How to approach the patient with **head trauma**

Victoria Doyle, European and RCVS Specialist in Veterinary Neurology, guides us through the approach to these critical patients using images from the *BSAVA Manual of Canine and Feline Neurology, 4th edition*

How to treat patients with head trauma remains a relatively controversial topic and, with limited studies in animals, much of the information is extrapolated from human studies. However, it is important to manage these patients appropriately from the outset as it is likely that the treatment they receive initially will dictate their outcome.

Advanced imaging and monitoring are beneficial in some cases but much can be achieved in the first opinion practice setting. Treating these patients is very labour-intensive but can be extremely rewarding. Although mortality rates are high after severe brain injury, dogs and cats have a huge ability to recover if they receive the appropriate treatment and are given the time to recover.

Head trauma can occur from a number of causes including:

- Road traffic accidents
- Falls
- Kicks from cattle/horses
- Bites from larger animals
- Gunshot wounds

Head trauma pathophysiology

Head trauma leads to primary and secondary brain injury.

Primary brain injury

Primary brain injury occurs during the traumatic event (i.e. lacerations, contusions and diffuse axonal injury). In general there is very little that can be done to reverse these processes, with a few exceptions:

- Unstable skull fractures and those resulting in penetrating bone fragments may benefit from surgery
- Foreign material (i.e. a gunshot pellet) may need to be removed surgically
- Intracranial haemorrhage occurring between the skull and brain parenchyma (extra-axial haemorrhage) may also benefit from decompressive surgery.

Secondary brain injury

Secondary brain injury is a complex series of interrelated biochemical pathways, which cause further brain injury and increased intracranial pressure (see Figure 1). This cascade of events is perpetuated by systemic abnormalities, including:

- Hypotension
- Hypoxaemia
- Hypo/hyperglycaemia
- Hypo/hypercapnia
- Hyperthermia

Therefore, there is scope for the clinician to reduce the amount of secondary brain injury and to improve the prognosis for the patient.

Intracranial pressure (ICP)

ICP and cerebral blood flow are interlinked and their relationship is explained in the following formulae:

$$\text{Cerebral blood flow (CBF)} = \frac{\text{Cerebral perfusion pressure (CPP)}}{\text{Cerebral vascular resistance (CVR)}}$$

$$\text{Cerebral perfusion pressure (CPP)} = \text{Mean arterial pressure (MAP)} - \text{Intracranial pressure (ICP)}$$

$$\text{So: CBF} = \frac{(\text{MAP}-\text{ICP})}{\text{CVR}}$$

In the normal animal, the ICP remains constant (5–12 mmHg) at mean arterial blood pressures between 50 and 150 mmHg. If blood pressure rises, then vasoconstriction occurs within the brain, conversely; if blood pressure falls, then vasodilation occurs in the brain to maintain cerebral blood flow. This is referred to as pressure autoregulation.

The brain vasculature also responds to the partial pressure of carbon dioxide in arterial blood (P_aCO_2). If the P_aCO_2 increases, cerebral vasodilation occurs. If the P_aCO_2 decreases, then vasoconstriction will occur. This is referred to as chemical autoregulation.

The brain is encased within the rigid confines of the skull and therefore if the brain parenchyma component increases in size (e.g. due to haemorrhage or oedema) then the other compartments (i.e. blood and cerebrospinal fluid, CSF) must compensate to prevent a rise in ICP (see Figure 2).

The CSF component is affected first, with CSF shunted out of the brain, leading

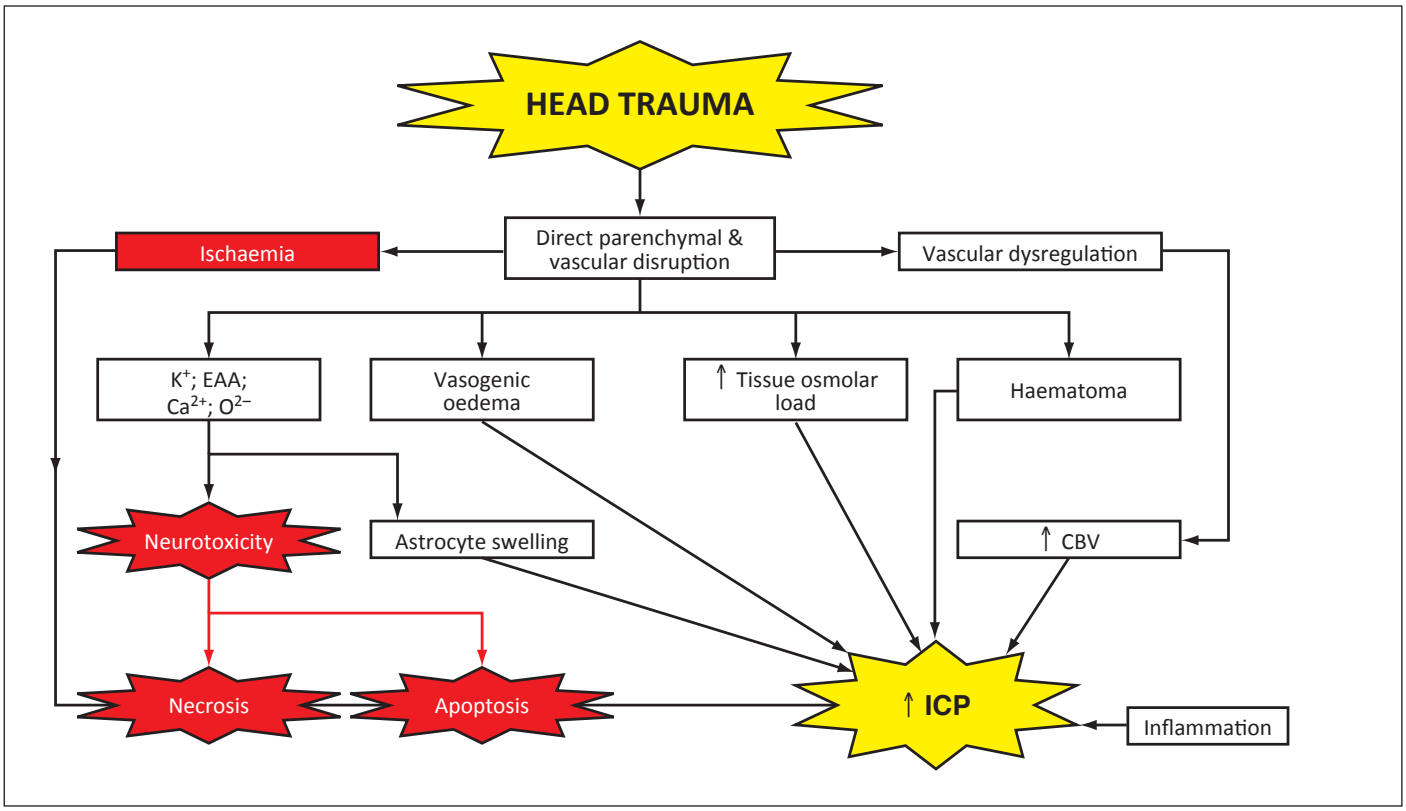

Figure 1: Overview of the pathophysiology of head trauma as it relates to the increase in intracranial pressure (ICP), showing the underlying vasculature and biochemical components of secondary injury.

CBV = cerebral blood flow; EAA = excitatory amino acids. Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

to a reduction in size of the ventricular system. Once this mechanism has been exhausted, there is a reduction in CSF production. Subsequently, cerebral blood flow is reduced.

Compensation for a rise in ICP can only occur for a limited period and once these mechanisms have been exhausted, small changes in brain parenchyma size will cause marked increases in ICP. It is impossible to tell clinically when the ability for the brain to compensate has been exhausted. Therefore, sudden rapid deterioration is possible and the patient needs to be monitored frequently.

Marked increases in ICP can lead to a Cushing's reflex (bradycardia and systemic hypertension) (see Figure 3). The increase in ICP will reduce the amount of blood available to perfuse the brain and will result in neurological deterioration. The Cushing's reflex will usually be accompanied by other neurological signs commonly seen with raised ICP. These include:

- Changes in pupil size and responsiveness
  - Initially bilateral miosis
  - Progressing to bilateral mydriasis with non- or poorly responsive pupils as the pressure continues to rise
- Decreased mentation (obtundation, stupor, coma)
- Ultimately vertical nystagmus and extensor rigidity (can occur when the brainstem is compressed due to herniation)

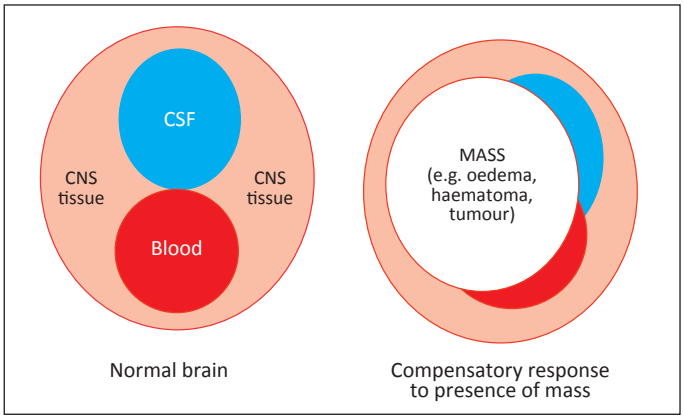

Figure 2: An increase in the volume of one tissue component within the skull requires a compensatory decrease in the volume of the other tissues to prevent an increase in ICP

Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

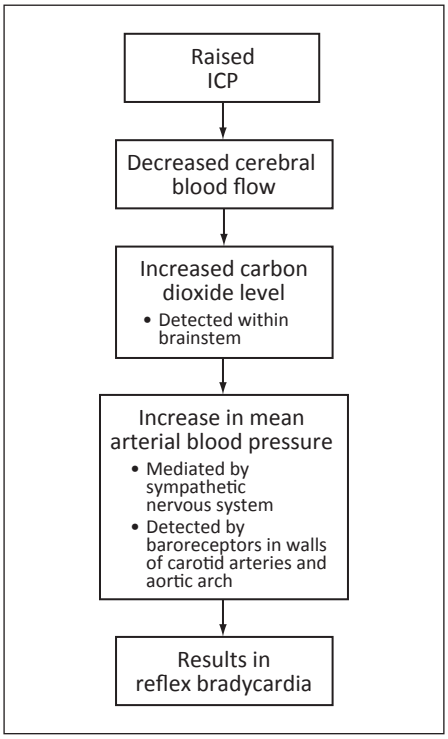

Figure 3: How the Cushing's reflex leads to bradycardia in patients with raised intracranial pressure

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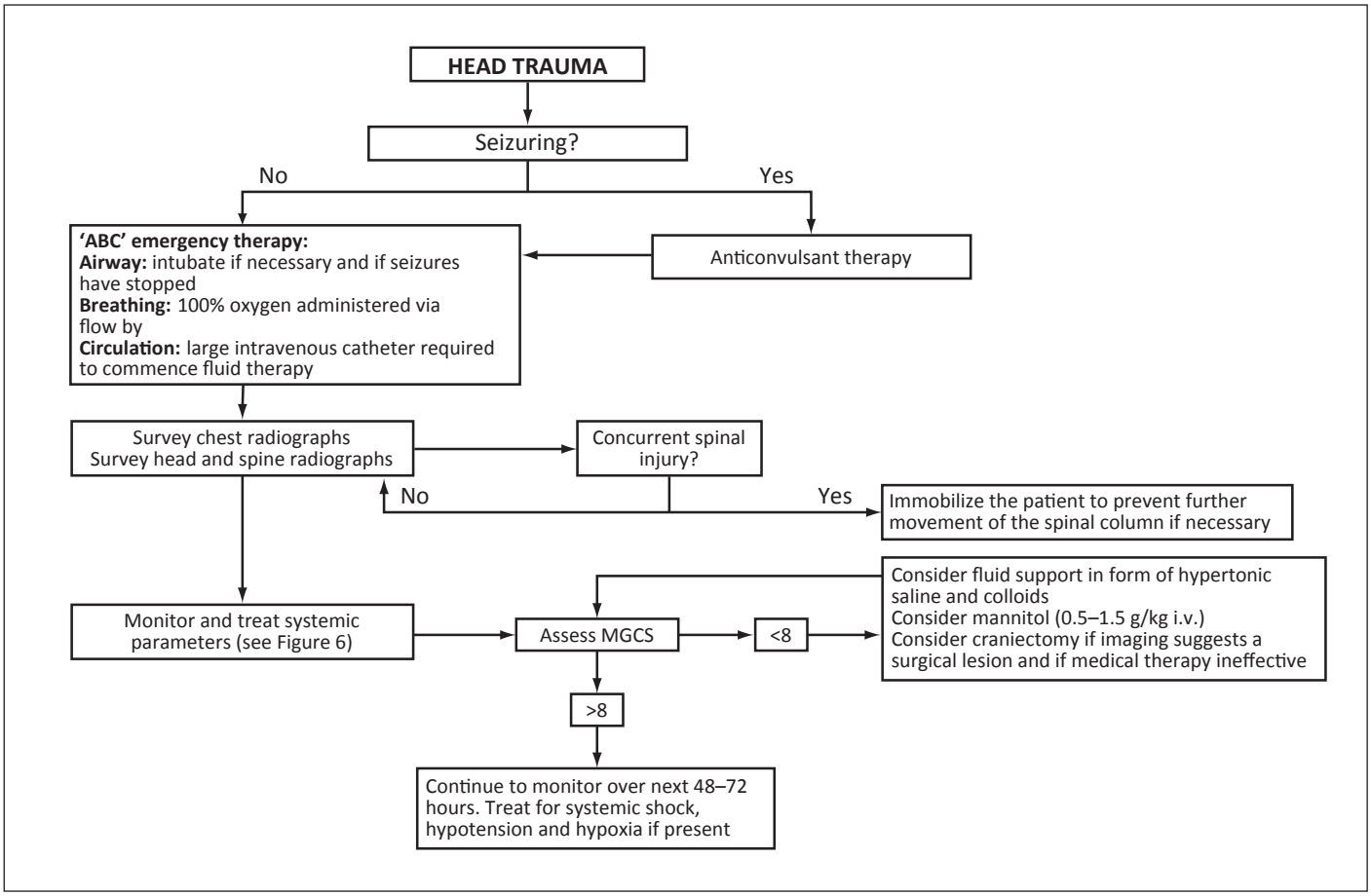

Figure 4: An approach to management of head trauma

MGCS = modified Glasgow coma score. Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

Initial patient assessment

Many patients presenting after severe trauma will be in hypovolaemic shock. They may also have significant hypoxaemia. Both of these can have a significant effect on the patient's mentation. It is therefore important to assess the whole patient and to institute treatment before focusing too closely on the neurological signs (see Figure 4).

However, the clinician needs to be mindful of the possibility of vertebral fractures. It is not uncommon for cervical fractures, in particular, to be sustained at the same time as a severe head trauma. Therefore, movement of the patient should be minimized, when possible, until a fracture has been ruled out.

Respiratory assessment

Patients should have oxygen provided whilst they are being assessed. The aim is to supply an inspired oxygen concentration of 40%. This can be via facemask, nasal prongs, nasal catheters or transtracheal oxygen catheter. The flow rate for nasal catheters is 100 ml/kg/minute and for transtracheal oxygen catheters is 50 ml/kg/minute.

Nasal prongs can make the patient sneeze, especially when high flow rates are used, which could increase ICP. If sneezing does occur then it would be advisable to change to an alternative source of flow-by oxygen.

Nasal catheters should not be advanced into the nasal cavity further than the medial canthus of the eye to try to avoid inadvertent entry into the cranial vault via a possible skull fracture.

If the patient is not ventilating appropriately (i.e. they are losing or have lost consciousness) they should be intubated and given oxygen via endotracheal tube.

Oxygen tents/cages are unhelpful during the early management of these patients as the patient requires continuous assessment, which does not allow the oxygen levels to increase within the tent/cage.

Respiratory factors to assess include:

- Breathing rate and pattern (i.e. shallow)
- Mucous membrane and tongue colour
- Thoracic auscultation
- Pulse oximetry
  - The oxygenation can be overestimated especially at low P_aO_2 levels
  - The patient's haemodynamic status can also affect the reading
- Arterial blood gas (if possible)
  - To measure P_aCO_2 and P_aO_2
- End tidal CO_2 (if the patient is intubated)
  - Can underestimate the P_aCO_2

Main use	Dose	Pros	Cons	Notes
Synthetic colloid e.g. Hetastarch				
Blood volume expansion	10–20 ml/kg	Rapid expansion of blood volume (half life of 25 hours)	Max dose 20 mg/kg to prevent bleeding	
			Patients <5 kg have a risk of fluid overload	
Hypertonic saline e.g. 7.5% hypertonic saline				
Blood volume expansion	4 ml/kg over 5–10 minutes in dogs 2 ml/kg over 5–10 minutes in cats	Improves systemic blood pressure, cerebral blood flow and reduces ICP. This effect lasts longer than the volume expansion effect	Do not use if the patient is dehydrated or hypernatraemic as it will worsen these conditions	Draws fluid from the interstitium to the intravascular space
		Decreases brain excitotoxicity	Volume expansion only lasts 15–75 minutes	Must administer crystalloids concurrently to prevent systemic dehydration caused by the hypertonic saline
			Use with care in patients with pulmonary oedema/contusions with underlying cardiac or respiratory disease, as hypertonic saline can exacerbate the oedema/contusions	Colloids can be given after the hypertonic saline to maintain the intravascular volume but will not prevent dehydration
Isotonic crystalloids e.g. 0.9% saline				
Rehydration	10–20 ml/kg boluses over 15–20 minutes to restore normal blood pressure. (The dose can be repeated up to shock dose 90 ml/kg for dogs and 60 ml/kg in cats, if necessary)	Corrects dehydration and used to prevent dehydration when used with hypertonic saline	Moves into the interstitium from the intravascular space within an hour, so larger volumes are needed to restore blood volume	
			Can cause overhydration, worsen brain oedema and increase ICP	
	Maintenance dose 2 ml/kg/h + % dehydration + losses		Prolonged use will cause hyperchloraemic metabolic acidosis	
Hypotonic crystalloids				
Lactated Ringer's/Hartmann's solution				
Rehydration	Maintenance dose 2 ml/kg/h + % dehydration + losses	Corrects dehydration and used to prevent dehydration when used with hypertonic saline	Moves into the interstitium from the intravascular space within an hour, so larger volumes are needed to restore blood volume	
			More likely to cause overhydration, worsen brain oedema and increase ICP	
5% glucose				
Correction of hypoglycaemia	2 ml/kg/h	Correction of hypoglycaemia which can cause neurological deterioration and seizures	Care that hyperglycaemia is not induced, as this can exacerbate neuronal injury in damaged tissues.	
Blood products				
Whole blood				
Correction of anaemia	2 ml/kg will increase PCV by 1% over 4 hours (can be given faster if patient is unstable)	Improved oxygenation to the brain	Monitor for signs of transfusion reaction	Aim for a PCV 25–30%
Packed red cells (pRBCs)				
Correction of anaemia	1 ml/kg will increase PCV by 1% over 4 hours (can be given faster if patient is unstable). Typically 10–15 ml/kg as a starting dose	Improved oxygenation to the brain	Monitor for signs of transfusion reaction	Aim for a PCV 25–30%
Fresh frozen plasma (FFP)				
Treatment of a coagulopathy	10–15 ml/kg two to three times per day until coagulopathy has resolved	Improvement of clotting status to reduce risk of further bleeding	Monitor for signs of transfusion reaction	

Figure 5: Table of fluid therapy options for initial stabilization in head trauma

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Within human medicine characteristic breathing patterns can indicate lesions within a specific part of the brain. However, this is yet to be proven in veterinary patients.

Hyperventilation

If the patient is intubated and the P_aCO_2 can be measured, the level should be kept between 35 and 40 mmHg. If the P_aCO_2 increases, this will cause brain vasodilation and a rise in ICP. If the P_aCO_2 falls below this range, brain vasoconstriction will occur and will impair perfusion to the brain.

Historically hyperventilation was used in patients with suspected raised ICP to reduce the ICP via hypocapnic vasoconstriction. However, if the P_aCO_2 is maintained below 35 mmHg for a prolonged period, then perfusion to the brain could be compromised due to vasoconstriction, ultimately leading to brain ischaemia and neurological deterioration. In patients who are acutely deteriorating, hyperventilation can be used for a short period but the clinician should remain mindful of the possibility for brain ischaemia with this technique.

Cardiovascular assessment

Cardiovascular factors to assess include:

- Mucous membrane colour
- Capillary refill time
- Heart rate and rhythm
- Presence of peripheral pulses and pulse quality
- Continuous ECG
- Non-invasive blood pressure (i.e. Doppler sphygmomanometry)

Patients should have a wide-bore intravenous catheter placed as soon as possible. When placing the intravenous catheter blood should be taken for a minimum database to include:

- Packed cell volume (PCV) and total protein (TP)
- Blood urea
- Glucose
- Electrolytes

Venepuncture of the jugular veins should be avoided, as jugular occlusion can reduce venous return from the brain and increase ICP.

Intravenous fluid therapy should be initiated promptly especially if the patient is showing signs consistent with hypovolaemia. Hypovolaemia will cause poor perfusion of the brain, which will exacerbate the neurological signs of the patient.

Options for intravenous fluid therapy (see Figure 5) include:

- Synthetic colloids
- Hypertonic saline
- Isotonic crystalloids
- Hypotonic crystalloids
- Blood products

Arterial blood pressure support

For some patients fluid resuscitation alone is insufficient to maintain an adequate mean arterial blood pressure. In these patients vasoactive agents, e.g. dopamine (2–10 μg/kg/min i.v. may be required).

If the patient is hypertensive then the clinician should be mindful that this could be part of the Cushing's reflex and indicate raised ICP. Therefore, treatment should be directed at reducing ICP rather than immediately focusing on lowering the systemic blood pressure.

Recording information

The patient's cardiovascular and respiratory status will change in response to your treatment and as the patient improves/deteriorates. The parameters should be monitored every 5–10 minutes initially. The frequency can decrease as the patient stabilizes but should be increased if there are any signs of deterioration. It is important to have a kennel sheet where all the clinical information can be recorded clearly, so that trends can be spotted readily and addressed. Some of the most useful kennel sheets have a chart for the heart rate, respiratory rate, blood pressure and pulse oximeter readings similar to that used to monitor general anaesthesia.

Secondary patient assessment

Once the patient's blood pressure, blood volume and oxygen status have been addressed, the patient should have a more thorough assessment of all body systems, including a full neurological examination to assess for other neurological injuries (e.g. vertebral or skull fractures). Other common injuries in polytrauma cases include orthopaedic injuries, damage to abdominal organs (e.g. splenic torsion and urinary tract ruptures) and damage to the thorax (e.g. pulmonary contusions, pneumothorax, haemothorax, neurogenic pulmonary oedema and rib fractures).

Additional clinical factors to assess

Rectal temperature should be assessed. It is important to try to normalize the temperature by passive cooling if the patient is hyperthermic or warming if the patient is hypothermic.

The patient's head should be raised to 15–30 degrees to maximize perfusion to the brain and venous drainage. It is vitally important that the jugular veins are not compressed or venous return will be impeded and ICP will increase. Laying the patient on an angled board, rather than using sandbags, is more likely to prevent jugular compression.

Urine specific gravity is also useful as part of the minimum database. In many of these patients, it is appropriate to place an indwelling urinary catheter with a closed collection system so that urine output can be closely monitored. If this is not possible, the clinician should be extremely cautious in obtaining the urine until the urinary tract has been fully assessed for trauma.

For those patients with an indwelling urinary catheter it is important to measure urine output. Urine output <1 ml/kg/h is defined as oliguria and urine output <2 ml/kg/h should be considered as relative oliguria in a hydrated animal with well perfused kidneys. If the urine output falls below 2 ml/kg/h then the clinician should become concerned regarding hydration, and fluid therapy should be adjusted. Other possibilities for oliguria include:

- Hypotension
- Pain
- Stress
- Inappropriate antidiuretic hormone (ADH) secretion
  - The patient will have hyponatraemia and high urine sodium level

If the urine output is sustained above 3 ml/kg/h this could indicate:

- Fluid overload
- Hyperglycaemia

- Diuresis (i.e. subsequent to mannitol or hypertonic saline administration)
- Central diabetes insipidus
  - This can occur with severe damage to the hypothalamus
  - The patient will have a high serum sodium, low urine sodium and low urine osmolality

The clinical parameters that can be monitored, their ideal ranges and suggested treatment options are contained within Figure 6. It may not be possible to directly measure central venous pressure and intracranial pressure in all patients. Even in the referral setting direct measurement of the intracranial pressure is not routinely performed. The resistive index of blood flow in the basilar artery can be measured using Doppler ultrasonography and this can give an indirect measure of ICP in dogs.

Neurological assessment

The neurological examination should be repeated every 30–60 minutes to monitor the patient for deterioration and to assess whether any treatment being given is effective. It can be useful to use the modified Glasgow coma scale (Figure 7) to objectively monitor the patient, especially if there are multiple people doing the monitoring. In general, the higher the score the better the prognosis for the patient (see Figure 8).

It is important to remember that significant impairment of mentation can be seen if the patient is hypovolaemic and hypoxaemic, which is why these need to be addressed before the patient's mentation is used to prognosticate. In addition, patients can show marked improvements with treatment in the first 24–48 hours and, consequently, trends in neurological status over this time are likely to be more beneficial to the clinician when assessing prognosis.

Osmotic diuretic *versus* hypertonic saline to reduce ICP

If the patient is showing signs of raised ICP, treatment with an osmotic diuretic (such as mannitol) or hypertonic crystalloid can be given (see Figure 9). Previous studies have shown that hypertonic saline is more effective at reducing ICP and can do so for longer than mannitol.

Monitoring parameter	Suggested goal	Suggested treatment
Neurological examination	Modified Glasgow coma score >15	Ensure head elevation (30 degrees); ensure all points below are addressed; consider mannitol; consider surgery
Blood pressure	Mean arterial blood pressure 80–120 mmHg	Adjust fluid therapy; pressor support (dopamine 2–10 μg/kg)
Blood gases	P_aO_2 ≥80 mmHg P_aCO_2 <35–40 mmHg	Oxygen supplementation; consider mechanical ventilation
Pulse oximetry	S_pO_2 ≥95%	Oxygen supplementation; consider mechanical ventilation
Heart rate and rhythm	Avoid tachycardia and bradycardia; avoid arrhythmia	Adjust fluid therapy; treat for pain; express bladder frequently; address intracranial pressure; treat arrhythmias specifically
Central venous pressure	5–12 cmH_2O	Adjust fluid therapy
Respiratory rate and rhythm	10–25 breaths/min	Ventilate if necessary
Body temperature	37–38.5°C	Passive warming or cooling; non-steroidal anti-inflammatory drugs if pyrexic
Electrolytes	(See laboratory normal values)	Adjust fluid therapy; supplement fluids accordingly
Blood glucose	4–6 mmol/l	Adjust fluid therapy; consider dextrose administration
Intracranial pressure	5–12 mmHg	Ensure head elevation (30 degrees); ensure all points above are addressed; consider mannitol; consider surgery

Figure 6: The monitoring parameters and suggested goals of treatment for patients with head trauma (Modified from Johnson and Murtaugh, 2000). Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

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

Figure 7: Modified Glasgow coma scale

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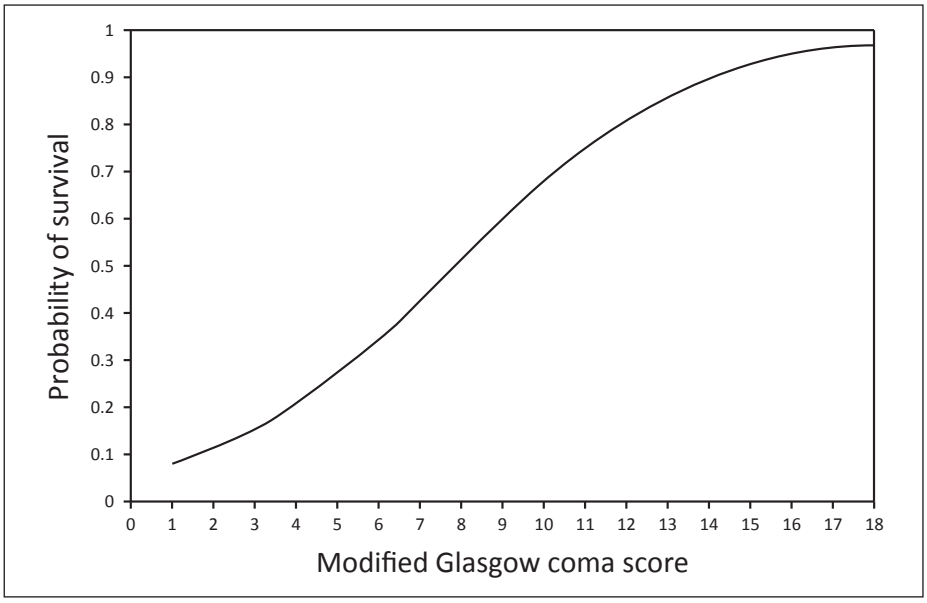

Figure 8: Probability of survival of the head trauma patient during the first 48 hours after admission, expressed as a function of the modified Glasgow coma score

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However, in euvolaemic patients either can be used; if the patient fails to respond to one, then the other may be more effective. The neurological examination and modified Glasgow coma scale should be repeated after the bolus of mannitol or hypertonic saline, to assess whether the treatment has had a beneficial effect. The clinician should also repeat the cardiovascular assessment, electrolytes and PCV/TP to monitor for any adverse effects, and institute treatment as appropriate.

Diagnostic investigation

When the patient is stable enough to be moved further, imaging can be performed to assess the extent of the patient's injuries. This may include:

- Radiographs (e.g. of the thorax, abdomen, long bones, skull and vertebral column)
- Ultrasonography (e.g. of the abdomen and thorax)
- Computed tomography (CT) or magnetic resonance imaging (MRI) of the head (and spine if indicated from the neurological assessment) when available
- Further blood work if necessary
- Clipping, cleaning and assessment of any skin injuries

Other possible treatments

Corticosteroids

At the current time, there is no evidence in human or veterinary medicine to support the use of corticosteroids (including methylprednisolone sodium succinate, MPSS) in patients with head trauma. Corticosteroids can have a significant detrimental effect on the patient, by:

- Increasing the risk of infection
- Immunosuppressing the patient
- Causing hyperglycaemia, which can exacerbate neuronal injury and cause cerebral acidosis.

There is also a significant increase in mortality reported in people receiving high-dose MPSS.

Therefore, their general use is not recommended.

Anticonvulsant therapy

It is important to remember that seizures can occur at three main time points after a head injury and the clinician should remain vigilant for them:

- Immediate – within 24 hours following head trauma
- Early – from 24 hours – 7 days following head trauma
- Late – occurring more than 7 days after head trauma

Seizure activity will increase ICP and therefore any seizure activity should be treated aggressively. Treatment is usually initiated with intravenous diazepam 0.5 mg/kg, which can be repeated up to a total of three times. Subsequent treatment with phenobarbital can be initiated if the seizure fails to stop (see Figures 10 and 11).

Levetiracetam can be used as an alternative to phenobarbitone or in addition to it. The dose of 20–60 mg/kg is given intravenously every 8 hours. Unfortunately, the intravenous preparation is expensive. However, levetiracetam tends to cause less sedation and respiratory depression than phenobarbital and so can be extremely useful in these patients. Phenobarbital has effects to reduce cerebral metabolic demands, which can protect the brain, but it can cause significant sedation and respiratory depression.

Treatment should continue for 3–6 months and if no further seizures occur then an attempt can be made to wean the patient off treatment. This must be done slowly with a dose reduction of 20–25% every month. The patient must be carefully monitored for any seizure activity and medication should be reinstituted if seizures occur.

There is some evidence to support the prophylactic use of anticonvulsant therapy in patients that have not seizured after head trauma. However, the pros and cons need to be weighed up carefully, as the medications can cause significant side effects of sedation and respiratory depression, which could be detrimental to the patient and can affect your monitoring.

Nutrition

After head trauma, the cytokines and hormones (e.g. cortisol, adrenaline, noradrenaline and glucagon) released cause metabolic changes resulting in catabolism and hypermetabolism, which is detrimental to the patient. Nutrition should be provided to the patient within 72 hours of the injury, as this has been shown to improve neurological recovery.

Dose	Indications	Effects	Time to effect	Duration of effect	Monitoring	Notes
Osmotic diuretic e.g. Mannitol						
0.5–1.5 g/kg (over 15 minutes as a bolus) doses appear to be equally effective	To reduce ICP once intravascular volume is stable	Immediate plasma-expanding effect to reduce blood viscosity, increase cerebral blood flow and brain oxygenation. A few minutes later vasoconstriction occurs, which reduces ICP	Few minutes	2–8 hours	Monitor electrolytes and keep within the normal range	Repeated administration can cause: ■ Diuresis ■ Volume contraction ■ Intracellular dehydration ■ Hypotension ■ Brain ischaemia ■ Increased serum osmolality ■ Renal vasoconstriction and renal failure
		Extravascular oedema in normal and damaged brain parenchyma is drawn into the intravascular space by reversing the blood–brain osmotic gradient.	15–30 minutes		If possible, measure serum osmolality and keep at or below 320 mOsm/l to reduce the risk of renal failure. (Measured not calculated osmolality should be used as mannitol is one of the unmeasured osmoles in the calculation)	Can be used in patients with intracranial haemorrhage
		Reduces CSF production and scavenges free radical species				Reverse osmotic shift can occur if repeated doses of mannitol are given above the recommended doses, as the mannitol will accumulate in the extravascular space and cause brain oedema
						It is no longer recommended to give furosemide with mannitol, as recent evidence suggests that it is ineffective at reducing cerebral oedema. Furosemide will also reduce intravascular volume
Hypertonic crystalloids e.g. Hypertonic saline 7%						
4 ml/kg over 5–10 minutes in dogs 2 ml/kg over 5–10 minutes in cats	Treatment of raised ICP in hypovolaemic or euvolaemic patients	Osmotically draws fluid into the intravascular space from the extravascular space, as sodium is unable to freely cross the intact blood–brain barrier	Minutes	Volume expansion only lasts 15–75 minutes	Monitor for: Hypernatraemia Dehydration	Rebound hypotension is uncommon (compared to mannitol) as sodium is actively reabsorbed in the kidney, especially in hypovolaemia
		Volume expansion				Hypertonic saline used in combination with synthetic colloids can prolong the volume expansion
						Use with isotonic crystalloids to prevent dehydration
		Positive inotropic effects				Do not use in hyponatraemic patient, as a rapid rise in sodium can cause pontine myelinolysis and further neurological deterioration
		Beneficial vasoregulatory and immunomodulatory effects				

Figure 9: Osmotic diuretic *versus* hypertonic saline for treatment of raised ICP

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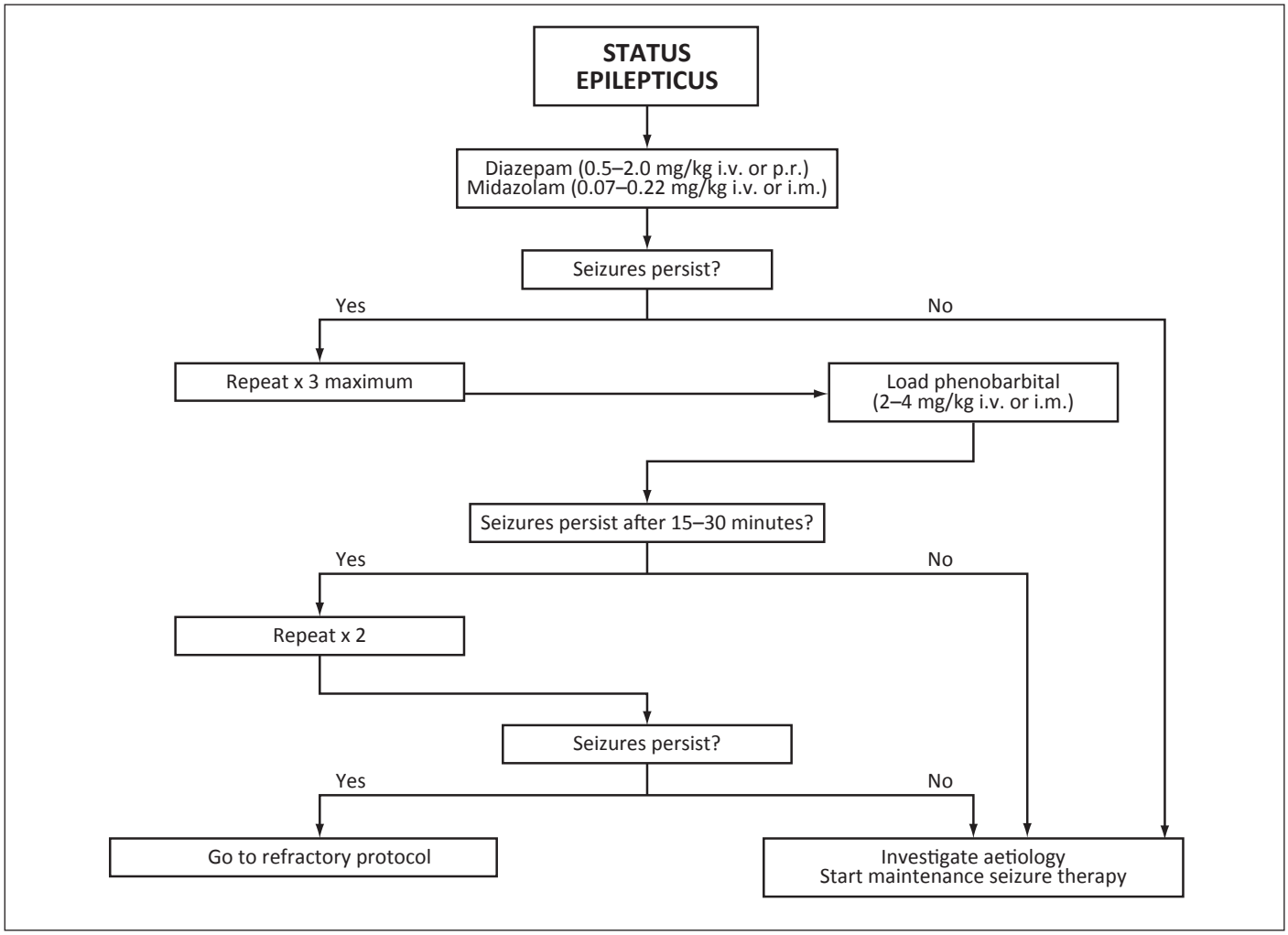

Figure 10: Approach to the initial pharmacological management of the status epilepticus patient
Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

Options for feeding tubes include:

- Nasogastric tube
  - Care, as placement can induce sneezing which can increase ICP
  - Oesophagostomy tube
    - Requires intact brainstem and oesophageal function
    - General anaesthesia is needed for placement and so should be combined with diagnostic investigation
  - Gastrostomy tube
    - Can be used in patients with brainstem lesions and poor oesophageal function
    - General anaesthesia is needed for placement and so should be combined with diagnostic investigation

The patient's blood glucose should be closely monitored, as hyperglycaemia can cause cerebral acidosis, further neuronal injury, increased free radical production, glutamate release and cerebral oedema, and so should be avoided.

Hyperglycaemia may also occur secondary to the brain injury, due to the body's response to trauma. In veterinary patients, hyperglycaemia is correlated with the severity of the head trauma, although it has not been correlated with outcome.

Pain management

The patient should be carefully assessed for signs of pain, as pain can cause an increase in ICP. The patient's mentation can be problematic to assess, as it is likely to be affected by the injury (e.g. the patient maybe delirious, obtunded, stuporous or comatose). Opioids can be given but the clinician should monitor for respiratory depression and hypotension.

Nursing care

It is vitally important that these patients receive excellent nursing care to aid in their recovery. Things to consider are listed below but should be tailored to the individual patient. Further information can

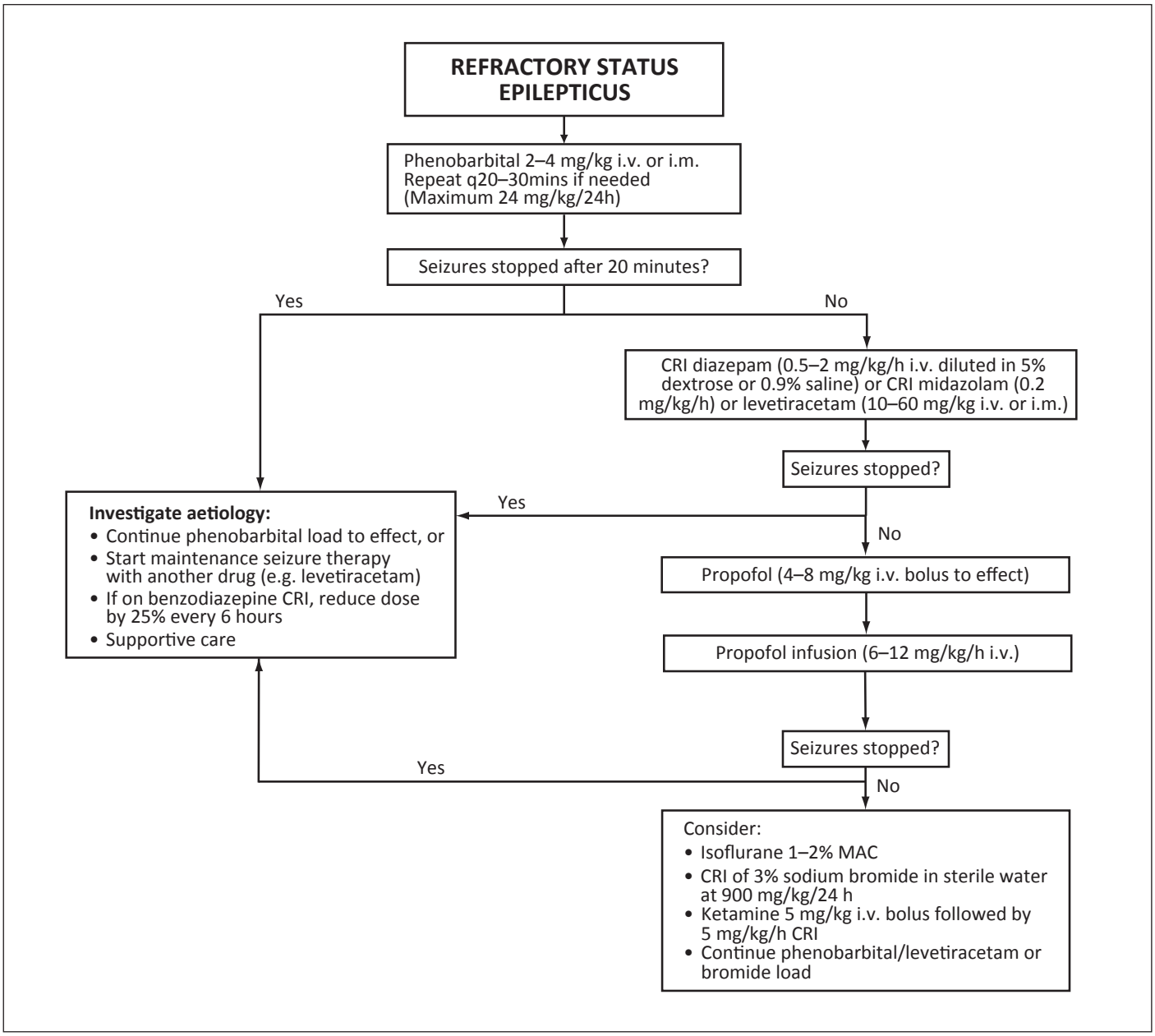

Figure 11: Approach to the pharmacological management of the refractory status epilepticus patient
Reproduced from the *BSAVA Manual of Canine and Feline Neurology, 4th edn*

be found in Chapter 25 of the *BSAVA Manual of Canine and Feline Neurology, 4th edition*.

- Appropriate bedding (i.e. mattress, with clean, dry bedding which is changed regularly)
- Padding between the limbs and over pressure points (e.g. 'doughnut')
- Turning every 4–6 hours
- Oral care – wet the tongue and gums, remove excess secretions from mouth and pharynx using suction
- Eye care – clean the eyes and apply lubrication if the patient is unable to blink
- Bladder care – monitoring bladder size and expression when large as long as there are no contraindications. Appropriate management of an indwelling catheter and closed collection system
- The patients should be checked regularly for defecation and cleaned promptly to prevent skin damage. Clipping of the hair around the perineum can be useful, as well as an application of a barrier cream
- Physiotherapy (i.e. massage and range of motion) – to maintain range of motion and muscle mass and to prevent stiffness as long as there are no contraindications
- Grooming – is important for the patient's general wellbeing and can reduce stress associated with hospitalization, especially in cats

Prognosis

For patients with a severe head trauma the prognosis is guarded to poor. However, many patients who appear to have a hopeless prognosis may recover given time and appropriate therapy. The first 24–48 hours are crucial after a brain injury to see if the patient is likely to respond.

Patients can show neurological improvement for weeks to months after a brain injury. However, they can be left with residual neurological deficits, the severity of which is extremely difficult to predict and so the client needs to be fully educated. Concurrent neurological or non-neurological injuries also need to be taken into account for the patient's prognosis. ■