resuscitated to remove the impact of shock on neurological function.

TBI is divided into primary and secondary injury. Primary injury is that which has already occurred as a direct result of the trauma, such as concussion or contusion. Secondary injury begins minutes after the trauma but may last several weeks and the target for treatment is to minimize neurological injury following trauma. Prevention of hypotension and hypoxaemia are the most important aspects of extracranial stabilization of brain injury.

Intracranial management of brain injury is focused on prevention of an increased intracranial pressure (ICP) that can result in brain herniation and sudden death. Trauma patients are highly susceptible to increases in ICP due to the fixed volume of the skull, with limited space for any oedema or haemorrhage. Severe acute increases in ICP can result in the Cushing reflex, requiring immediate intervention.

There are many aspects to treatment of the patient with severe TBI. Oxygen therapy should be provided to prevent harmful brain hypoxia but should also be titrated to prevent hyperoxia. More aggressive respiratory support may be needed to treat any hypoventilation. Fluid therapy to maintain adequate perfusion is essential, with 0.9% sodium chloride being the isotonic crystalloid fluid of choice at present. Opioid analgesia is also considered an essential part of treatment.

Specific treatment strategies include hyperosmolar therapy to treat intracranial hypertension if present. Mannitol should be given to euvoelaemic patients with increased ICP only and not on a prophylactic basis. Hypertonic saline would be the fluid of choice for patients with evidence of increased ICP and hypovolaemia. Elevation of the head by 15–30 degrees and avoidance of jugular vein compression is also important. There is no evidence that high-dose methylprednisolone is effective. Any seizure activity occurring post TBI requires urgent therapy.

Whilst intracranial imaging has previously been considered for patients with TBI, its use should be reserved for those failing medical management and those for

whom the treatment course will be altered as a result of imaging.

Good supportive and nursing care is essential for a good patient outcome following head trauma. The ultimate prognosis depends on the severity, cause and nature of trauma, as well as the treatment given, but small animals with head trauma have a tremendous ability to compensate and recover from trauma back to a normal quality of life.

KEY LEARNING OBJECTIVES

- Learn how to fully assess the patient with head trauma to detect any traumatic brain injury (TBI)
- Learn about emergency treatment of brain injury
- Understand long-term possible sequelae of head trauma

MULTIPLE CHOICE QUESTIONS

1. Which one of the following is NOT a category assessed in the Modified Glasgow Coma Scale?
 - (A) Brainstem reflexes
 - (B) Motor activity
 - (C) Pain sensation
 - (D) Level of consciousness
2. Which one of the following is NOT a consistent finding seen in the Cushing reflex?
 - (A) Bradycardia
 - (B) Systemic hypertension
 - (C) Decreased mentation
 - (D) Hyperglycaemia
3. Which one of the following is NOT indicated in patients with traumatic brain injury?
 - (A) Methylprednisolone
 - (B) Hyperosmolar therapy
 - (C) Analgesia
 - (D) Intravenous fluid therapy

Uroabdomen: presentation, diagnosis and management

Lindsay Kellett-Gregory

Uroabdomen is defined as leakage of urine into the peritoneal and/or retroperitoneal space, and may occur secondary to trauma, urinary tract obstruction, neoplasia or dehiscence following urinary tract surgery. Consequences include azotaemia, hyperkalaemia, metabolic acidosis, dehydration and, in some cases, shock. These result from retention of potassium and other waste products, in addition to third spacing of fluid into the peritoneal cavity. The chemical peritonitis resulting from

direct contact of urine with the peritoneum may cause acute abdominal pain in some cases.

Clinical signs of uroabdomen are typically non-specific and may include vomiting, anorexia and lethargy. Physical examination findings may include evidence of hypoperfusion, cardiac arrhythmias and abdominal pain. The ability to palpate the bladder does not rule out the possibility of a uroabdomen. Patients with urethral injury may develop inguinal or perineal swelling and extensive bruising.

A diagnosis of uroabdomen is based on a combination of the patient's history and physical examination findings, laboratory assessment of peripheral blood in comparison to abdominal fluid, and imaging findings. A biochemistry panel including electrolytes to detect any hyperkalaemia and azotaemia should be performed in all suspected cases. The degree of hyperkalaemia and azotaemia may be variable, with abnormalities progressing over time. The rise in blood urea nitrogen may be

proportionally greater than the rise in creatinine with uroabomen in comparison to other causes of azotaemia.

Abdominocentesis is the most useful diagnostic tool to confirm the presence of a uroabdomen although it does not provide any information as to the site of the injury. Biochemical testing of the fluid collected and comparison to blood should be performed to confirm a diagnosis. The potassium and creatinine concentrations of abdominal fluid will be higher than in blood in patients with uroabdomen (Figure 1). Fluid cytology is also important to detect cases of urosepsis and associated septic peritonitis.

Plain abdominal radiographs are poorly sensitive for the detection of uroabdomen but may show additional relevant abnormalities such as urolithiasis or pelvic fractures, depending on the cause. Contrast radiographic studies are of use to detect the location of injury to the urinary tract and plan definitive treatment following patient stabilization.

Patient stabilization should be prioritized and may include fluid therapy to correct any hypoperfusion and management of any clinically significant hyperkalaemia. Placement of a urinary catheter connected to a closed collection system is useful to prevent further leakage of urine into the abdomen. Peritoneal drainage is also indicated. A cystostomy tube may be considered to provide urinary diversion in cases of urethral rupture. Once the patient is more stable and any concurrent traumatic

injuries have been addressed, surgical correction may be considered where indicated.

KEY LEARNING OBJECTIVES

- Learn clinical presentation of a uroabdomen
- Understand emergency stabilization of the patient with a uroabdomen
- Know how to confirm a diagnosis of uroabdomen in practice

MULTIPLE CHOICE QUESTIONS

1. Which one of the following treatments would be the most appropriate first-line action when treating a patient with a uroabdomen and cardiovascular compromise from hyperkalaemia?
(A) Intravenous calcium gluconate
(B) Drainage of fluid the abdominal cavity
(C) Intravenous regular insulin and dextrose
(D) Intravenous dextrose
2. What is the optimal peritoneal fluid to blood ratio of creatinine in order to confirm a diagnosis of uroabdomen in dogs?
(A) 4:1
(B) 2:1
(C) 1.9:1
(D) 1.4:1
3. Which of the following drugs would be most appropriate for analgesia in a patient with a uroabdomen?
(A) Ketamine
(B) Dexmedetomidine
(C) Methadone
(D) Meloxicam

	Abdominal fluid:peripheral blood creatinine	Abdominal fluid:peripheral blood potassium
Dogs	>2:1 (86%, 100%) ^a	>1.4:1 (100%, 100%) ^a
Cats	>2:1 ^b	>1.9:1 ^b

Figure 1: Abdominal fluid to peripheral blood ratios of creatinine and potassium in uroabdomen (^asensitivity, specificity; ^bsensitivity and specificity not reported).

Assessing dirty wounds

Samantha Woods

Immediate management of the open wound aims to reduce microbial numbers and prevent further contamination. In an emergency situation, this is likely to consist of wound lavage, broad-spectrum antimicrobial therapy and application of a dressing, with plans to perform more definitive assessment and debridement once the patient is stable.

Lavage of wounds is important to remove gross contamination and reduce the likelihood of infection developing. Copious quantities of lavage solution are needed in the first instance. Tap water, high-volume, low pressure lavage with sterile saline and jet irrigation of wounds are all potential methods of wound decontamination.

Topical antimicrobial therapy may be applied early in wound management to assist in the reduction of surface

bacteria. Appropriate systemic antibacterial therapy may be chosen to treat wound infections, based on culture and sensitivity but in the early stages of management, broad-spectrum topical therapies are preferred.

Any open wound should be covered to avoid the potential for wound contamination, provide comfort to the patient, reduce further bleeding and protect the wound surface. Dressings should be left in place until definitive management is undertaken.

Once the patient is stable, the dressing is removed, the area around the wound is aseptically prepared and the wound is debrided to remove any foreign material or necrotic debris. Surgical debridement is performed where possible, although further debridement may be achieved by choosing appropriate debridement dressings.

Any part of the wound that can be closed surgically should be closed – this reduces the open surface area and therefore time to healing. A moist wound bed is maintained to facilitate granulation tissue formation and epithelialization. Some wounds will heal fully via second-intention healing, in others a healthy granulation bed is