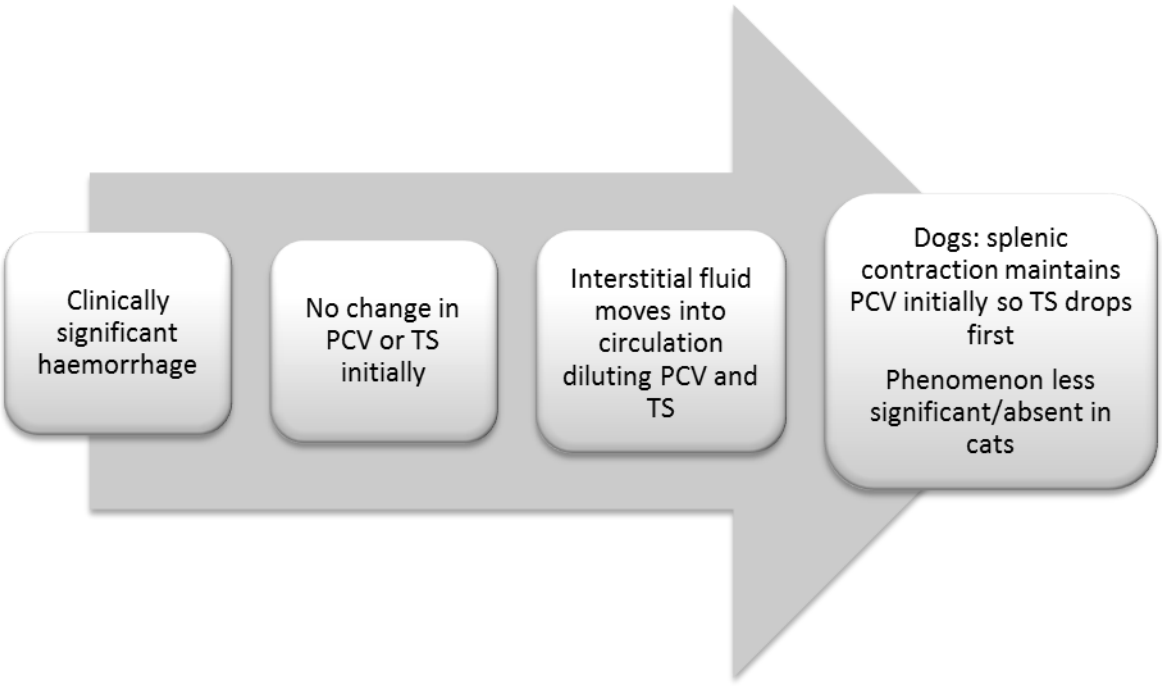
Manual packed cell volume (PCV) and plasma total solids/protein (TS/TP) measurement may be suggestive of significant haemorrhage within the patient although changes are clearly not specific to intra-abdominal haemorrhage.

Both red blood cells and protein are lost from the circulation in haemorrhage and therefore PCV and TS do not change initially.

- Remember that PCV is a percentage and TS a concentration, i.e. neither is a measure of absolute quantity.
- In the first few minutes following haemorrhage, the absolute number of red blood cells and plasma protein molecules will be reduced but PCV and TS are unchanged.

Fluid then moves from the interstitial compartment into the bloodstream diluting the remaining red cells and protein and causing a decrease in the measured PCV and TS.

- It takes a while for fluid shift and therefore dilution to occur although it is not possible to be too precise about the exact length of time in clinical patients.
- In dogs the spleen contracts in response to haemorrhage and expels a large amount of stored red blood cells into the circulation; therefore PCV may remain in the normal range for a while despite low TS, i.e. with blood loss, TS is usually expected to fall first followed by PCV in dogs.
    - The response of the spleen in cats is either much less substantial or in fact non-existent depending on which reference one consults.
- As yet more time passes PCV will also fall depending on whether haemorrhage is on-going, the severity of any on-going haemorrhage, and any treatment instituted.



Hypoglycaemia is a relatively rare finding in the trauma patient but may for example occur in very young or small animals. Mild(-to-moderate) hyperglycaemia on the other hand is relatively common but does not require any specific intervention. The significance of hyperglycaemia in traumatic brain injury is discussed below.

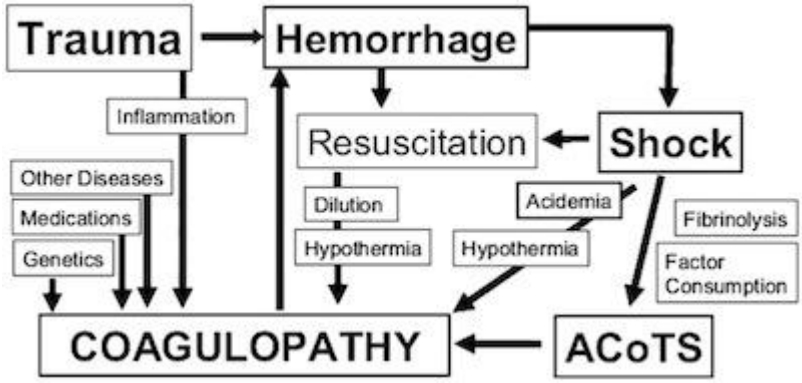
Blood urea may be elevated, either mildly-to-moderately due to pre-renal azotaemia and/or gastrointestinal haemorrhage, or more severely due to post-renal azotaemia secondary to urinary tract rupture.

Other plasma parameters such as potassium, creatinine or bilirubin may need to be measured for comparative purposes if abdominal free fluid is detected and uroabdomen or biliary peritonitis is suspected.

Transfusion in trauma

As mentioned above, haemostatic resuscitation is increasingly preferred in human traumatology essentially replacing loss of whole blood with equivalent constituents in the form of packed red blood cells, plasma and platelets. However this strategy is highly unrealistic given the limitations in financial and practical resources in veterinary medicine. Nevertheless following the initial resuscitative phase, some trauma patients will benefit from transfusion. Anaemia and coagulopathy may occur due to blood loss and be exacerbated by dilution from crystalloid and colloid fluid therapy; synthetic colloids can also cause a dose-dependent multifactorial coagulopathy.

As shown below there are various mechanisms of coagulopathy in trauma:



(ACoTS = Acute Coagulopathy of Trauma Shock)

An in-depth discussion is beyond the scope of these notes. Anaemic patients should be transfused not just on the basis of a specific PCV level but also with consideration to whether the anaemia appears to be clinically significant and whether blood loss is considered to be on-going. A relatively liberal approach to the use of plasma is suggested in patients with measured coagulopathy.

Nutrition in trauma

The benefits of ensuring adequate nutrition at the appropriate time are undisputed and several studies have documented a correlation between nutritional status and outcome during critical illness including trauma. Starvation of ill or injured animals may be associated with **hypermetabolism** (i.e. increased resting metabolic rate) and **stress starvation**, characterised by:

- Increase in protein catabolism proportional to the extent of disease leading to breakdown of stored protein
- Up-regulated production of catecholamines and other stress hormones leading to
    - Increased cardiac output and systemic vascular resistance
    - Insulin resistance and glucose intolerance
    - Proliferation of inflammatory mediators
    - Rapid onset of malnutrition
- An inability to store, mobilise, and interconvert nutrient substrates

Malnutrition results in:

- Persistent catabolic state and depletion of body protein stores
- Impaired wound healing
- Increase in infectious complications

Trauma patients frequently have decreased voluntary food intake for many reasons, including nausea, pain, and anxiety. Patients should be **cardiovascularly stable** and also have hydration, acid-base, blood glucose, and electrolyte abnormalities resolved before assisted nutrition is considered. Enteral nutrition is preferred if the gastrointestinal tract is completely or partially functional (i.e. functional enough to allow digestion and absorption), it is tolerated by the patient, and it is not contraindicated. Enteral nutrition is more physiological, safer and less expensive. Ideally avoid syringe feeding as this provides inadequate calorific intake, poses the potential risk of aspiration and is not especially patient friendly. A naso-oesophageal or naso-gastric tube is an option although may not be ideal with facial injuries. An oesophagostomy tube is preferred once the patient is stable for anaesthesia. Parenteral nutrition may be employed in the interim if available.

The calorific requirements should be calculated using the following equation, which approximates the animal's resting energy requirements (RER):

Resting energy requirements (RER) (kcal) = (30 x Bodyweight) + 70

Patients suffering from trauma do not require an illness factor to be added in when calculating daily requirements. RER are more than adequate in most patients. Feeding more than the RER may lead to complications associated with overfeeding.

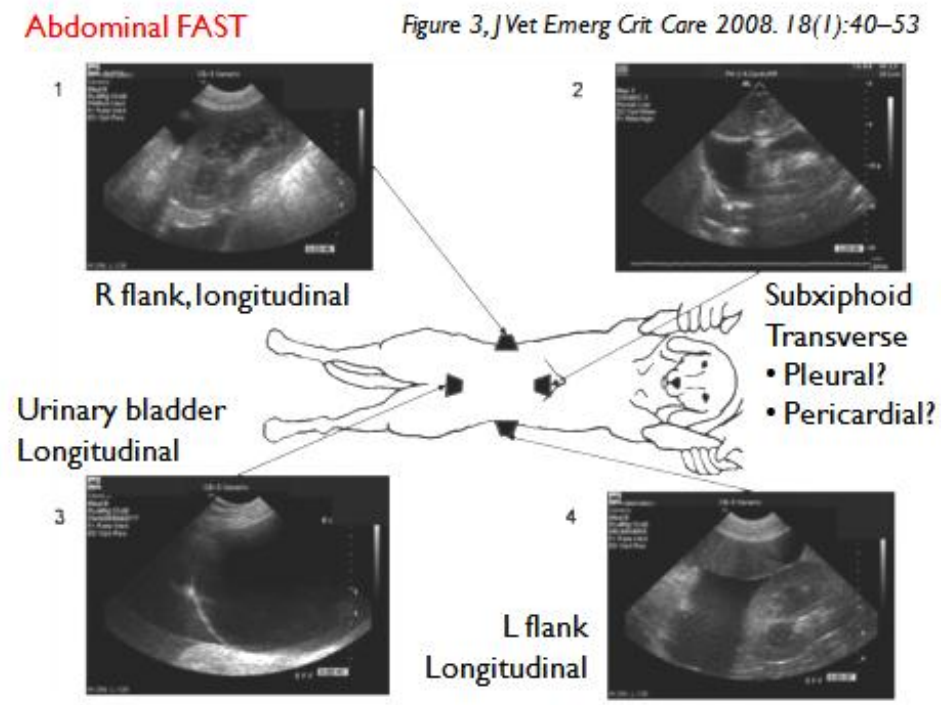
THE ABDOMEN IN TRAUMA

The abdomen should be gently palpated for pain, focal lesions and fluid thrill (fluid wave). If suspected, pain should be categorised as localised or diffuse. It is important when palpating the abdomen to avoid pressing on the spine (e.g. with the thumbs in a standing animal) in order not to misdiagnose spinal pain for abdominal pain.

A significant volume of free peritoneal fluid is necessary for a fluid thrill to be detected and palpation with the patient standing is most likely to be successful. If available, ultrasonography provides a much more sensitive and patient-friendly means of detecting peritoneal fluid as compared to both palpation and radiography. 'Abdominal focussed assessment with sonography for trauma, triage and tracking (AFAST)' has been evaluated in dogs and shown to be extremely useful for detecting peritoneal fluid in both dogs and cats. This technique involves having the patient in left (or right) lateral recumbency and then performing transverse and longitudinal scans at the following sites:

- Just caudal to the xiphoid process
- On the midline over the urinary bladder
- At the left and right flank regions

Peritoneal fluid appears as anechoic-to-hypoechoic material bathing the intra-abdominal structures. Failure to identify fluid using the above protocol means that it is nearly 100% certain that there is none *at that point in time* (i.e. some may be identified on repeat scanning) – this is true even in inexperienced hands as long as the protocol is followed…



In the author's experience, clinically significant intra-abdominal injuries are relatively rare in companion animals that sustain blunt trauma (clearly some animals suffer bite or other wounds to the abdomen that may or may not penetrate full-thickness). However they do occur and most will manifest with free peritoneal fluid that can be detected by ultrasonography as described above; as such the author would strongly encourage free fluid scans to be performed in all patients that have suffered abdominal trauma. Where fluid is detected aspiration should be performed, preferably with ultrasound guidance, to allow analysis – chemistry analysis, cytology etc – to be performed and a diagnosis to be achieved.

It is noteworthy that in humans the concept of 'occult' injuries is described. These are injuries that do not manifest initially with free fluid on ultrasonography but are detected on CT scanning. Detecting injuries at this time allows patients to be monitored with a greater index of concern and/or interventions to be performed as deemed necessary. In veterinary medicine the most realistic approach may be to recognise that free fluid should develop in patients with initially occult injuries. For patients that have a negative initial free fluid scan, on an individual patient basis based on progression, it may therefore be sensible to repeat the scan at least once subsequently (e.g. after 2-4 hours). It is important to note that at this time, to the author's knowledge, there is little or no information published on the prevalence and progression of so-called occult injuries in veterinary patients.

The incidence of peritoneal haemorrhage following blunt trauma is thought to be low; however this may be because for many years we did not look for evidence of haemorrhage until and unless a patient became clinically compromised – it may be more accurate to say that at this time the incidence of *clinically significant* peritoneal haemorrhage is low in patients presenting alive. Thankfully surgical intervention is rarely required for intra-abdominal haemorrhage following trauma in companion animals.

If a free fluid scan is not performed routinely in these patients, then it certainly should be considered in a patient in which hypovolaemia on presentation and assessment of venous packed cell volume/total protein as part of the emergency database suggest haemorrhage has occurred – is the source in the abdomen? Similarly elevations of urea, creatinine or potassium should prompt evaluation for urinary tract rupture (be this peritoneal, retroperitoneal or outside of the abdomen). Likewise for elevated plasma

bilirubin and bile peritonitis and it should always be remembered that gastrointestinal tract rupture resulting in septic peritonitis does occur….

Abdominal fluid parameter	Finding	Suggestive of…
Packed cell volume (PCV)	Similar to peripheral blood PCV	Haemoabdomen
Potassium	Significantly greater than peripheral blood potassium (typically more than 1.4:1)	Uroabdomen
Creatinine	Significantly greater than peripheral blood creatinine (typically more than 2:1)	Uroabdomen
Glucose	Lower than peripheral blood glucose	Septic peritonitis
Lactate	(Significantly) higher than peripheral blood lactate	Septic peritonitis
Bilirubin	Significantly greater than peripheral blood bilirubin	Bile peritonitis – abdominal fluid may also be dark green or black in appearance
Microscopy	Degenerate neutrophils with intracellular bacteria Intestinal/plant/food material Golden refractile pigment	Diagnostic of septic (bacterial) peritonitis Diagnostic of septic peritonitis due to GI leakage Bile peritonitis

Accidental aspiration of blood vessels, liver and spleen can be differentiated in non-coagulopathic animals as blood will normally clot with time, while haemorrhagic fluid will not.

Septic peritonitis

- Cytology gold standard BUT

Peritoneal glucose frequently lower than plasma glucose

- One study showed a gradient of > 2.8mmol/L to be 100% specific for septic peritonitis
- Another study suggested especially if more than 1.1 mmol/L lower in dogs

Uroabdomen

- Measure fluid and concurrent plasma levels of:
  - Urea
  - Potassium
  - CREATININE
- Gradient may be quite small
- May get false positive if animal has just received large volume IV fluids rapidly

Trauma is the most common cause of urinary tract rupture:

- Especially blunt abdominal/pelvic trauma – urine leakage usually from the bladder
- Also iatrogenic – especially urethral rupture during urethral catheter placement

Neoplasia and prolonged urinary tract obstruction are less common causes.

While the loss of large amounts of urine into the abdomen will quickly become apparent, small leaks may take several days to produce clinical signs such as ascites or azotaemia. Note that the presence of a palpable bladder does not rule out bladder rupture, nor does the ability to pass apparently normal streams of urine.

It is important to consider not only the bladder but also the urethra and ureters as potential sites of rupture. Positive contrast radiography is helpful in determining the site of urinary tract rupture:

- Antegrade pyelography or intravenous urography for ureters
- Retrograde urethrocystography for urethra and bladder

Aside from the bladder, surgical repair or management of urinary tract rupture in other sites is often best done in the hands of a specialist surgeon. As such it may be appropriate to refer animals with uroabdomen following initial stabilisation without performing general anaesthesia for contrast radiography.

Urethral rupture is easily missed, as it rarely leads to uroperitoneum due to the retroperitoneal location of the urethra. Instead, such cases often present after a few days with soiling and possible sloughing of the skin in the perineal region caused by leakage of large volumes of urine into the tissues. Retrograde urethrography is a simple technique and should be performed whenever urethral injury is suspected. If a urethral tear is documented, passage of a urinary catheter should be attempted; if this is not possible, a cystotomy tube should be placed surgically (or potentially percutaneously if facilities allow) to provide urinary diversion while the urethra heals. Traumatic avulsion of the ureter is also an uncommon occurrence, but can result in uroabdomen or uroretroperitoneum 12-24 hours after injury. This is most easily identified by performing intravenous urography. Leakage of urine from the kidneys or proximal ureters will result in uroretroperitoneum potentially without uroabdomen.

In most cases of uroperitoneum, the peritoneal fluid will be serosanguineous, which may be misleading. Animals with uroabdomen typically have severe azotaemia, and variable hyperkalaemia may be present. Diagnosis is aided by comparing fluid and blood creatinine and potassium concentrations. Increased abdominal concentrations of creatinine and potassium compared with blood concentrations are suggestive of urinary tract rupture. Creatinine and potassium in peritoneal effusion equilibrate more slowly with the intravascular space and are therefore considered more reliable than blood urea nitrogen for detecting uroabdomen. Note that blood urea, creatinine and potassium may not be increased at presentation, even in the presence of a ruptured bladder and may take up to 24 hours or longer to develop depending on the extent of the leak. Urine is hyperosmotic and therefore will tend to draw fluid from the tissues into the peritoneal cavity, resulting in dehydration and hypovolaemia. As creatinine is a large molecule, it remains trapped within the peritoneum causing on-going fluid shifts.

Management of urinary tract injuries should be delayed until the animal is otherwise stable.

Treatment of urinary tract rupture involves initial stabilisation of the patient's major body systems followed by re-establishment of urine drainage.

Fluid therapy:

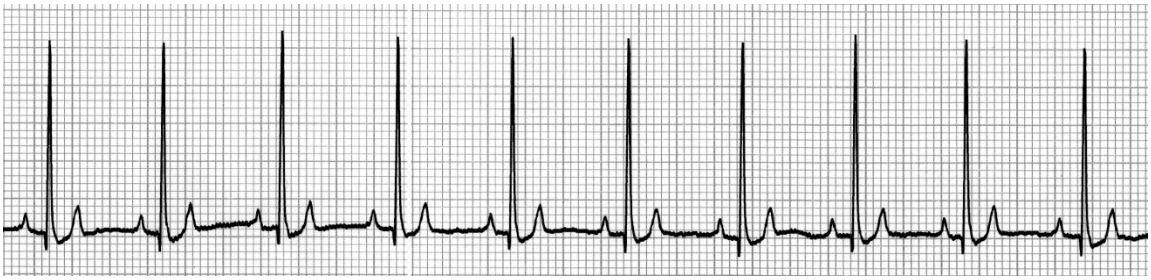
All patients will benefit from intravenous fluid therapy. An *isotonic replacement crystalloid* is the fluid of choice and should be administered as appropriate to restore perfusion. Normal saline (0.9% sodium chloride) has traditionally been recommended due to its lack of potassium. However this fluid may contribute to existing metabolic acidosis and there is no clinically significant difference between the use of 0.9% sodium chloride solution and Hartmann's (buffered lactated ringer's solution) which contains a small amount of potassium.

The priority is very much to start the patient on one or other of these solutions at a rate that is appropriate for the degree of hypovolaemia (if present).

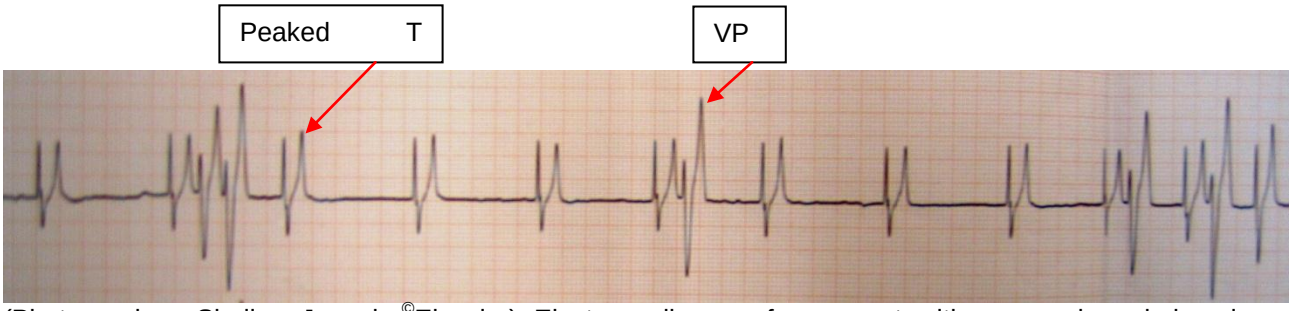
Hyperkalaemia:

Hyperkalaemia occurs mainly due to impaired urinary excretion of potassium. The clinical manifestations of hyperkalaemia reflect alterations in cell membrane excitability and of greatest concern are the potentially life-threatening effects on cardiac conduction:

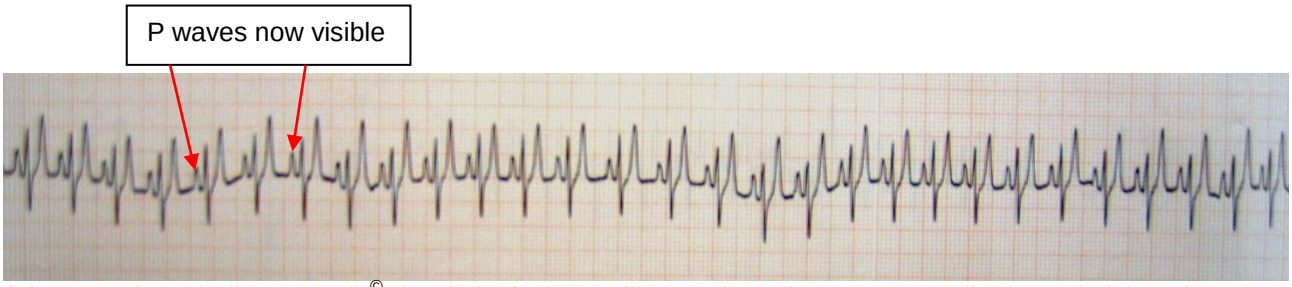
- Sinus bradycardia
- Prolongation of the PR interval
- Widening and bizarre appearance of the QRS complex, including sinoventricular rhythm
- Decreased amplitude, widening or complete absence (atrial standstill) of the P wave
- Peaked/tall T waves



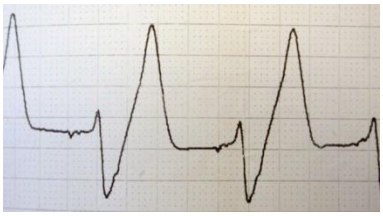
Normal sinus rhythm



(Photograph – Shailen Jasani, ©Elsevier) Electrocardiogram from a cat with severe hyperkalaemia showing atrial standstill with absence of P waves, as well as peaked T waves and ventricular premature complexes (VPCs).



(Photograph – Shailen Jasani, ©Elsevier) Electrocardiogram from the same cat following administration of calcium gluconate. P waves are visible and there are no VPCs in the post-treatment strip.



(Photograph courtesy of Dez Hughes) Atrial standstill and wide-complex sinoventricular rhythm in a cat due to severe hyperkalaemia showing merging of the QRS complex.

The severity of clinical signs resulting from hyperkalaemia does not necessarily correlate with the absolute increase in serum potassium concentration. The treatment required is therefore dependent not just on the serum potassium level but also very importantly on whether the hyperkalaemia is assessed to be clinically significant.

Treatment is directed at improving the patient's clinical status and not at the serum potassium concentration per se.

In some cases, intravenous fluid therapy and restoring urine drainage is all that is necessary for hyperkalaemia to resolve. Further treatment options for hyperkalaemia are summarised in the table below:

Agent	Dose / route	Comments
10% calcium gluconate	0.5-1.0 mL/kg i.v. bolus over 30-60 secs*	Rapid onset of action (can be a few secs) and first line choice in a crisis Short duration of action (often 10-15 min) Monitor ECG during administration Does not lower serum potassium concentration but restores normal cell membrane excitability Will also address possible ionised hypocalcaemia Bolus can be repeated while other measures are used to directly reduce hyperkalaemia
Neutral (Regular, Soluble) insulin **Glucose solution**	0.25-0.5 IU/kg i.v. 0.25-0.5 g/kg i.v.	Slower onset of action (can be more than 15 min) Lowers serum potassium concentration by moving potassium into cells Intravenous glucose supplementation typically required for several hours (monitor and adjust accordingly)
Sodium bicarbonate	1-2 mmol/kg slow i.v. (repeat if necessary)	Also lowers serum potassium concentration by moving potassium into cells Effect can persist for several hours Access to on-site acid-base analyser much preferred

(* Note: when calcium gluconate is given intravenously for the treatment of hypocalcaemia, it should be given slowly (e.g. over 20-30 min) as a constant rate infusion. However this is clearly not a rational approach in a patient with life-threatening hyperkalaemia and calcium gluconate is administered much more quickly in these cases.)

If serum electrolytes cannot be measured but clinically significant hyperkalaemia is suspected on the basis of electrocardiography or perhaps physical examination alone, the author firmly believes that empirical use of calcium gluconate, and potentially insulin with glucose, is appropriate. This treatment can be life-saving and is unlikely to cause any significant harm if not required.

Analgesia:

All of the causes of urinary tract rupture are likely to be associated with some degree of pain and as always, analgesia should be administered as part of the initial stabilisation. Pure opioids are the usual first choice and non-steroid anti-inflammatory drugs (NSAIDs) are contraindicated in animals that are hypovolaemic or azotaemic.

Re-establishing urine drainage:

The approach to re-establishing urine drainage depends on the site of urine leakage as well as the severity of the animal's condition including other injuries and the extent of initial stabilisation required. The decision to be made essentially is between:

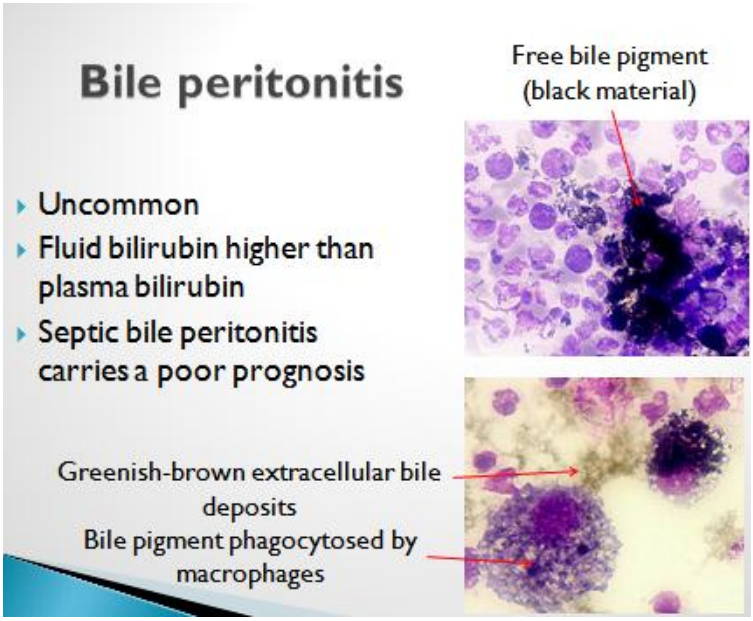
- Initial stabilisation followed by immediate diagnostic imaging to identify the site of leakage and surgical intervention, or
- Initial stabilisation that includes some form of temporary urinary drainage, and delayed surgical intervention

Bladder rupture often requires surgical intervention, although small leaks may heal with continuous decompression provided by a urinary catheter and drainage collection system. Urethral rupture is often managed by placing a urethral catheter and leaving it in situ for several days to act as a stent for urethral healing. Aside from the bladder, surgical repair or management of urinary tract rupture in other sites is often best done in the hands of a specialist surgeon. As such it may be appropriate to refer animals with urinary tract rupture following initial stabilisation without performing general anaesthesia for contrast radiography to identify the site of leakage.

In animals with uroabdomen that are more severely affected or if surgical intervention is to be delayed – for example due to referral – consider abdominal drainage. This will prevent reabsorption of electrolytes and waste products and help stabilise the patient for anaesthesia. A variety of drains have been used ranging from closed systems (e.g. proprietary peritoneal dialysis catheters) to over-the-needle catheters (e.g. central venous catheters, chest tubes) to Penrose drains. Omental occlusion of drain holes is a potential problem that can render drains dysfunctional.

If a suitable drain cannot be placed, needle abdominocentesis may be performed although repeated needle abdominocentesis is not ideal as a short-term management strategy.

Bile peritonitis:



Bile peritonitis occurs due to leakage from or rupture of the gallbladder and/or biliary tract. This may result from:

- Blunt or penetrating abdominal trauma; also iatrogenic
- Inflammation with or without infection, e.g. necrotising cholecystitis
- Obstruction, e.g. cholelithiasis, neoplasia

Clinical jaundice and hyperbilirubinaemia may be identified in animals with bile peritonitis, depending on the cause, along with varying degrees of cardiovascular compromise. Marked elevations in liver enzymes may also be seen.

Peritoneal fluid may be greenish in appearance and clusters of golden refractile pigment are identified. Septic bile peritonitis carries a worse prognosis. The bilirubin concentration of the peritoneal fluid is considerably higher than the plasma bilirubin concentration.

Bile peritonitis is a surgical emergency but the author cannot stress strongly enough the importance of referral of these cases to a specialist surgeon. Biliary tract surgery is highly challenging with the potential for disastrous consequences in inexperience hands.

Blunt trauma may result in body wall rupture with or without herniation of body organs. Blunt trauma involving ventral, femoral, paralumbar and prepubic sites may be detectable by careful palpation or may be apparent on radiographs. Can also be detected by ultrasonography – scanning a superficial structure like the body wall can be significantly enhanced by using a standoff; if a standoff pad is not available, a glove filled with ultrasound gel can work very well for this purpose. Strangulation of organs is uncommon, enabling the timing of these repairs to be semi-elective.

Summary approach to the trauma patient

Perform major system examination (primary survey) and consider especially:

Is the patient hypoperfused due to hypovolaemia; if so the patient needs immediate intravenous fluid resuscitation.
A replacement isotonic crystalloid is the first choice in most cases.

Is the patient dyspnoeic? Provide oxygen supplementation.
Is dyspnoea due to thoracic injury?
Pulmonary contusions (lung sounds louder/harsher than normal +/- crackles) and pneumothorax (lung sounds dull dorsally) are the two most common causes; if pneumothorax is suspected or you are not sure, consider thoracocentesis.

Provide analgesia.
Ideally use a pure opioid (e.g. morphine, methadone) as buprenorphine has a slower onset of action
NB. NSAIDs are contraindicated in hypovolaemic or dehydrated patients – if in doubt, do not give them early on! They are likely to be helpful though in trauma patients due to their anti-inflammatory effects so keep them in mind for later.
Can you employ topical local anaesthesia for wounds etc?

What about scanning the abdomen for free fluid (FAST)?
Remember with traumatic brain injury, the priority is to ensure the brain receives a good supply of well-oxygenated blood: correct perfusion, ensure oxygenation.

What about an emergency blood database of some sort – e.g. PCV/total protein, urea, glucose…
Any indication for more extensive chemistry testing – e.g. creatinine, electrolytes, bilirubin?

Perform a more thorough examination (secondary survey):

Does the patient need any specific interventions for injuries you have identified? If so, remember, think *risk-benefit assessment at all times*.

Going forward….

Maintain a liberal approach to opioid analgesia and consider NSAIDs at the appropriate time.
Continue fluid therapy including to correct any pre-existing dehydration (unlikely in peracute/acute trauma patients).
And, remember that nutrition is very important in the trauma patient:
Don't start feeding until and unless the patient is cardiovascularly stable, and withhold if there are neurological or other contraindications.
Consider a feeding tube (syringe feeding is very unlikely to meet calorific requirements, is stressful, may cause aspiration etc.)

THORACIC INJURIES

Thoracic injuries following blunt trauma include:

- Pneumothorax
- Pulmonary contusions
- Haemothorax
- Rib fractures and flail segment
- Diaphragmatic rupture
- (Airway rupture)

In the author's experience, pulmonary contusions and pneumothorax are the most common.

It is often possible on the basis of observation and auscultation to make reliable judgements as to the most likely cause of dyspnoea in trauma patients and thoracic radiography is not recommended in distressed, painful or unstable patients. This is especially applicable to dyspnoeic cats as they may decompensate more easily than dogs.

Where significant pleural space disease (pneumothorax, haemothorax) is suspected, thoracocentesis (see below) may be both diagnostic and therapeutic. This procedure is relatively simple and clinically significant complications are rare. It is recommended to perform thoracocentesis rather than subject unstable trauma patients to radiography initially. Ultrasonography is a quick and less stressful way of confirming haemothorax and can also be used to detect pneumothorax – although the latter requires much more experience and expertise.

If radiography is performed, everything should be prepared before bringing the patient to the radiography room and minimal restraint should be used. If tolerated, oxygen supplementation is provided by mask or flow-by. The intention here is to produce an image that allows the anatomical area affected to be identified and not to produce the perfect thoracic radiograph. Even a rotated dorsoventral radiograph can provide more than enough information for the patient's initial management. A dyspnoeic animal should never be restrained in dorsal recumbency and it is typically best to minimise the amount of time spent in lateral recumbency.

Pneumothorax

Pneumothorax is essentially the accumulation of air in the pleural cavity; lung sounds will be dull dorsally although in more severe cases they may be quieter more diffusely. **Closed** pneumothorax usually results from leakage of air secondary to a lesion within the lung parenchyma, although it may also occur for example due to damage to the airways or oesophagus. **Open** pneumothorax involves loss of integrity of the thoracic wall (e.g. following penetrating trauma) while a **tension** pneumothorax occurs if a one-way valve is formed at the site of air leakage such that air taken in during inspiration leaks into the pleural space but cannot be expelled. The result is a rapid and potentially life-threatening increase in intrapleural pressure (essentially a pleural compartment syndrome) with severe respiratory and cardiovascular compromise and immediate thoracocentesis followed by continuous drainage is required. In the author's experience tension pneumothorax is rare.

Thoracocentesis (see below) is by no means required in all cases of traumatic closed pneumothorax and the air will be resorbed over days to weeks. The decision to perform thoracocentesis should be guided by whether the pneumothorax is thought to be compromising respiration in a clinically significant way.

Clinical improvement following thoracocentesis is generally associated with aspiration of 20-30 ml/kg or more of air although improvement may be noted with removal of smaller volumes in animals with other concurrent thoracic injuries, most commonly pulmonary contusions. In the author's experience thoracocentesis does not usually need to be performed more than twice for traumatic pneumothorax and chest drain placement is infrequently indicated. If a chest drain is needed, bear in mind that a wide bore drain is not needed for draining air and a small bore drain, for example one designed to be placed percutaneously using the Seldinger technique, can be successfully employed. The need for surgical intervention is rare.

As well as occurring as a result of trauma (blunt or penetrating), pneumothorax may also be *spontaneous* following rupture of pulmonary lesions (e.g. bullae, tumours, sub-pleural blebs) or iatrogenic (e.g. following thoracocentesis or fine needle lung aspiration). Pneumothorax that occurs spontaneously is often more severe than following trauma and chest drain placement and surgical intervention are more likely to be indicated.

Pulmonary contusions

Pulmonary contusions represent areas of alveolar and interstitial haemorrhage and oedema and probably represent the most common thoracic injury in dogs and cats following trauma. Not all affected animals develop associated clinical signs and pulmonary contusions may occur with or without other thoracic injuries. Clinical signs may develop acutely or over several hours and lung auscultation will reveal louder lung sounds, either harshness or crackles; occasionally severe contusions resulting in consolidation of a lobe occur and then lung sounds may be quieter over the affected area as there is no air movement through the consolidated lobe. It can be difficult to hear crackles in animals with a low tidal volume, e.g. during panting or tachypnoea.

Radiographic changes may lag behind clinical signs by up to 24 hours. Radiographic abnormalities (patchy or diffuse alveolar or interstitial lung changes) may persist for a variable period of time despite clinical improvement. There is no specific treatment for pulmonary contusions and management typically involves oxygen supplementation, cage rest, analgesia as indicated for concurrent injuries, minimal stress and time.

Intravenous fluid therapy does not have to be withheld but caution is advised. Hypovolaemia is common in patients with pulmonary contusions at time of presentation and the aim of fluid therapy should be to restore acceptable tissue perfusion while avoiding excessive fluid administration. It may be acceptable in some cases to leave the patient mildly hypovolaemic rather than risk worsening pulmonary contusions; current evidence in humans seems to support the notion of keeping patients with parenchymal lung injury 'on the dry side' without leaving them hypoperfused or allowing them to become dehydrated.

In the author's experience clinical signs associated with pulmonary contusions resolve in most cases (generally within 2-7 days) but their presence may necessitate that investigation and management of other non-life-threatening injuries (e.g. long bone fractures) be postponed until respiratory status has improved adequately.

Diuretics are not recommended in the treatment of pulmonary contusions and are contraindicated in hypovolaemia. There is probably a rationale to administer one single low dose of furosemide (e.g. 0.5 mg/kg IV to a cat, 1 mg/kg IV to a dog) to these patients as furosemide is thought to have a number of different mechanisms of action that may potentially improve oxygenation beyond just the clearance of congestive oedema. However these patients should not be treated in the same way as a patient with pulmonary oedema from congestive heart failure and remember that furosemide is contraindicated in the presence of hypovolaemia or dehydration.

The incidence of bacterial pneumonia following pulmonary contusions is very low and the indiscriminate use of antibiotics in these cases is not recommended.

Haemothorax

Haemothorax is accumulation of blood in the pleural space. It is not uncommon to detect a small volume of pleural effusion following blunt thoracic trauma that is presumed to be secondary to haemorrhage. However, in the author's experience, clinically significant haemothorax is relatively rare; it would be associated with lung sounds that are dull ventrally. The pleural space can accommodate a considerable volume of blood without causing clinically significant respiratory compromise and haemothorax should not be drained unless it is thought to be significantly contributing to dyspnoea; the blood will be resorbed over several days. This is especially important as anaemia secondary to haemorrhage is relatively common following trauma, particularly in cats. Potential secondary complications of having blood in the pleural space for a few days are infection and a retained fibrohaemothorax causing lung restriction; in humans the former is considered rare and the latter even rarer – to the author's knowledge there is no veterinary data available on this.

If haemothorax is drained, it is sensible to remove as small a volume (e.g. 20 ml/kg) as possible that is expected to allow clinical improvement in respiratory status (i.e. so that the remainder may be resorbed). Surgical intervention for traumatic haemothorax is seldom required.

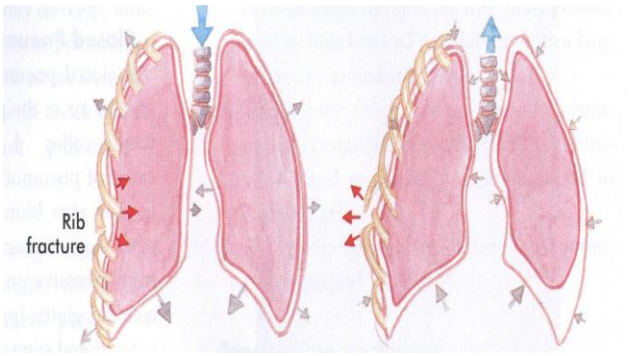
Aside from trauma, haemothorax may also occur as a result of severe coagulopathy, in particular due to anticoagulant rodenticide intoxication; other less common causes are neoplasia, pulmonary thromboembolism, lung lobe torsion and iatrogenic causes.

Rib Fractures and Flail segment

It is very unusual for rib fractures to occur in dogs and cats without at least one other significant thoracic injury.

Rib fractures are often only diagnosed radiographically and they typically do not require specific intervention. Rib fractures are however reported to be very painful and a liberal approach to analgesia is therefore recommended in these cases. Ventilation may be compromised in painful animals especially if rib fractures are present.

A flail segment may be created if 2 or more adjacent ribs are fractured both dorsally and ventrally, i.e. so that the segment is no longer stabilised by attachment to the sternum or spine. This flail segment then moves paradoxically in relation to the rest of the chest wall during respiration, i.e. the flail segment moves in on inspiration and out on expiration.



Although large flail segments may cause hypoventilation, flail segments typically do not contribute significantly to dyspnoea and stabilisation is not usually indicated. Their significance lies more in the fact that they are typically associated with other significant thoracic injuries, especially pulmonary contusions, and that they are often very painful. These factors are more likely to be responsible for any respiratory compromise identified. Flail segments are rare in dogs and cats.

Traumatic diaphragmatic rupture

Although listed here as a thoracic injury, acquired diaphragmatic rupture typically occurs as a result of blunt abdominal trauma. Circumferential and right-sided diaphragmatic tears are most common in dogs and cats and the liver is reportedly the most commonly herniated organ. In addition, the stomach, small intestine and spleen are often involved in left-sided hernias and the small intestine and pancreas in right-sided hernias.

Respiratory dysfunction is a common presenting sign of traumatic diaphragmatic rupture although it may be minimal or even absent in a number of cases. The aetiology of respiratory compromise is usually multifactorial and may include:

- Loss of mechanical function of the diaphragm (most important muscle for inspiration)
- Pleural space-occupying material causing lung lobe compression and progressive atelectasis. This material predominantly constitutes abdominal organs but possibly also air (pneumothorax) and fluid (haemothorax, transudate)
- Concurrent thoracic injuries – e.g. pulmonary contusions, rib fractures
- Other factors such as pain and hypovolaemia

In some animals with diaphragmatic rupture acute respiratory decompensation may occur as a result of accumulation of a significant volume of abdominal viscera in the pleural space. In such cases, holding the animal upright to allow abdominal contents to return to the peritoneal cavity is potentially life-saving.

Diagnosis

Radiography is commonly used in animals with suspected diaphragmatic rupture and consistent findings include:

- Complete or partial loss of the diaphragmatic line
- Mediastinal shift
- Obscuring of the cardiac silhouette
- Cranial displacement of abdominal viscera and gas shadows

Orthogonal views are recommended but diagnosis may be difficult for example if displaced viscera are obscured by pleural fluid or in the absence of visceral displacement. Positive contrast gastrography or peritoneography may be required in these cases.

Timing of radiography must be carefully chosen and it is not a priority in unstable patients in which the additional stress may cause rapid decompensation.

If facilities and expertise allow, **ultrasonography** provides an alternative, reliable and less stressful means of diagnosing diaphragmatic rupture. This may be especially helpful earlier on in unstable patients or in those with significant pleural effusion obscuring radiographic detail.

Timing of surgical intervention

The timing of surgical intervention for diaphragmatic rupture has been the subject of some debate. Some authors recommend a delay of more than 24 hours following trauma to ensure adequate time for stabilisation of other concurrent conditions (e.g. hypovolaemic shock) and injuries (e.g. pulmonary contusions) thereby reducing the risks associated with general anaesthesia and major surgery. However current literature suggests that surgical intervention within 24 hours of admission in stable patients does not worsen the prognosis and therefore timing of surgical intervention should be made on an *individual case basis.*

Diaphragmatic rupture repair is more likely to be associated with a successful outcome when performed as an elective procedure with adequate staffing and time for thorough planning and preparation.
If a prolonged period of stabilisation is provided, patients must be monitored very closely for any deterioration in clinical status that may for example occur as a result of gastric entrapment and distension or strangulation of displaced intestine. More emergent surgical intervention may then be required and needle decompression of the stomach is appropriate in a severely compromised patient while being prepared for surgery.

A thoracostomy tube may be placed intraoperatively prior to closure of the diaphragmatic defect to allow residual air to be removed from the pleural cavity following repair. Bear in mind that a wide bore drain is not needed for draining air and a small bore drain, for example one designed to be placed percutaneously using the Seldinger technique, can be successfully employed. In order to avoid re-expansion pulmonary oedema (thought to be associated with cytokines and inflammation) some authors recommend only aspirating a proportion of the residual air immediately postoperatively, i.e. as much as is necessary for satisfactory ventilation to occur, with the remainder being removed over the subsequent 8 -12 hours. A thoracostomy tube will also allow monitoring for complications such as pneumothorax or haemothorax as well as repeated administration of local anaesthesia.

If a thoracostomy tube is not placed, it is important to remove residual air from the pleural cavity by other means to prevent respiratory compromise. This is ideally done through the diaphragm before the final suture is tightened but may be achieved via thoracocentesis during recovery.

Thoracocentesis

Equipment

Equipment list:

- Clippers
- Surgical scrub materials
- Sterile gloves
- Needle:
    - Butterfly needle: typically 21-23 gauge for cats and small dogs; or
    - Over-the needle intravenous catheter and extension tubing: 14-20 gauge depending on patient size
    - Appropriate needle or catheter size depends on patient size and nature of material to be aspirated
- Sterile 3-way tap or 'centesis valve'
- Syringe: typically 20 ml for cats and small dogs, 60 ml for larger dogs
- Sample pots: sterile EDTA, serum and additive-free containers
- Kidney dish, jug or similar
- Three people (ideally)

Procedure

Procedure:

1. Allow the patient to stand or to sit in sternal recumbency as preferred, and restrain gently; minimal restraint often works best
    - Lateral recumbency may be acceptable for pneumothorax
2. Provide flow-by/mask oxygen supplementation (if tolerated)
3. Clip a patch of fur in the 7^{th}-9^{th} intercostal space on both sides of the chest and scrub the area on one side
    - The choice of which side to start with should be guided by auscultation, ultrasonography (or a dorsoventral radiograph if one has been taken)
    - If pneumothorax is suspected, use an area in the dorsal third of the thorax
    - If pleural fluid is suspected, use an area in the ventral third of the thorax
    - If pleural air or fluid has not yet been confirmed, use an area half-way up the thorax
4. Wearing sterile gloves attach the butterfly needle to the 3-way tap or centesis valve and attach the 3-way tap/centesis valve to the syringe. Note that the 3-way tap/centesis valve is therefore located away from the patient. In larger animals an intravenous catheter is attached to extension tubing that is then attached to the 3-way tap/centesis valve.
5. Insert the needle gently but swiftly into the pleural cavity at a right angle to the chest wall and with the bevel facing dorsally; apply gentle suction to the syringe as the needle is inserted:
    - The needle should be inserted just in front of (cranial to) the rib to avoid hitting the intercostal nerves and vessels that lie behind/caudal to each rib
    - Once in the pleural cavity the needle can be angled caudally or ventrally to lie flat against the thoracic wall thereby keeping the needle away from the lung surface
6. Continue to aspirate until negative pressure is reached or it feels like the lung is being scratched by the needle.
    - With (pocketed) pleural fluid in particular, it is often necessary to reposition the needle (and sometimes the patient) to remove as much as possible
7. Record the total volume of air or fluid removed from the pleural cavity; aseptically transfer samples of pleural fluid into the sample pots

8. Aspiration may need to be repeated on the other side of the thorax. This will depend on:
    - Whether the patient has improved adequately after aspiration on the first side
    - Individual patient considerations, e.g.
        - Is residual fluid or air likely to be reabsorbed?
        - Is additional fluid or air likely to form?
        - Are there other thoracic abnormalities (e.g. pulmonary contusions) that would make the patient less able to cope with residual fluid or air?
        - How well did the patient tolerate the procedure on the first side?

Complications/Notes

Complications:

Clinically significant complications associated with thoracocentesis are rare; it is typically a simple and safe procedure that is potentially life-saving. Reported complications include pneumothorax and haemorrhage from laceration of the intercostal vessels.

Notes:

Excessive manual restraint is contraindicated in all dyspnoeic animals including those in which thoracocentesis is being performed; this is especially the case for cats

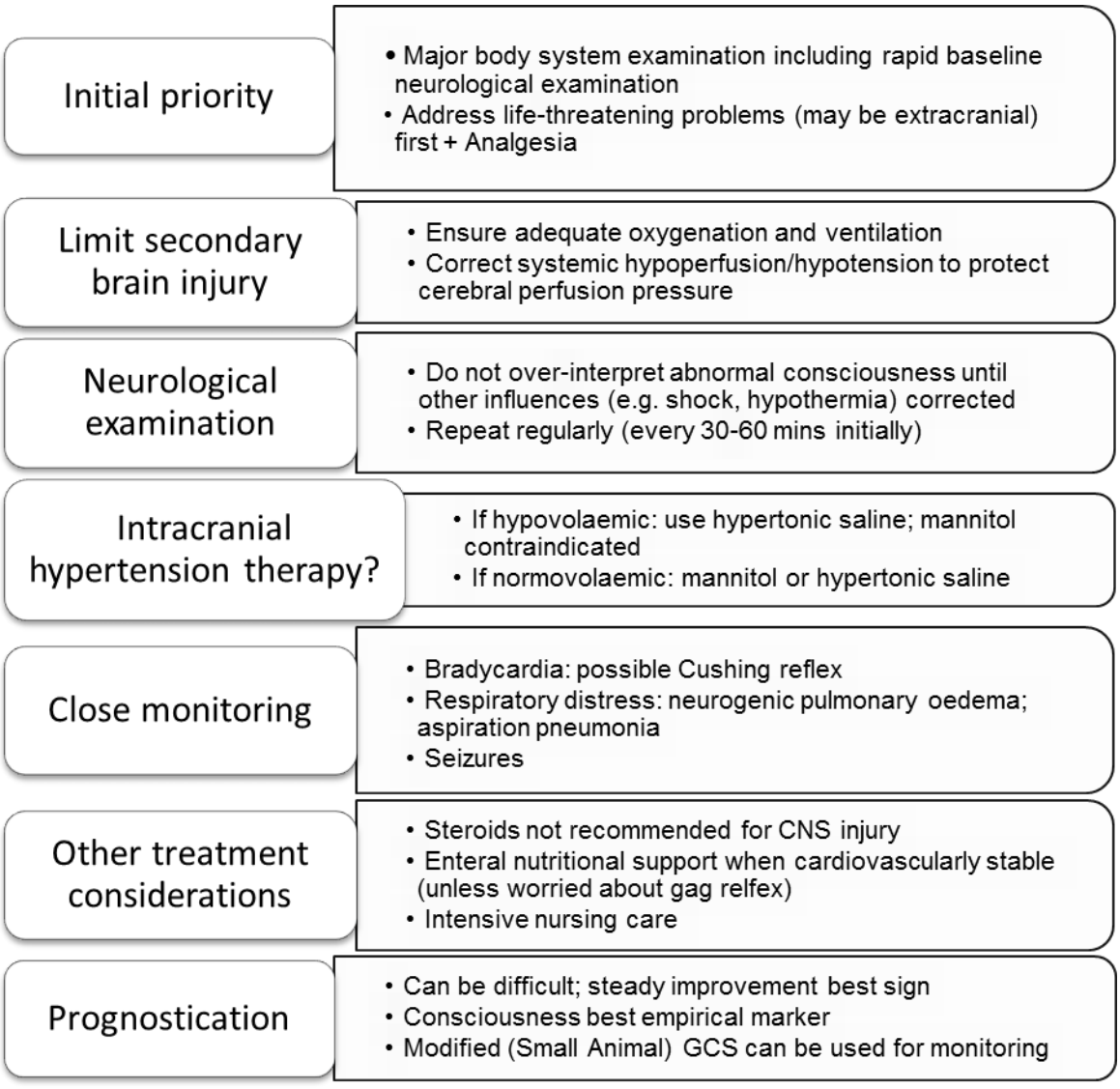
Many animals with severe dyspnoea due to pleural space disease will allow thoracocentesis to be performed with minimal manual restraint and without any chemical restraint. It is important to allow the patient to direct the procedure to some extent – work with the patient rather than try and force him/her into submission!

A low dose of a pure opioid (e.g. 0.1 mg/kg methadone) may be administered prior to the procedure both as an anxiolytic and to provide some analgesia. However this delay is not appropriate in patients with very severe dyspnoea or considered to be in imminent danger of death.

Conservative sedation (e.g. 0.3 mg/kg butorphanol ± 0.01 mg/kg acepromazine) may be required to allow thoracocentesis to be performed in one smooth and successful procedure and judicious chemical restraint is safer than excessive manual restraint in all cases. Drugs with potentially significant respiratory or cardiovascular depressive effects at clinical doses must be avoided. Clearly drug therapy in the trauma patient needs to account for analgesia as indicated and this may in itself facilitate thoracocentesis.

It is generally necessary to remove approximately 10-20 ml/kg or more of air or fluid from the pleural cavity to make a significant difference to an animal's breathing. In animals with multiple causes of respiratory compromise (e.g. concurrent traumatic pneumothorax and pulmonary contusions), it should be noted that removal of smaller volumes of air or fluid may result in clinical improvement as even some increased expansion may be of benefit to already compromised lungs.

TRAUMATIC BRAIN INJURY (Closed head injury)



Rational approach

As always, the priority in the management of traumatic brain injury (TBI) is to address potentially life-threatening problems first and it must be remembered that these may not be intracranial, i.e. some animals with TBI will have more pressing extracranial injuries. In reality however, all these abnormalities will be addressed to some degree by any interventions provided as they do not exist in isolation.

The underlying injuries that result from head trauma can be separated into two categories: primary injury and secondary injury. Primary injury (e.g. concussion, contusion – parenchymal haemorrhage and oedema, laceration; extra-axial haemorrhage may not be as rare as previously thought) occurs as an immediate result of the traumatic event and nothing can therefore be done about this.

The prime aim in the management of traumatic brain injury (TBI) is to limit secondary brain injury that may occur as a result of various mechanisms including:

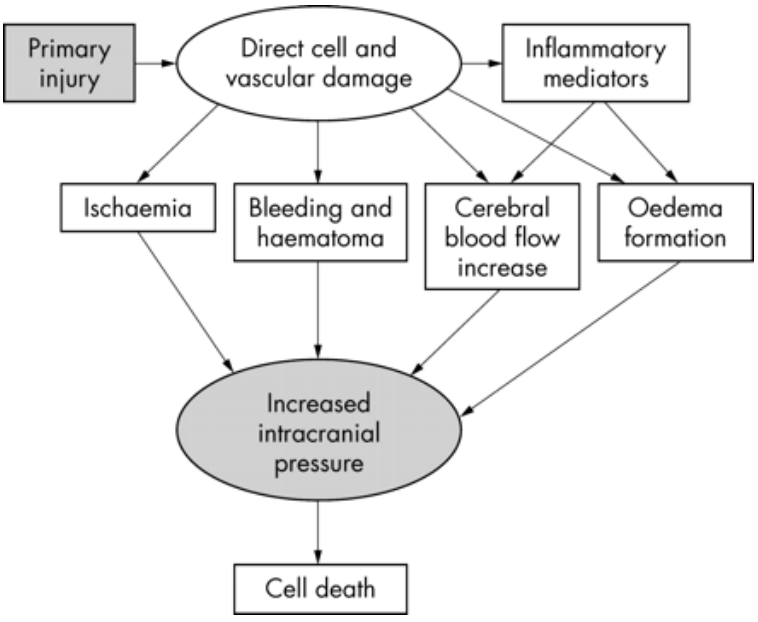
- Hypoxia
- Ischaemia due to hypoperfusion
- Raised intracranial pressure (ICP)
- Active haemorrhage
- Compromise to blood brain barrier

Secondary injury is largely mediated through:

- Increased activity of excitatory neurotransmitters
- Generation of reactive oxygen species (ROS)
- Production of pro-inflammatory cytokines
- All can contribute to neuronal cell damage and possibly cell death

Oxidative damage:

- ROS preferentially damaging to cell membranes containing high levels of polyunsaturated fats and cholesterol
- Brain tissue lipid-rich so particularly sensitive
- Thought to play a major role



Earlier discussions of TBI tended to focus on the detrimental effects of raised ICP via impairment of cerebral blood flow and the potential for brainstem compression and herniation. However secondary brain

injury is significantly perpetuated by hypoxia and systemic hypoperfusion; it is also affected by hypo/hypercapnia, hyperthermia, and hypo/hyperglycaemia. **The priority is therefore to ensure that the brain receives an adequate supply of well-oxygenated (but not excessively oxygenated!) arterial blood in a patient with adequate ventilation.** Treatment for possible raised ICP is just one part of this therapy.

Ensuring adequate oxygenation and ventilation

Cerebral hypoxia must be avoided as much as possible and this is partly dependent on there being sufficient oxygen in the systemic arterial circulation. Ideally this is monitored using arterial blood gas analysis to measure arterial partial pressure of oxygen (P_aO_2). In humans, based on some clinical data and some animal experimental data, there is a suggestion that the aim of oxygen supplementation should be to find a balance between too little and too much. Too little, i.e. hypoxia, is clearly likely to be harmful but there is also increasing evidence that, as has been suggested/shown for other organs such as the lungs, too much oxygen, i.e. hyperoxia, may also be harmful by promoting oxidative injury. As described above, oxidative injury is one of the main mechanisms of secondary injury in TBI.

Arterial blood gas analysis is unavailable in many clinics and pulse oximetry must therefore be used. Remember that this measures the saturation of haemoglobin with oxygen (S_pO_2) and once a patient reaches 100% saturation, it is not possible to know how much additional oxygen is being taken on board (dissolving in the plasma, contributing to oxygen partial pressure). A patient with an S_pO_2 of 100% may have a P_aO_2 of for example 120 mmHg or 600 mmHg! In other words for our TBI patient, once we have achieved an S_pO_2 of (close to) 100%, it will be difficult to tell whether we are 'overdoing it' in terms of supplementation in the absence of arterial blood gas analysis.
If pulse oximetry is not available then essentially the take home message is that all patients with significant TBI should receive empirical oxygen supplementation for a timeframe that is decided based on the individual patient.

As well as considering the patient's oxygenation status, their carbon dioxide status is something else that ideally should be considered and is predominantly dependent on ventilation. In the past hyperventilation was recommended as a treatment for raised intracranial pressure because it lowers arterial CO_2 levels which causes cerebral vasoconstriction; it can therefore potentially reduce intracranial pressure. However it is now believed that this cerebral vasoconstriction is harmful, considerably more so than the raised ICP it was attempting to address, because it reduces cerebral perfusion. Hyperventilation may worsen morbidity and mortality and it is therefore no longer recommended. Hypercarbia is also to be avoided and the recommended target is a P_aCO_2 or end-tidal CO_2 within the normal range (approximately 35-45 mmHg). This will require either access to arterial blood gas analysis or a capnograph/capnometry. Moreover unless the patient is intubated our ability to influence ventilation is relatively limited. Nevertheless any processes that may interfere with adequate ventilation (e.g. pneumothorax; pain) must be addressed appropriately.

Ensuring adequate cerebral perfusion

Cerebral blood flow, and hence oxygen and nutrient delivery to the brain and carbon dioxide removal from the brain, is driven by the pressure gradient between mean arterial pressure (MAP) and intracranial pressure (ICP):

CPP = MAP – ICP
(CPP = cerebral perfusion pressure)

Normal homeostatic mechanisms protecting cerebral blood flow may be lost in TBI and cerebral blood flow (CBF) becomes largely dependent on systemic blood pressure. The normal brain is capable of maintaining a constant CBF over a systemic MAP range of 50–150mmHg. TBI may compromise this

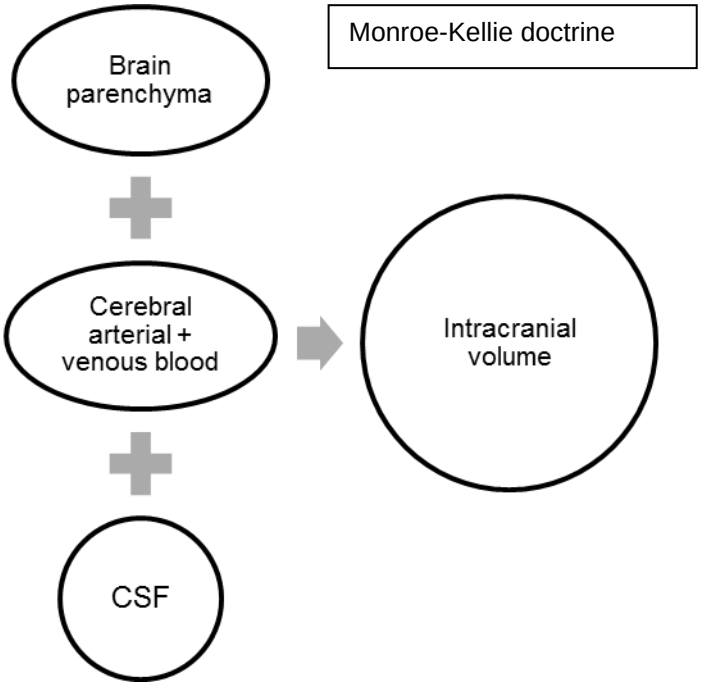
cerebral pressure autoregulation and CBF becomes even more dependent on CPP. Even small decreases in CPP can lead to changes in CBF and result in ischemic injury to the brain parenchyma. **Maintenance of an adequate CPP is a cornerstone of modern brain injury therapy.**

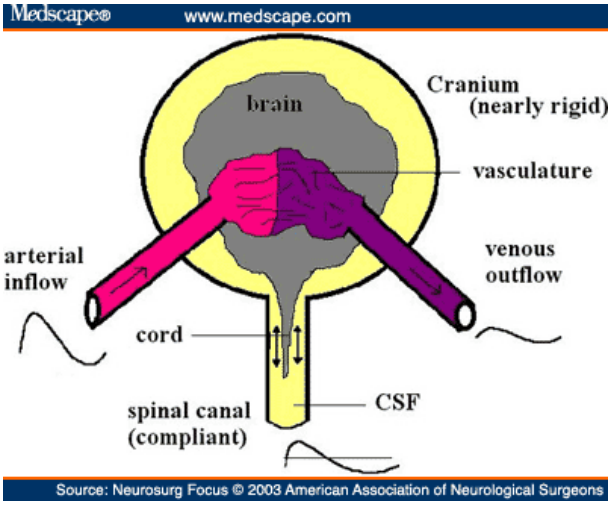
Mean arterial pressure (MAP):

The head trauma patient may well present with systemic hypotension (i.e. decreased MAP) especially if there has been multisystem trauma with significant blood loss. Traumatic brain injury may also result in a systemic inflammatory state with subsequent systemic vasodilation that may cause or contribute to hypotension. Mean arterial pressure can be measured non-invasively using an oscillometric device and a reasonable target MAP is 80 mmHg. If only Doppler sphygmomanometry is available, then a systolic blood pressure of 100 mmHg is considered equivalent.

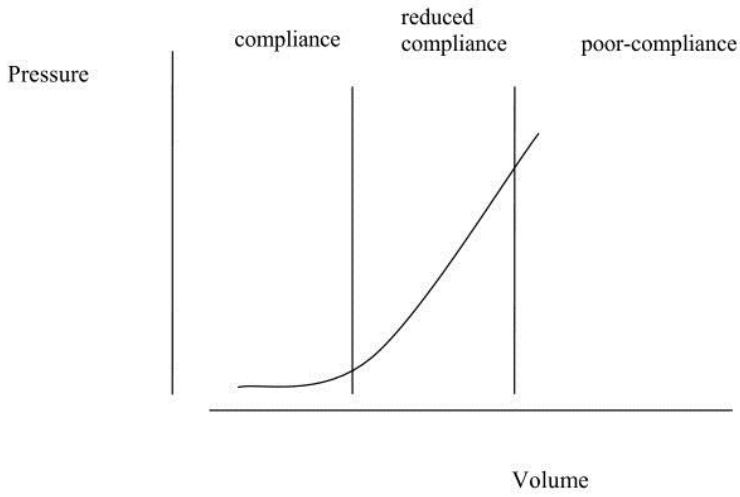
Regardless of whether blood pressure monitoring is or is not available, it is essential to ensure that hypoperfusion identified on the basis of physical examination is corrected. Blood pressure monitoring is not a substitute for regular assessment of physical perfusion parameters.

Raised intracranial pressure:

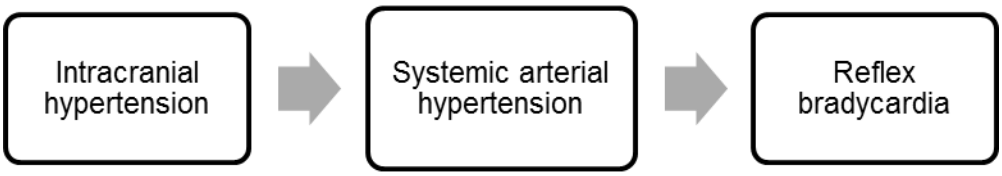




The Monroe-Kellie doctrine states that intracranial volume is equal to the volume of the brain parenchyma plus the volume of the cerebral arterial and venous blood plus the volume of the cerebrospinal fluid (CSF). Intracranial pressure is the pressure exerted between the incompressible rigid skull and these 3 intracranial compartments. There is normally a balanced dynamic equilibrium such that if the volume of one component increases, the volume of one or more of the other components must decrease (Monroe-Kellie hypothesis) or ICP increases – this is known as *intracranial compliance* and it occurs by fluid shifts in the brain vasculature and CSF pathways. However intracranial compliance has limitations and its efficacy decreases as ICP increases. If ICP increases beyond the limits of compensation, cerebral perfusion is compromised and ischaemia occurs; eventually global brain ischaemia and subsequent brain death results. This is a possibility in closed head injury where the skull is intact.



Direct measurement of ICP is not practical in general in veterinary medicine at this time and is restricted to research and perhaps a few referral centres. Therefore *the presence of elevated ICP is inferred*. The cerebral ischaemic response (Cushing reflex) is the most specific marker available – intracranial hypertension causes systemic hypertension and a consequent reflex bradycardia.



These findings should prompt aggressive treatment for raised intracranial pressure as the Cushing reflex occurs late and signals possible/probable life-threatening intracranial hypertension. Other less specific findings that may suggest intracranial hypertension include otherwise unexplained deterioration of mental status, dilated non-responsive pupils, loss of physiological nystagmus and decerebrate posturing.

Neurological examination

Hypoperfusion can have a significant effect on neurological status as can hypothermia and hypoxaemia. These abnormalities must therefore be addressed before drawing conclusions about the patient's level of consciousness.

Following initial stabilisation perform a more in-depth neurological assessment
Repeat regularly (e.g. every 30-60 mins initially) to detect either deterioration or efficacy of any therapy instituted

Mentation[1] (Consciousness)	Normal Depressed/obtunded Stuporous Comatose Hyperaesthetic Confused/disorientated	Abnormalities typically relate to: ➢ Diffuse lesion or widespread multifocal lesions of both cerebral hemispheres ➢ Or, focal lesion affecting (ascending reticular activating system (ARAS) of) brainstem Coma typically indicates: ➢ Severe bilateral or global cerebral injury ➢ Or, severe brainstem damage ➢ Guarded prognosis
Pupils	Symmetry, size, PLRs (direct + consensual) ➢ Normal size pupil with slow PLR (least severe) ➢ Miotic pupil with intact PLR ➢ Pinpoint pupil with no PLR ➢ Mydriatic pupil with no PLR (most severe)	Normal PLRs = adequate function of rostral brainstem, optic chiasm, optic nerves, retinae Miotic pupils = diffuse forebrain injury Progression to mydriasis may indicate brain herniation ➢ Herniation places pressure on oculomotor n. (III) which interrupts parasympathetic input resulting in dilated pupils Herniation may also compress brainstem Fixed, unresponsive and midrange pupils seen with cerebellar herniation Brain herniation associated with severe disability and death if not treated rapidly and aggressively
Other cranial nerves	Especially:	Menace responses Blink and gag reflexes Jaw tone
Oculocephalic reflex	Moving head side-to-side or up/down should elicit physiological nystagmus[2]	Abnormality following TBI typically due to brainstem lesion
Respiratory pattern	TBI may cause irregular respiratory patterns	Hyperventilation Cheyne-Stokes respiration: progressively deeper and sometimes faster breathing followed by gradual

		decrease that results in apnoea Ataxic respiration (irregular rate, rhythm and excursion) Apnoea
Motor function[3]	Hemiparesis/plegia: Tetraparesis/plegia:	Unilateral brainstem damage (also some spinal cord lesions) More diffuse brainstem damage (also cervical spinal cord lesion)
Posture[4]	Decerebrate rigidity: Decerebellate rigidity:	Extreme extensor rigidity of all 4 limbs with no periods of relaxation; lateral recumbency and possible opisthotonos; marked changes in mentation (usually stuporous or comatose); very grave prognosis. Extensor rigidity of thoracic limbs with flexion of pelvic limbs due to sublumbar muscle contraction flexing hips; or extensor rigidity of all 4 limbs if ventral cerebellum affected. May occur episodically. Mentation usually normal. Better prognosis than decerebrate rigidity.

[1] Mentation:

- Depressed/obtunded = Decreased functional activity; mentally dulled
- Stuporous = Unresponsive to normal environmental stimuli but responsive to painful stimuli
- Comatose = Unresponsive to environmental or painful stimuli
- Hyperaesthesia = Increased sensitivity to normal level of stimulation
- Confused/disorientated (delirious) = Responding to environmental stimuli in an inappropriate manner

[2] In a normal animal as the head is moved, the pupils will initially remain looking in the original direction (i.e. forwards) but will then 'catch up' with the rest of the head. At the end of each movement, the head should be stabilised and cessation of nystagmus noted.)

[3] Animals that are not comatose should maintain voluntary motor activity unless trauma has occurred to the brainstem, the spinal cord, or a peripheral nerve.)

[4] Schiff-Sherrington posture:

- Must be distinguished from decerebrate and decerebellate rigidity
- May occur as a result of an acute severe lesion of the spinal cord between T2 and L3
- Pelvic limb flaccid paralysis accompanied by extensor rigidity or at least increased tone of thoracic limbs when the animal is in lateral recumbency
    - Thoracic limbs otherwise neurologically normal
- Spinal cord lesion interrupts ascending inhibitory impulses originating in lumbar grey matter and terminating on cells responsible for extension of thoracic limbs

Schiff-Sherrington posture does not indicate that the spinal cord injury is irreversible.

Findings consistent with brainstem injury:

- Mentation: comatose
- Bilateral fixed (mid-position) dilated unresponsive pupils
- Absence of gag reflex
- Irregular respiratory patterns or apnoea
- Abnormal oculocephalic reflex

Diagnostic imaging

Diagnostic imaging of the head is not routinely indicated but would be indicated in a patient that:

- Fails to respond to aggressive extracranial and intracranial stabilisation
- Deteriorates acutely after initially responding to therapy
- Shows progressive neurological signs

Although plain skull radiographs may reveal fractures, it can be difficult to obtain radiographs of interpretable quality and plain radiographs do not provide clinically useful information with respect to brain injury. Referral to a specialist centre for advanced imaging (computed tomography (CT) or magnetic resonance imaging (MRI)) should therefore be considered if affordable and once the patient has been stabilised.

CT is the standard of care in human TBI, being widely available and less expensive than MRI. CT offers rapid scan times that are faster than MRI as well as better visualisation of fractures and per-acute haemorrhage. Abnormalities associated with increased ICP include:

- Subdural or intracerebral haematomas
- Subarachnoid haemorrhage
- Cerebral infarcts
- Diffuse brain injury
- Generalised cerebral oedema often with shift of midline structures and ventricular compression

However it is possible that MRI may provide key prognostic information by detecting subtle parenchymal damage not evident on CT.

Modified Glasgow Coma Scale (The Small Animal Coma Scale)

The MGCS is a quantitative way of grading and monitoring brain injury by scoring three categories from 1-6; the categories are motor activity; brainstem reflexes; and, level of consciousness. We'll come back to the MGCS when we discuss prognostication.

Treatment

As mentioned, the priority in the management of TBI is to **ensure the brain receives an adequate supply of well-oxygenated blood**. This involves:

Oxygen supplementation

See notes above. If adequate oxygenation cannot be achieved non-invasively the patient should be anaesthetised, intubated and ventilated; however there are practical and financial considerations to providing this management.

Optimising cerebral perfusion

Intravenous fluid therapy:

Early fluid resuscitation is essential to correct systemic hypoperfusion and ensure adequate cerebral blood flow. A replacement isotonic crystalloid is the typical first choice but a synthetic colloid may be needed if there is an inadequate response. Although commonly referred to as isotonic, Hartmann's (buffered lactated Ringer's solution, compound sodium lactate) is in fact mildly hypotonic with a slightly lower sodium concentration than that of the extracellular fluid. In theory the tendency towards plasma hypoosmolarity that may follow its administration may have consequences in terms of promoting some cerebral oedema (as water moves down a concentration gradient from the capillaries into the brain parenchyma). Whether or not this occurs in any clinically significant way remains to be investigated but it may be more rational to use normal saline (0.9% sodium chloride), which is isotonic, rather than Hartmann's in resuscitating TBI patients. Hypertonic saline is an appropriate choice in some cases (see below). It is preferable to avoid hypervolaemia as this may increase intracranial pressure, especially when there is a damaged blood-brain barrier, but it is crucial to only be conservative once hypovolaemia has been corrected.

Intracranial hypertension therapy (see below)

Minimise increases in cerebral metabolic rate:

Steps must be taken to minimise increases in cerebral metabolic rate that may worsen TBI further. Anti-seizure therapy should be used immediately if indicated and any patient that is distressed (flailing, constant vocalisation) sedated; this sedation can be achieved using opioids but also with anticonvulsant medications even if the patient is not seizuring as anticonvulsant agents often have sedative properties.
Treat hyperthermia that may occur secondary to direct trauma to the thermoregulatory centre in the hypothalamus and/or seizure activity (also due to pain or excitement). Hyperthermia is undesirable as it increases cellular metabolism and vasodilation leading to increased ICP.

Intracranial hypertension therapy

Treatment for raised ICP includes measures to promote venous drainage from the brain, e.g. keep the head elevated 15-30° above horizontal; minimise jugular compression (e.g. avoid jugular venepuncture, do not restrain around neck area). However the most effective therapy involves medical therapy for cerebral oedema.

"The mainstay of intracranial-pressure reduction is…the rudimentary approach of shrinking the brain by exposing it to the dehydrating effects of serum hyperosmolarity."

The brain parenchyma contains a large proportion of water (more so than for other organs) making brain volume very responsive to changes in water content. The intact blood-brain barrier is highly impermeable to both sodium and mannitol; these substances therefore induce an osmotic gradient between blood and brain tissue. The beneficial effect of hyperosmolar therapy requires that the blood–brain barrier be intact. In regions of brain-tissue damage, as in traumatic contusion, the barrier is disrupted and allows equilibration of molecules between blood and the interstitial fluid of the brain. Thus, hyperosmolar agents exert their effect largely by removing water from the remaining normal brain tissue. It follows that hyperosmolarity reduces intracranial pressure in proportion to the volume of undamaged brain tissue.

When should medical therapy be implemented?

It is not always entirely clear when this point is reached however the Cushing reflex would definitely be considered an indication. Medical therapy is also considered rational in a patient with progressive neurological signs secondary to TBI and there may be an argument for its use in any patient with moderate-to-severe head injury that is refractory to aggressive extracranial stabilisation.

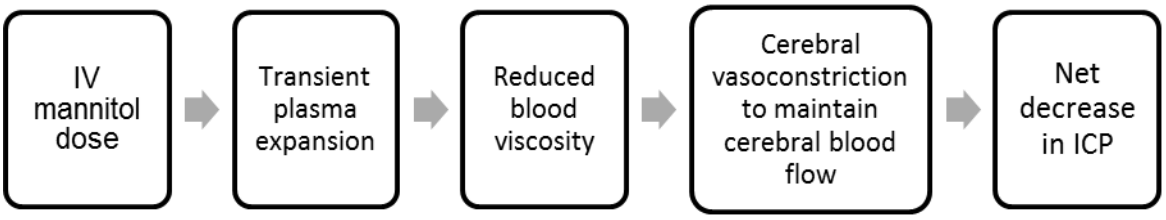
Hypertonic saline or mannitol?

At the present time, the evidence base that exists does not support one of these fluids being more effective than the other for the reduction of intracranial hypertension. In a normovolaemic patient, either can be chosen.

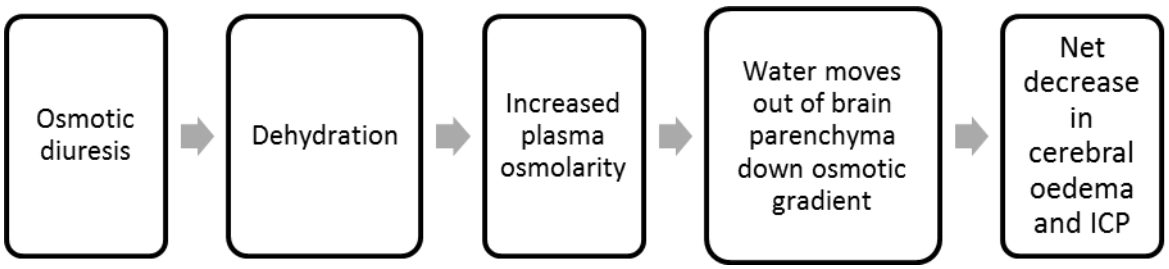
Mannitol (dehydrating osmotic agent) is contraindicated in hypovolaemic patients; hypertonic saline (volume-expanding solution) on the other hand is a rational choice in hypovolaemia as it may serve to both restore intravascular volume (albeit transiently) and reduce intracranial pressure at the same time.

Mannitol

Mannitol is the most longstanding medical therapy for cerebral oedema. It is a white crystalline organic compound that is dissolved to form a hyperosmolar sugar alcohol solution. There is some debate about how mannitol achieves a reduction in intracranial pressure. One main effect may be via a rheological mechanism as follows: intravenous administration of mannitol causes transient plasma expansion which reduces blood viscosity; this leads to cerebral vasoconstriction to maintain cerebral blood flow and the net result is a decrease in ICP. This effect occurs immediately and may persist for up to 75 minutes.



Because mannitol solution is hyperosmolar, it is also likely to cause an osmotic diuresis; the resulting dehydration increases plasma osmolarity that then reduces cerebral oedema as water moves out of the brain down a concentration gradient. The onset of this osmotic effect is likely to be after 15-30 minutes and it may peak after 1 hour and persist for 6-8 hours.



Mannitol may also have an effect as a reactive oxygen species scavenger which may limit secondary oxidative injury.

Mannitol is given at 0.5-1.5 g/kg IV over 10-20 min and can be repeated every 2-8 hours (max 3 doses in 24 hours) but discontinue therapy if there is no positive response to a single dose.

Contraindications for mannitol use include hypovolaemia/dehydration, congestive heart failure, pulmonary oedema and anuric renal failure. High doses of mannitol can cause acute renal failure by as yet unknown mechanisms. Older resources quote active intracranial haemorrhage as a contraindication; however there is no evidence base for this as such and there is no way of knowing whether our patients are experiencing active haemorrhage so this author's recommendation is to disregard this as a concern. Given the osmotic diuretic effect, mannitol administration should be followed with an isotonic crystalloid to prevent dehydration or hypovolaemia. The solution can crystallise when stored at room temperature so make sure to dissolve all crystals before use and the use of an in-line filter (i.e. same as is used for blood transfusion) is also recommended.

Hypertonic saline

Hypertonic saline (e.g. 7.2-7.5% sodium chloride (~ 2400 mOsm/l; normal plasma osmolality ~ 290 mOsm/l) is osmotically active. The infused sodium raises the plasma osmolarity creating a concentration gradient for water to leave the brain tissue thereby reducing brain water and ICP. It also dehydrates endothelial cells thereby increasing blood vessel diameter, improving CBF and reducing ICP. Hypertonic saline may also modulate any inflammatory response. It is especially indicated in hypovolaemic TBI patients as it will both restore intravascular volume and reduce cerebral oedema.

Recommended doses (over 5 mins): Dogs: 4-7 ml/kg IV; cats: 2-4 ml/kg IV

Other treatment considerations

Analgesia

Once a rapid baseline neurological examination has been performed, analgesia should be administered. A pure opioid is the agent of choice as these agents are rapidly acting; methadone is preferable to morphine as the latter may induce emesis which could worsen raised ICP. If available, fentanyl can be an excellent choice in these patients. Remember that NSAIDs are contraindicated in hypovolaemic patients which many trauma patients are likely to be. Analgesia may also contribute to sedation which can be helpful as mentioned previously.

For quite some time it had been said that ketamine is contraindicated in the presence of raised ICP as it may worsen this. However there is increasing evidence to suggest that by increasing systemic arterial pressure via vasoconstriction ketamine may increase cerebral perfusion pressure; this effect of ketamine may in fact be more beneficial for the brain than any adverse effect it may have by directly increasing ICP. Regardless, a current perspective is that in the presence of raised ICP ketamine is no better but also no worse than any other agent that may be being considered in its place; moreover it lacks hypotension as an adverse effect which may make it an attractive choice over other sedatives.

Steroids

The use of methylprednisolone succinate for central nervous system injury was a longstanding practice in both human and veterinary medicine. However it was not one based on clinical evidence. More recent clinical trials in humans have not shown positive effects on outcome and some have suggested possible

increases in morbidity and/or mortality. Considering the potential adverse effects (e.g. gastrointestinal ulceration, increased risk of infection/immunosuppression, hyperglycaemia, increased catabolism) of these agents, current recommendations are that methylprednisolone should not be used for traumatic brain injury. “Given the lack of evidence for any beneficial effect of corticosteroids after TBI, and strong evidence in the human literature showing a detrimental effect on neurological outcome, corticosteroids should not be administered in TBI”. [In patients with brain tumours, glucocorticoids may lower intracranial pressure by reducing vasogenic oedema surrounding the brain tumor; oedema results from the flow of fluid into the extracellular space of the brain parenchyma through an incompetent blood–brain barrier (BBB).]

The author will occasionally use corticosteroids at an anti-inflammatory dose (e.g. dexamethasone 0.1 mg/kg) for animals with significant soft tissue swelling due to head trauma but this is a different indication and dosing regimen to the previous use of steroids for CNS injury.

Nutritional support

TBI may result in a hypermetabolic and catabolic state. Nutritional support is very important once the patient is stable and improving but should not be started in animals that remain cardiovascularly unstable (feeding may worsen cardiovascular status further) or severely neurologically compromised (due to risks of aspiration if gag reflex is still absent). Ideally avoid syringe feeding as this provides inadequate calorific intake, poses the potential risk of aspiration and is unlikely to be patient friendly in the presence of head injuries. A naso-oesophageal or naso-gastric tube is an option although may not be ideal with facial injuries; some animals do not tolerate these tubes well and may also sneeze which could worsen raised ICP. An oesophagostomy tube is preferred once the patient is stable for anaesthesia. Parenteral nutrition may be employed in the interim if available.

Decompressive surgical therapy

There is still no consensus in people as to if and when surgery should be pursued and the effect of surgical intervention on clinical outcome remains unclear. With the increasing availability of advanced imaging surgical therapy may come to play a greater role in veterinary medicine. Possible indications include:

- Open or depressed skull fractures
- On-going haemorrhage
- Foreign body or haematoma removal
- Declining neurologic status despite aggressive medical therapy

Comment on hyperglycaemia following TBI

Hyperglycaemia is seen relatively commonly following TBI and is thought to be due to a response that includes the sympathetic nervous system and the adrenal medulla (i.e. catecholamines!). It is associated with increased mortality or worsened neurological outcomes in humans and experimental animals but does it actually worsen injury, is it just a marker of injury severity, or both? Hyperglycaemia following TBI in dogs and cats does appear to at the very least be a marker of the severity of injury but at this time there is no evidence to suggest that it has a detrimental effect on outcome. Therefore specific therapy is not indicated but it is important to avoid iatrogenic hyperglycaemia – e.g. don’t use corticosteroids!

Monitoring

Animals with traumatic brain injury must be monitored very closely including regular reassessment of neurological status, possibly with repeat MGCS assessments. In particular look out for the following three developments.

Bradycardia: the onset of previously undetected bradycardia may signal an increase in ICP and should prompt immediate treatment.

Respiratory distress: brain injury can give rise to **neurogenic (non-cardiogenic) pulmonary oedema** that usually develops within minutes to a few hours of the event. Thoracic radiographs typically show a caudodorsal distribution of lung field changes consistent with pulmonary oedema (interstitial to alveolar pattern). The mainstay of treatment is oxygen supplementation, strict rest, minimal stress, usual supportive measures for dyspnoeic patients, and time – and addressing the primary cause if that is appropriate/on-going. Clinical improvement is typically noted within 24 - 48 hours. The mechanisms behind neurogenic pulmonary oedema are still to be fully unravelled. We know what types of events cause it (e.g. traumatic brain injury, neck injury, seizures, upper respiratory obstruction), and some of the likely mechanisms have been identified. These mechanisms result in oedema that is probably due to a combination of increased capillary permeability, and hydrostatic pressure changes (as with cardiogenic oedema). There is probably a rationale to administer one single low dose of furosemide (e.g. 0.5 mg/kg IV to a cat, 1 mg/kg IV to a dog) to these patients as furosemide is thought to have a number of different mechanisms of action that may potentially improve oxygenation beyond just the clearance of congestive oedema. However these patients should not be treated in the same way as a patient with pulmonary oedema from congestive heart failure and remember that furosemide is contraindicated in the presence of hypovolaemia or dehydration.

Aspiration pneumonia is another possible cause of respiratory distress in a neurologically compromised patient following TBI.

Seizure activity: seizure activity will increase cerebral metabolic rate, increasing cerebral oxygen demand and therefore the risk of hypoxic injury. It may also worsen cerebral oedema thereby exacerbating raised intracranial pressure (ICP) and can cause hyperthermia. Anti-seizure medication must therefore be administered at the first sign of seizure activity. Seizures are rare following head trauma in dogs and cats, both acutely and also in the long-term; although the role of prophylactic anticonvulsant therapy remains unclear, it is generally not recommended to be used in either the short- or long-term.

Nursing care

Patients with traumatic brain injury may be recumbent for prolonged periods and the value of adequate nursing in such cases should not be underestimated. The most important measures are as follows:

- TLC, gentle handling (independent of interventions)
- Minimise stress: be calm and quiet around the patient, provide a dimly lit quiet environment, use gentle restraint and minimise/group interventions
- Use well-padded bedding to prevent pressure sores and ensure bedding is dry, clean and comfortable
- Turn every 2-4 hours if recumbent to prevent atelectasis (lung collapse)
- Lubricate eyes every 4-6 hours to prevent corneal drying and ulceration
- Appropriate bladder management: express or catheterise if necessary and prevent urinary soiling; catheterisation increases the incidence of urinary tract infection – the risk is greater with in-dwelling catheters versus intermittent catheterisation, and the risk increases the longer an in-dwelling catheter is left in situ.
- Tempt to eat if appropriate

- Perform regular physiotherapy in patients that are likely to be recumbent for prolonged periods

Prognosis

Clearly the desired end-point is for a live patient with a good quality of life. It is important not to make too hasty judgements as dogs and cats can compensate for considerable loss of cerebral tissue. Prognostication can be very difficult. In human medicine clinically-derived prognostic calculators such as the IMPACT Prognostic calculator (International Mission for Prognosis and Analysis of Clinical Trials in TBI; www.tbi-impact.org) or the CRASH Prognosis model (www.crash.lshtm.ac.uk) can help to at least guide prognostication if not being completely definitive. In human and veterinary patients signs of slow but steady improvement are likely to be the most practical guide. Level of consciousness is the most reliable empirical measure of impaired cerebral function and it provides information about the functional capabilities of the cerebral cortex and the ascending reticular activating system (RAS) in the brainstem. The paper mentioned previously suggests:

MGCS score	Suggested prognosis
3-8	Grave
9-14	Guarded
15-18	Good

Platt SR, Radaelli ST, McDonnell JJ. The Prognostic Value of the Modified Glasgow Coma Scale in Head Trauma in Dogs. J Vet Int Med 2001. 15(6):581-584.

However there is limited data available correlating survival with MGCS score so this system may be better used to objectively assess progression rather than as a prognostic indicator. This is especially the case in patients that have polysystemic injuries – such injuries clearly also affect prognosis but this patient cohort was excluded from the above study.

FELINE SACROCAUDAL/TAIL PULL INJURY

The sacrocaudal region of the vertebral column contains the peripheral nerve roots of the cauda equina rather than the spinal cord. Injury to the sacral and caudal (coccygeal) nerve roots most commonly occurs as a result of sacrocaudal fractures or luxations/subluxations secondary to motor vehicle accidents and bite wounds. Distraction and possible avulsion of the nerve roots may also occur as a result of a tail pull injury where traction is applied to the tail as it is trapped (e.g. beneath a tyre) and the cat tries to escape. Complete or partial, reversible or irreversible neurological dysfunction may occur depending on the extent of the injury to the nerve roots.

The sacral spinal cord segments (S1 to S3) and nerve roots contribute to the pelvic and pudendal nerves – see diagram in attached In Practice article:

Pelvic nerve:

- Transmits sensory information from and parasympathetic motor innervation to the detrusor muscle of the bladder
- Transmits sensory information from and parasympathetic motor innervation to smooth muscle of the descending colon

Pudendal nerve:

- Transmits sensory information from the external urethral sphincter, anal sphincter and perineal area
- Provides motor innervation to the external urethral sphincter and the striated muscle of the anal sphincter

Neurons from the S1 segment also contribute to the sciatic nerve. The caudal spinal cord segments provide sensory and motor innervation to the tail via the caudal (coccygeal) nerves.

Neurological examination

Tail

Reduced or absent tail sensation, motor function or tone
Tail sensation test may have more prognostic value if performed at the tail base

Perineum

Perineal urinary and/or faecal soiling
Sacral nerve injury causes reduced or absent perineal reflex and anal sphincter tone (dilated anal sphincter) with possible constipation and faecal incontinence.

- Perineal reflex: stimulation of either side of the anus with a pair of forceps results in reflex contraction of the anal sphincter and flexion of the tail

Perineal sensation (e.g. by stimulating the perineum by closing forceps on the perineal skin area and assessing any conscious response from the cat) may also be reduced or absent.

Bladder

Flaccid distended bladder that is usually easy to express manually due to reduced urethral sphincter tone; urinary incontinence may occur (loss of the detrusor reflex may result in overflow of a full bladder with dribbling of urine)

Pelvic limbs

Pelvic limb weakness, lameness or ataxia – may be due to sciatic nerve involvement and/or pelvic or pelvic limb fractures
Pelvic limb postural reactions (paw placement, hopping) may also be reduced and pelvic limb segmental reflexes (withdrawal, patellar) may be abnormal.

Prognosis

At this time there are no prognostic indicators that allow clinicians to definitively distinguish between animals that will recover and those that will not.

The prognosis for cats in which only the tail is affected is very good including with respect to possible return of tail function. Thereafter the prognosis is generally associated with the severity of neurological injury to the peripheral nerve – see attached In Practice article.

The most severely affected animals (dilated anus with no anal tone; absent tail sensation and motor function) may not recover. Recovery, in particular with respect to tail sensation and motor function, can take weeks to months to occur. However, a prolonged absence (longer than 2 weeks) of perineal deep pain perception, particularly when this is associated with a flaccid, areflexic anal sphincter, is suggestive of a severe lesion to the nerves exiting from the sacral segments (pudendal and pelvic nerves) and is a poor prognostic indicator. As a guideline, most cats that do not recover urinary control after 4 weeks are likely to remain incontinent.

A discussion of appropriate management for animals with prolonged recovery is beyond the scope of these notes.