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Emergency Surgery Mini Series

Session 1: Trauma Surgery and Respiratory Emergencies

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Webinar notes: Trauma Surgery and Respiratory Emergencies

Introduction:

Trauma is a frequent presentation in general practice situations and knowing how to deal with emergency situations and direct your team can significantly reduce patient morbidity and mortality and improve streamlining of diagnostics and treatment. This webinar aims to give participants an overview of initial patient assessment in emergency situations and then looks in more detail at some of the more common soft tissue emergencies seen associated with trauma patients. In the second part of the webinar we will look briefly at the management of brachycephalic obstructive airway syndrome and laryngeal paralysis.

Triage:

Triage is the process of prioritising patients based on their need and the severity of their condition. Triage begins with initial owner contact to the practice. The reception team can aid in the prioritisation of patients by asking specific questions to owners during phone contact to determine the severity of the situation. These questions include:

- What is the nature of the injury?
- Is the patient breathing/conscious/ambulatory?
- Can the owner determine mucous membrane colour?
- Is the patient bleeding and if so where from?
- Is there a history of vomiting, diarrhoea/non-productive retching?

It is important that owners are made aware of the risk of sustaining a bite wound when transporting painful patients or in patients which are seizuring. Bear in mind the owner's interpretation of situation may be unreliable as they may be very upset. Ensure the advice given is simple advice e.g. apply pressure to stem bleeding, and ensure the owner knows what to do on arrival at the practice and who to report to.

Upon arrival at the practice, an initial assessment should be made to determine if the patient needs to be removed for its owner immediately for emergency treatment or if the patient can remain with the owner during the consultation. This decision is often made by a member of the veterinary nursing team. Initial points for assessment at this juncture include:

- Is the patient conscious, responsive and ambulatory?
- Is there evidence of haemorrhage?
- Is the airway patent?
- Observe respiratory rate, noise and effort (inspiratory versus expiratory)
- Assess heart rate, pulse quality, mucous membrane colour, capillary refill time
- Is analgesia required?
- Is oxygen supplementation required?

Rapid analgesia is essential for the majority of patients presenting post trauma. Analgesia should be tailored to the individual but an opioid (and preferably a full mu agonist e.g. methadone) is usually indicated. Realistically the detrimental effect of pain on normal respiration (e.g. fractured ribs) will usually far outweigh the respiratory depression caused by opiods. If an opioid alone is inadequate, other options for analgesia include either a ketamine constant rate infusion (CRI) or a lidocaine CRI. Intravenous paracetamol can also be considered (10mg/kg as a slow intravenous infusion over 15-30 minutes). Non-steroidal anti-inflammatory drugs e.g. meloxicam should be avoided in hypovolaemic, hypotensive patients as there is an increased risk of renal toxicity and gastric ulceration. These drugs can be introduced once the patient is stable and any volume deficits have been corrected.

Primary survey:

The primary survey aims to assess the most essential body systems i.e. respiratory, cardiovascular, urinary and neurological systems. Trauma to these systems can lead to significant morbidity and mortality and is unfortunately not uncommon. In one study of 235 dogs experiencing severe blunt trauma, pulmonary contusions were reported in 58% of all patients and pneumothorax in 47% (Simpson et al 2009).

Signs of respiratory compromise include dyspnoea, tachypnoea, increased abdominal effort, stertor/stridor/wheeze, abducted elbows (orthopnea), open mouth breathing and cyanosis. Open mouth breathing in cats is usually a sign of significant respiratory or intra-pleural disease and care should be taken to stress such patients as little as possible as they can deteriorate rapidly. Observing the patient prior to handling can provide you with a significant amount of information regarding the possible source of respiratory distress. Increased noise or effort on inspiration suggests an issue with the upper respiratory tract. Stertor is more commonly associated with pharyngeal obstructions whereas stridor is seen with laryngeal oedema/obstruction. Expiratory effort is more commonly seen with lower respiratory disease.

When presented with a patient in respiratory distress, oxygen should be provided if tolerated by the patient. Ideally a patient will be maintained in sternal recumbency to prevent atelectasis of the dependent portion of the lung. Analgesia should be provided if appropriate. Observe the patient as detailed above and then auscultate the thorax. Lung sounds should be audible in all four quadrants of the thorax and an absence of lung sounds can indicate the presence of free air or fluid within the pleural cavity. Intravenous access should be obtained as soon as it is practical to do so. Ensure you place as large a bore catheter as possible. Some patients experiencing respiratory distress, particularly those with upper respiratory tract obstruction, can become hyperthermic and this can further exacerbate respiratory distress due to panting. Actively cooling such patients through the use of air conditioning, fans or wet towels should be considered. Sedation is also useful in these patients. In trauma patients, however, hypothermia is more common and active warming through the use of warm air blankets such as a Bairhugger or a heated incubator is indicated. The use of heat pads in such patients could lead to thermal burns as patients with significant musculoskeletal trauma may be unwilling or unable to move away from the heat source.

Oxygen supplementation can be provided via a number of routes. Face masks provide a maximum fractional inspired oxygen (FiO2) of 50-60% with oxygen flows of 8-12L/min. More commonly flows of 2-5L/min are used. There is a risk of rebreathing and also a risk of hyperthermia.

Nasal prongs/catheters achieve a FiO2 of ~50% can be achieved using flows of 2l per 10kg per minute. However this is often not well tolerated in clinical patients and 2-5 l/min is more commonly used. It is important to humidify gas, if possible, to prevent irritation of the nasal mucosa. Oxygen cages and incubators can achieve FiO2 of up to 60% (more commonly 40-50%) but there is again a risk of hyperthermia and patient temperature should be closely monitored.

Thoracic ultrasound is a quick and easy way of determining if pleural fluid is present and can be performed with the patient in sternal recumbency and with minimal handling. Thoracic radiography is the imaging modality of choice for the diagnosis of significant pneumothorax as it can be performed relatively quickly in the conscious patient. However, this does require some degree of restraining which may not be tolerated in the unstable patient. Diagnostic needle thoracocentesis can be considered but can be unreliable in very large patients or patients where the fluid or air is compartmentalised.

Basic assessment of the cardiovascular system should include assessment of:

- Mental state
- Mucous membrane colour - pale/cyanotic/hyperaemic
- CRT ↑ or ↓
- Heart rate
- Pulse Quality
- Presence of peripheral (digital) pulses
- Temperature of extremities
- Blood pressure

If possible, a continuous non recording ECG should be attached to the patient. Ventricular premature complexes, ventricular tachycardia and junctional rhythms can all be seen post significant trauma. Minimum bloodwork should include PCV/TP/electrolytes/urea/creatinine/acid base measurements/lactate and glucose if possible. Lactate can be a good indicator of peripheral perfusion and has been shown in some studies to be a predictor of survival in certain emergency situations. Normal lactate readings are usually <2mmol/L. However this is not essential. Glucose can be low in septic patients.

Potential underlying aetiologies of abnormal cardiovascular parameters include reduced systemic vascular resistance (sepsis, hypovolaemia or anaphylaxis), structural cardiac disease and hypoxic damage to cardiac muscle and pain.

Crystalloids represent the mainstay of initial fluid resuscitation although it should be remembered around 75% of the volume given will be lost to the interstitium within one hour of administration. A balanced electrolyte solution is preferable to 0.9% saline as the latter can lead to metabolic acidosis secondary to elevated chloride levels.

Rather than considering “shock-rate” fluids, it is preferable to give intravenous fluids in incremental boluses (10-20ml/kg given over 15 minutes) and monitor heart rate (which should reduce) and blood pressure (hypotension should improve) to assess response. Please note these doses may not be appropriate in patients with concurrent structural cardiac disease. Hypertonic saline can also be used (although the author has limited experience of it) and causes expansion of circulating volume by dehydration of the interstitium. It must therefore be followed by an isotonic solution.

In patients which are unresponsive to crystalloid therapy or where it is anticipated that improvement of oncotic pressure will be required, colloids should be considered. Options for colloid therapy include hydroxyethyl starches and gelatins. Hydroxyethyl starches come in a variety of molecule sizes and therefore have variable duration of effect. There has been significant research efforts within human medicine over the past few years looking at the morbidity and mortality associated with the use of hydroxyethyl starches in critically ill patients. Recent randomised trials in human medicine have suggested that the use of hydroxyethyl starches in critically ill patients can lead to significant acute kidney injury and mortality; particularly in sepsis. This has led to a move away from the use of such colloids in veterinary medicine also although the same research has not currently been duplicated. At the present time, the use of hydroxyethyl starches in human medicine is limited to management of haemorrhage and certainly there is an argument that the use of such colloids should be avoided in septic veterinary patients also. Coagulopathy is reported as a side effect of hydroxyethyl starches due to a reduction in the concentration of factor VIII and von-Willebrand factor but this is not frequently recognised clinically.

Gelatins are generally smaller sized molecules than those contained in hydroxyethyl starch solutions but are present in larger numbers. As the molecules are smaller, they do not remain in the circulation as long as the hydroxyethyl starch molecules. Increased anaphylaxis is reported in humans treated with gelatins than hydroxylethyl starches but this is not very common in dogs. Gelatins are currently the colloid of choice in human sepsis. Colloids are usually administered in 5ml/kg boluses up to a maximum of 20ml/kg/24 hours.

In patients where significant blood loss is the underlying cause of hypovolaemia, replacement of circulating volume is best achieved using either packed red cells (which provide a higher number of erythrocytes/ml and thus will increase oxygen carrying capacity but do not replace clotting factors or plasma proteins) or whole blood which provides all blood components although there is reduced survival of platelets. Since the introduction of the Pet Blood Bank, packed red cells are often more readily available and represent convenience but whole blood continues to have significant benefits in patients where there has been significant haemorrhage. Packed cells can be combined with fresh frozen plasma (usually 10-20ml/kg or 1:1 ratio of bag of packed cells: fresh frozen plasma); however this does not address thrombocytopaenia.

Cardiovascular resuscitation goals:

- **Normalization of heart rate and restoration of peripheral pulses.**
- **Mean arterial blood pressure (MAP) > 65mmHg.**
- **Urine output > 0.5 ml/kg/hr averaged over at least four hours.**
- **Lactate levels < 2 mmol/l.**

Primary neurological assessment can be brief but should be performed in a logical manner. Assessment of demeanour can indicate signs of increased intra-cranial pressure or diffuse brain disease. Perform a basic cranial nerve examination. Is the patient ambulatory? If not, is there still voluntary movement of the limbs (do not confuse this with segmental spinal reflexes)? A patient with voluntary movement will have deep pain sensation and therefore this only needs to be assessed if voluntary movement is not present. When assessing deep pain sensation, ensure your patient makes a visible response to the stimulus you apply. If the reflex arc is intact, a patient with a completely severed spinal cord will still withdraw the limb when an interdigital stimulus is applied.

Secondary survey:

The secondary survey should be performed once your patient has been stabilised as detailed above. A full patient history should also be obtained at this time if this has not already been done. This should include confirming a full physical examination to identify both more subtle lesions and also concurrent disease processes such as cardiac disease or diabetes mellitus which may impact ongoing patient management. Once your physical examination is complete, make a problem list and subsequently a list of differential diagnoses. With this information, appropriate diagnostic tests can be chosen. Obviously, body systems should be prioritised on the basis of the potential morbidity; e.g. pneumothorax is of higher priority than a fracture. Diagnostics can include thoracic and abdominal radiography, ultrasound and advanced imaging techniques such as CT and MRI.

Pneumothorax:

Intarapanich et al (2016) reviewed 426 cases of blunt trauma and reported a 29% incidence of pneumothorax and 44% incidence of pulmonary contusions in patients experiencing motor vehicle trauma. Sources of air leakage include thoracic wall trauma, oesophagus or airway (lung, bronchus or trachea) rupture. This usually occurs secondary to blunt thoracic wall trauma but can be spontaneous due to rupture of pulmonary bullae or blebs (which usually occurs secondary to minimal trauma). Clinical signs of pneumothorax include a restrictive (shallow) breathing pattern, hypoventilation and reduced lung sounds on auscultation (particularly dorsally). Life-threatening dyspnoea is seen in patients with a tension pneumothorax. This occurs when the entry point of air in to the chest cavity acts like a one way valve, leading to a rapid and life-threatening accumulation of free air in the thorax. This represents an emergency and thoracic drainage is required rapidly. Should clinical examination raise suspicion for the presence of a pneumothorax, radiography is the simplest diagnostic tool as previously described.

Management of a pneumothorax depends on the severity of the air leak, likelihood of recurrence and patient stability. A small volume pneumothorax with no ongoing air leakage may not require any drainage if the patient is asymptomatic and any free air will be resorbed over the coming days. When physical examination findings suggest a clinically significant pneumothorax, options for drainage include needle thoracocentesis and the placement of a thoracostomy tube. The author tends towards needle thoracocentesis in conscious trauma patients in the first instance as this can be performed utilising local infiltration of lidocaine. The needle should be positioned dorsally within the thorax and may need to be repositioned if initial attempts are not successful. In situations where a negative seal cannot be achieved or there is rapid re-accumulation of air within the thoracic cavity, a thoracostomy tube should be placed. This requires general anaesthesia and therefore needle thoracocentesis may need to be performed initially to stabilise the patient.

Very infrequently, e.g. in the case of rupture of a bronchus, an emergency thoracotomy may be required to address the underlying cause due to the rapidity of air loss in to the thoracic cavity.

Three main types of thoracostomy drain are available but only two are suitable for closed placement (The third type is reserved for placement when the thoracic cavity is open at surgery). These are the traditional "trochar" drain and Seldinger drains where a peel-away sheath is used to place a guide wire through the thoracic wall over which the drain is threaded. As much of the clipping and aseptic preparation should be performed prior to induction of anaesthesia to reduce anaesthesia time.

Patients with a thoracostomy tube in situ require constant supervision and a rigid protection collar should be in place to prevent interference with the drain. A gate clamp should also be in place. Drainage frequency should be determined on the basis of production of either air or fluid. In patients where there is significant air leakage, a Heimlich valve can be considered. This valve is connected to the end of the drain and when pressure in the thoracic cavity exceeds atmospheric pressure, air is discharged from the thorax through a rubber sleeve. This valve is not suitable for use on patients with a body weight of <20kg. The valve can be cumbersome and should be carefully monitored for dislodgement. Furthermore the valve ceases to function if it becomes wet so is not suitable for use in patients with pleural fluid.

Surgical exploration of the thorax should be considered in any patient with significant ongoing air leakage which does not respond to conservative management. However, in the author's experience this is not common.

Diaphragmatic rupture:

Diaphragmatic rupture or herniation is a well-recognised consequence of severe blunt trauma particularly in smaller patients. The mechanism of injury is thought to be a sudden increase in intra-abdominal pressure with the glottis open. The diaphragmatic costal muscles are the most common area of rupture and tears can be circumferential, radial or a combination of both (circumferential tears are more common in cats than dogs). The liver is the most commonly herniated organ followed by small intestine, stomach, spleen, omentum and the pancreas. Herniated organs may become strangulated or obstructed and liver herniation can lead to venous stasis, necrosis, bacterial proliferation, biliary obstruction and the development of pleural effusion.

The chief clinical sign is dyspnoea due to lack of a functioning diaphragm, lung compression by viscera or pleural fluid, dysfunction of the chest wall secondary to trauma/pain and concurrent pulmonary contusions. Other signs include reluctance to lie in lateral recumbency, lethargy, vomiting or diarrhoea (if gastrointestinal involvement) although some patients may be asymptomatic initially. Examination can reveal muffled or abnormally positioned heart sounds, reduced lung sounds and an "empty" feeling abdomen. **ALL** patients presenting with musculoskeletal injury secondary to blunt trauma should have thoracic radiographs or ultrasound performed. Cardinal radiographic signs (66-97% accuracy of diagnosis is reported with a single lateral projection) include loss of the diaphragmatic contour and visualisation of viscera in the thoracic cavity. Thoracic ultrasound (93% accuracy) is especially useful if pleural fluid is present.

Historically, delaying surgical repair of a diaphragmatic rupture was thought to correlate with increased survival to discharge. However this is not supported by more recent publications.

Indeed surgery should be performed as soon as the patient is stable as further delay risks further herniation of organs or incarceration of herniated organs which can be of particular significance with respect to organs such as the stomach where gas distension can lead to a rapid deterioration in the patient's clinical status. Gibson et al 2005 reported 92.6% of cases undergoing surgery within 24 hours and had a 93% survival to discharge rate.

Well managed anaesthesia is the key to successful surgical management of diaphragmatic rupture. Ideally clipping should be performed prior to induction of anaesthesia as insofar as this is tolerated by the patient in an attempt to reduce anaesthesia time. Pre-oxygenation should be performed and once anaesthesia is induced, the thorax should be elevated above the abdomen to reduce pressure on the thorax and discourage herniation of further organs in to the thorax. Ventilation is required for this surgery as the thoracic cavity will be open to the environment via the diaphragmatic defect. High inspiratory pressures (>20mmHg) should be avoided as this can increase the risk of development of re-expansion pulmonary oedema.

Once these patients are anaesthetised you need to move quickly and reduce the hernial contents as quickly as possible. Once there is more room for pulmonary expansion, anaesthesia often becomes much more stable. Sometimes the size of the hole in the diaphragm is too small to allow easy reduction of the herniated organs. In this case, the defect may need to be enlarged with a ventrally (i.e. towards the surgeon) directed incision under direct visualisation. Be careful of the vena cava and oesophagus as they pass through their respective hiatuses in the diaphragm. Incarcerated liver lobes and the spleen are often congested and friable. Careful handling is therefore required if rupture is to be avoided. In more established hernias, adhesions may have formed and a caudal median sternotomy may need to be performed to allow these adhesions to be transected under direct visualisation. Once reduced to a "normal" anatomical location, organs should be inspected for viability. The defect can usually be closed using an absorbable monofilament suture such as polydioxanone (2/0-0) in a simple continuous pattern.

Drainage of the surgically induced pneumothorax needs to be considered prior to closure of the diaphragmatic defect. In acute cases where a reasonable volume of aerated lung remains, the thoracic cavity can be drained via a large bore intravenous catheter which is passed through the muscular portion of the diaphragm under direct visualisation and connected to an extension set and a three way tap for drainage. In more chronic ruptures or in cases where there is very little aerated lung identified on the pre-operative thoracic radiographs, a thoracostomy tube should be placed to allow for more gradual re-expansion of chronically atelectic lung over the first 24 hours post-surgery. Rapid re-expansion can result in reperfusion injury of collapsed vascular beds and the development of pulmonary oedema.

Oxygen supplementation is often required in recovery. The patient should be maintained in sternal recumbency to prevent lung atelectasis. If hypoxia is noted post extubation consider a conscious chest radiograph to assess the degree of remaining pneumothorax and drain more air as required. Approximately 15% of cats die prior to surgery due to multiple comorbidities. Death within the first 24 hours post-surgery is usually secondary to haemothorax, pneumothorax, pulmonary oedema, pleural effusion and cardiac dysrhythmias. Other post-operative complications include ascites, gastric ulceration, oesophagitis, megaoesophagus and recurrence of the hernia.

Body wall rupture:

Another potential consequence of blunt abdominal trauma is body wall rupture. Inguinal, prepubic, intercostal and paracostal locations are the most common. Bite wounds can also lead to penetrating abdominal wounds. 44-75% of patients will have concurrent soft tissue or orthopaedic injuries. The hernia may not initially be obvious depending on the degree of soft tissue swelling and volume of abdominal contents which are herniated. Thoracic radiography should also be performed to rule out concurrent diaphragmatic herniation.

In many cases, a diagnosis of body wall rupture can be made on palpation or visualisation of herniated organs in the cases of penetrating injury. However in some more subtle cases, radiography (plain or contrast) or ultrasound is needed to confirm the diagnosis.

Patient stabilisation, surgical timing and preparation have been previously described. Ideally, hernias/ruptures should be explored via a ventral midline celiotomy incision to allow exploration and examination of ALL abdominal organs. Incision directly over the herniated contents is also described. Hernial contents should be reduced via gentle traction and any necrotic or friable tissues debrided. The wound and abdominal cavity should be thoroughly lavaged prior to closure. Selection of suture material is important. Ideally a monofilament absorbable suture e.g. polydioxanone should be used as and the hernia should be apposed in anatomical layers. In patients with significant defects in the muscle of the abdominal wall, it is occasionally necessary to use an autogenous muscle flap to reconstruct the abdominal wall. Options include the external abdominal oblique or latissimus dorsi for caudal thoracic or lateral cranial abdominal wall defects. Autogenous tissues have the significant benefit of bringing a blood supply to the area resulting in a much lower complication rate.

In defects where appropriate autogenous tissue cannot be sourced to close the defect, polypropylene mesh can be used for reconstruction of the abdominal wall. Edges of the mesh should be double thickness and it should be attached to surrounding musculature using simple interrupted or horizontal mattress sutures of polydioxanone or polypropylene. Ideally omentum should be sutured in to the defect prior to placement of the mesh to reduce the risk of serosal adhesions. In patients where there is significant necrosis of tissues and wound contamination, mesh should not be used as if bacteria become attached to the implant, it can be very difficult to eradicate this without removal of the implant leading to the development of draining fistulae. In rare cases where there is a large necrotic defect where closure with autogenous tissues is not an option, the use of negative pressure wound dressings to temporarily seal the abdomen until the tissues are healthy enough to permit closure via another route is reported.

In paracostal ruptures, the first priority is always re-establishment of the integrity of the thoracic cavity and therefore efforts should focus on turning caudal thoracic defects in to cranial abdominal defects. This can be achieved by advancing the diaphragm and re-attaching it to a more cranial rib using cirumcostal sutures. This suture placement technique can also be used to re-attach avulsed portion of the abdominal wall musculature. In pre-pubic tendon rupture, hernial contents are reduced via a ventral midline celiotomy incision as previously described. If there is sufficient soft tissue remaining on the pubis, the defect can be closed using cruciate mattress sutures of polydioxanone.

However (and more commonly) holes need to be pre-drilled in the cranial brim of the pubis to facilitate reattachment of the musculature of the body wall to the pubis via simple interrupted or horizontal mattress sutures. Ensure you protect the structures running dorsal to where you are drilling with a periosteal elevator or swab and avoid interfering with the obturator nerve which runs through the obturator foramen.

Ensure adequate analgesia provision (opioids +/- NSAIDs depending on hydration status) and consider the use of a wound diffusion catheter depending on the extent of the injury. Wound diffusion catheters are flexible fenestrated catheters placed at or near to the site of surgery for the intermittent delivery of local anaesthetics as part of a multi-modal analgesia plan during hospitalisation. Local anaesthetics work by blocking the influx of sodium into the nerve axon and inhibiting the action potential which disrupts the generation and transmission of nerve impulses. This approach provides excellent local analgesia and thus reduces dependence on systemic analgesics which in turn can improve appetite etc. The author uses bupivacaine (1-1.5mg/kg q6 hours) and the dosing frequency should be strictly adhered to if possible as injection in to a wound bed can cause a stinging sensation if the effects of the previous dose have completely worn off. Asepsis should be strictly adhered to when handling the catheter and a bacterial filter should be placed on the end of the catheter which should be changed every 24 hours. In patients with polytrauma, consider placement of a feeding tube.

Urinary tract rupture:

The bladder is the most common site of urinary tract rupture. The majority of urinary bladder ruptures occur secondary to major external trauma. Male dogs have a higher incidence of urinary bladder rupture, likely due to a long, narrow urethra with a limited ability to dilate rapidly after a sudden increase in intravesicular pressure. It may also be due to a higher incidence of roaming and motor vehicle trauma in male dogs. Rupture of the urinary bladder in male cats is most often the result of forceful manual compression of a distended bladder in an attempt to dislodge a urethral plug or secondary to road traffic trauma. Trauma to the kidneys and ureters requiring surgical intervention is rare due to the protection afforded by their retroperitoneal location. Bladder rupture leads to uroperitoneum. Few clinical signs may be evident initially but there is rapid progression to azotaemia, dehydration, metabolic acidosis, hyperkalemia and ultimately death. Clinical signs associated with urethral rupture include pain, oedema and bruising secondary to subcutaneous urine leakage.

After performing your initial body system assessment and cardiovascular and respiratory stabilisation, urinary tract trauma should be considered in all patients presenting with major trauma. Fractures of the ribs, pelvis, or pelvic limbs indicate substantial abdominal trauma and should alert the clinician to the possibility of concomitant urinary tract injury. Baseline blood work should be performed to look for signs of azotemia and/or electrolyte disturbances; particularly evidence of hyperkalemia or metabolic acidosis. If possible, fluid therapy should be initiated in conjunction with establishing urine diversion. A cystostomy tube may need to be considered if the site of urethral trauma prevents urethral catheterisation. Options for cystostomy tube placement include use of a Foley catheter placed via a flank approach (Bray et al 2009) or use of a locking loop nephrostomy catheter. In rare cases, a peritoneal drainage catheter can be placed to drain fluid from the abdomen.

Potassium is normally excreted into the urine by the kidneys. In cases of uroabdomen, equilibration across the peritoneum occurs as urine accumulates in the abdomen and the potassium concentration increases. Hyperkalemia causes an increase in the resting membrane potential of cells in the body, reducing the gradient between the resting membrane and threshold potential, ultimately causing an increase in cell membrane excitability. Increased membrane excitability in cardiac myocytes results in characteristic ECG findings. Early signs include the presence of spiked T waves; eventually leading to atrial standstill. Bear in mind animals with uroabdomen will have multiple other metabolic abnormalities, including metabolic acidosis, hypocalcemia, and hyponatremia and therefore ECGs do not always follow the textbook pictures. If the patient has mild to moderately hyperkalemia with a serum K+ >5.5 mmol/L but <7.5 mmol/L, fluid therapy alone may promote potassium excretion by improving glomerular filtration rate. If serum K+ exceeds7.5 mmol/L, active steps should be taken to reduce it.

- Insulin and 50% Dextrose
    - Give 0.5 units/kg regular insulin IV and, for every unit of insulin administered, give 2 grams (4 mL) of 50% dextrose diluted IV to prevent hypoglycemia
- 10% Calcium Gluconate
    - Give 0.5–1.5 mL/kg IV slowly over 5–10 minutes. An ECG should be monitored during the infusion for evidence of bradycardia or exacerbation of the arrhythmia

The easiest way of diagnosing bladder rupture is to look for free abdominal fluid using abdominal fluid. Urethral rupture can be more challenging to diagnose although signs such as pain, subcutaneous swelling or bruising and repeated stranguria can raise suspicion for urethral trauma. If free fluid is identified, it should be sampled and the creatinine levels compared between the fluid and serum levels. Creatinine is a large molecule and it cannot equilibrate across membranes once excreted in to urine unlike urea which is smaller and therefore can. If the creatinine level in the abdominal fluid is twice that of serum levels or greater, it is highly likely you are dealing with an uroabdomen. In patients which are stable, contrast radiography is the most definitive way of confirming rupture of the urethral tract via a retrograde (vagino) urethrogram.

Bladder rupture should be managed via a ventral midline celiotomy. Small urethral tears can be managed conservatively using an indwelling silicone urethral catheter for 10-14 days. Larger tears or complete urethral rupture should be managed surgically. Options include perineal urethrostomy, transpelvic urethrostomy and prepubic urethrostomy depending on the location of the rupture.

Feeding:

Provision of assisted feeding should be considered in all polytrauma cases. Options for assisted feeding include naso-oesophageal tubes, oesphagostomy tubes, gastrotomy tubes and jejunostomy tubes. The decision should be made based on the underlying pathology, length of expected tube use, the consistency of the food which you wish to use and co-morbidities which may affect suitability for anaesthesia should all be considered. For many trauma patients, an oesophagostomy tube may be the most sensible compromise.

Pharyngeal trauma:

Pharyngeal trauma usually occurs at exercise associated with jumping to catch or running on to a stick. In acute trauma cases, the owner may not witness the trauma but will often report hearing the dog cry out or notice haemorrhage from the oral cavity. In more chronic cases, the date of the original trauma may be unknown the dog may present with cervical swelling and pyrexia. After triage as detailed above and the provision of analgesia, general anaesthesia is required to permit thorough oral examination. Bear in mind intubation may be challenging and therefore have a selection of endotracheal tubes available. Suction should be ready to use to remove blood/saliva etc from the pharynx if necessary. A good light source and laryngoscope should also be available. In patients where difficult intubation is anticipated, a rigid 6Fr or 8Fr dog catheter can be connected to a small endotracheal tube connector to facilitate oxygen delivery in to the trachea if an endotracheal tube cannot be passed.

Following induction of anaesthesia, intubation and oral examination, imaging of the cervical region and thorax should be performed. The presence of gas bubbles or gas filled tracts in the soft tissues of the neck surrounding the trachea and oesophagus is often supportive of pharyngeal penetration and warrants further surgical exploration as does the presence of a pneumomediastinum or a pneumothorax. Bear in mind that a pneumothorax can develop secondary to a pneumomediastinum but not vice versa. CT of the neck and thorax is preferred when available.

Small superficial tracts within the oral cavity can be debrided and either left open to drain and granulate or sutured depending on location. Tracts extending from the peri-laryngeal region should be explored via ventral midline approach to allow debridement and lavage. A closed suction drain should be placed. For shorter or more rostral tracts or where finances are an issue, exploration and flushing using pressurised saline passed through a rigid endoscope is also described (Robinson et al 2014) as an alternative to surgery.

Brachycephalic obstructive airway syndrome (BOAS):

BOAS is an “umbrella” term for a number of anatomical abnormalities (both primary and secondary) which lead to clinical signs of upper airway obstruction.

Primary abnormalities

- stenotic nares
- elongation and thickening of the soft palate
- tracheal hypoplasia
- excessive pharyngeal tissue
- macroglossia
- increased nasal mucosal contact points

Assessment of stenotic nares is done visually and can be done in the conscious patient. Assessment of soft palate length requires general anaesthesia.

Palate length should be assessed with the tongue in a neutral position. If the tip of the soft palate extends beyond the tip of the epiglottis more than 5mm and if there were clinical signs consistent with BOAS, the author would consider surgical management of palate length. This is not necessarily always a clear cut decision but certainly if direct laryngoscopy revealed evidence of laryngeal collapse, the author feels this adds weight to the decision for surgery. CT of the head is now becoming a more common tool in the investigation of this condition and can be used in conjunction with rhinoscopy for the assessment of the nasal turbinates.

Tracheal hypoplasia is recognised most commonly in brachycephalic breeds and represents a significant challenge to outcome in these patients as it cannot be surgically corrected. A ratio comparing thoracic inlet diameter to tracheal diameter at the level of the thoracic inlet is described by Harvey and Fink (1982). However Coyne and Fink reported no correlation between these dimensions and degree of dyspnoea (1992). Kaye et al (2015) compared CT, radiographic and endoscopic tracheal dimensions in British Bulldogs with tracheal hypoplasia. Findings indicated that some CT and radiographic tracheal diameter measurements were comparable in English Bulldogs however diameters for both imaging techniques were not comparable with tracheoscopy scores. Therefore these measurements should be interpreted with caution.

In 1957, Leonard described laryngeal saccule eversion in five brachycephalic dogs and concluded this may be a factor in the respiratory issues seen in these patients. In 1960 he further proposed that laryngeal saccule eversion was not an isolated entity but was in fact the first stage of laryngeal collapse. Obstruction of the rima glottis was caused by elongation of the soft palate; therefore increasing negative inspiratory pressure within the larynx. Over time he proposed the larynx would undergo changes in response to this negative pressure stress. These changes would occur first in the place of least resistance – the laryngeal saccules but over time the cuneiform and subsequently corniculate processes would lose their rigidity and collapse medially leading to further obstruction of the rima glottis. He therefore proposed a three tier grading system:

- Grade I – eversion of laryngeal saccules
- Grade II – collapse of the cuneiform processes and the aryepiglottic folds
- Grade III – collapse of the corniculate processes

There is, however, some debate in the profession as to whether saccule eversion really is part of laryngeal collapse. In humans, the function of the saccule is unknown but is thought to be involved in lubrication of the vocal folds. At this time, this author recommends removal of the laryngeal saccules if everted although a recent publication by Hughes et al (2018) reported an apparently increased post-operative complication rate in BOAS patients post surgery. Unilateral arytenoid lateralisation is reported for more severe grades of laryngeal collapse. Partial arytenoidectomy is also reported.

Grading of the severity of clinical signs and thus deciding in which patients to pursue surgical management can be challenging. Various techniques have been reported include direct laryngoscopy for grading of laryngeal collapse, qualitative grading schemes such as those reported by Poncet et al (2005) or whole body plesthysmography (Liu et al 2016).

History should include questions on exercise tolerance in both high and normal ambient temperatures, syncopal episodes, gastrointestinal signs, snoring etc. The author also performs a three minute exercise tolerance test in the clinic setting to assess the degree of stertor seen at exercise first hand. Clinical signs include stertor – predominantly at exercise or excitement, exercise intolerance, syncope, gastrointestinal signs – drooling, regurgitation, gagging, vomiting, reverse sneezing, snoring and sleep apnoea.

Surgical management of BOAS is aimed at reducing resistance to airflow through the upper respiratory tract on inspiration. The procedures required at surgery depending on the findings of your airway assessment on induction of anaesthesia. If the soft palate is considered to be elongated, palatoplasty (also called staphlectomy) is performed. Various techniques are reported including traditional "cut and sew" techniques, CO2 laser resections and use of tissue sealant devices. The author performs a technique called a "folded-flap" palatoplasty which aims to thin the soft palate as well as shorten it (Findji et al 2008).

If laryngeal saccules are everted, they should be removed at the base using a pair of scissors. This is performed to increase the cross sectional area of the rima glottis and can require extubation and then re-intubation. Nares assessment is subjective but the majority of BOAS dogs may benefit from vertical wedge nares resection to reduce resistance to airflow at the point of entry into the nasal cavity. Again various techniques are reported but the author performs a vertical wedge nares resection by removing a 3D wedge of tissue from the alar cartilage as shown and suturing with 5/0 polyglactin 910.

Performing palatoplasty and vertical wedge nares resection (+/- sacculectomy) does not address all the components of BOAS which lead to increased airflow resistance and indeed it should be remember that as with many "syndromes", not all patients are equally affected nor have the same components. Recently, turbinectomy has been described as an additional technique to improve airflow through the nasal cavity. Aberrant conchae occur more commonly in dogs with BOAS as do nasopharyngeal turbinates. Mucosal contact points are reported in 14% of meso or dolicephalic dogs versus 87% of brachycephalic dogs (Schuenemann et al). Hypertrophic nasal conchae reduce the lumen of intranasal passageways and maximum hypertrophy can lead to an increase in mucosal contact points. The smaller the air passages, the higher the intranasal resistance will become which contributes to nasal obstruction. Laser-assisted turbinectomy is reported to be effective at improving clinical signs in a subset of BOAS patients. Turbinate regrowth is common (up to 68% of cases) but this does not necessarily lead to recurrence of mucosal contact points. This is still a relatively new technique.

Anaesthesia for BOAS dogs is complex to say the least. This should not be undertaken unless you have the capability to perform corrective surgery and the facilities for tracheostomy tube placement/management. Intravenous access should ideally be obtained PRIOR to premedication. Our standard practice protocol is to begin omeprazole therapy either prior to or at admission to the hospital as this patient group are pre-disposed to gastro-oesophageal reflux. The author usually uses a combination of low dose ACP (0.005mg/kg-0.01mg/kg) and methadone (0.3mg/kg) for premedication. Patients should be continually monitored following premedication as obstruction can occur as pharyngeal relaxation occurs. Pre-oxygenation should be performed prior to induction (in case of challenging induction).

For induction, ensure all items are prepared and in easy reach prior to administration of the induction anaesthesia. A good light source and suction should be available. Assess palate length with the tongue in a NEUTRAL position and then grade the degree of laryngeal collapse.

Once anaesthetised, it is preferable to use an air/oxygen mix and positive end pressure ventilation to reduce collapse of the alveoli. Anaesthesia duration should be kept to a minimum. Recovery often represents the period of highest risk for these patients. Recovery should be slow and fully observed. Patients should be left intubated for as long as possible and the head should be kept elevated. At our clinic, all BOAS patients are nebulised post extubation with a dilute adrenaline solution. This is usually a one off treatment but can be repeated in patients who exhibit dyspnoea following recovery from anaesthesia. The aim of this treatment is to reduce mucosal oedema both at the level of the larynx but also in the larger airways. It has been the author's experience that recovery is subjectively more complex in patients with significant tracheal hypoplasia and mucosal oedema secondary to intubation can be a significant issue in these patients.

Any patients which exhibit significant mucosal oedema at any point during assessment, surgery or in the post-operative recovery period should receive intravenous dexamethasone (the author usually uses 0.2mg/kg) which can be repeated at 24 hours if required. Although this will theoretically increase the risk of incisional breakdown, this is not something the author has experienced. Intravenous access should be maintained until discharge and each patient is given an individual sedation plan to be used to should any signs of oedema become apparent (usually seen as increased respiratory effort and stridor). Nebulisation with adrenaline is also performed on recovery. Laryngeal/pharyngeal oedema, oesophagitis and gastro-oesophageal reflux leading to aspiration pneumonia are the most significant complications seen following this surgery. These patients are hospitalised on the night following surgery and require close monitoring. In the author's institute, over 95% of patients are discharged on the morning following surgery.

Whilst the majority of BOAS patients present as elective cases, these cases can deteriorate rapidly and emergency presentation is not unusual especially during the summer months. Clinical signs include significant inspiratory dyspnoea, hyperthermia and expiratory dyspnoea due to pulmonary oedema and aspiration pneumonia/pneumonitis. Management is similar to that described for post-operative recovery and includes:

- Provision of oxygen
- Actively cooling if patient is hyperthermic
- Obtain intravenous access
- Consider sedation - ACP 0.005mg/kg-0.01mg/kg dilute IV plus methadone 0.2mg/kg dilute IV
- Intravenous dexamethasone if you suspect upper respiratory tract oedema (0.2mg/kg IV)
- Nebulisation with adrenaline

The two most common causes of lower respiratory tract dyspnoea in these patients are non-cardiogenic pulmonary oedema secondary to acute respiratory distress syndrome and aspiration pneumonitis/pneumonia.

Acute respiratory distress syndrome (ARDS) leads to loss of endothelial integrity. The reason for the loss of vascular and alveolar integrity has not been fully defined.. The pathogenesis of ARDS is extremely complex and involves numerous inflammatory mediators as well as cellular responses. Loss of integrity leads to the uncontrolled flow of fluid and protein into the interstitial space, exceeding the capacity of the lymphatic system. Therefore, fluid begins to accumulate in the pulmonary interstitium and the peribronchovascular cuffs. There is also damage to the alveolar epithelium, and fluid and proteins leak from the interstitium into the alveoli, causing alveolar flooding. The inhalation of irritants such as oropharyngeal or gastric contents into the airways can also cause lung injury and is referred to as aspiration pneumonitis. Pulmonary damage may result from exposure to gastric fluid, aspiration volume, or large particulate matter. Gastric acid-mediated injury is initially an aseptic injury, but eventually reduces airway defence mechanisms, predisposing the lung to secondary infection via bacterial colonization. The term aspiration pneumonia refers to the development of a presumed secondary infection after the initial injury.

Management of such patients is challenging. Oxygen supplementation and gastroprotectants are used in addition to inhaled bronchodilators e.g. salbutamol and nebulisation with hypertonic saline to rehydrate thick secretions and make it easier for them to move up the mucociliary escalator. Simple physiotherapy including walking or encourage the patient to get up and move around or change position helps to encourage movement of secretions. Anti-microbial therapy is indicated but is important to remember that following aspiration, the injury is initially mechanical inflammation rather than infection. Severely affected cases can require mechanical ventilation for a period of days which is costly (around £1000 per day). An 80% survival rate is reported (Tart et al 2010).

In patients with severe laryngeal or pharyngeal oedema either prior to or following surgery can require temporary tracheostomy tube placement. In the author's institution the incidence of tracheostomy tube placement is less than 3% but aggressive post-operative management and monitoring is performed. General anaesthesia is required. The tube diameter selected should fill no more than 75% of the lumen of the trachea so air can move around the tube if it becomes blocked. There is no indication for the use of a cuffed tracheostomy tube unless you intend to ventilate your patient. Stay sutures should be placed around the tracheal rings above and below the location of the tracheal incision to facilitate location of the stoma in an emergency or should tube exchange be required.

As the upper airway is bypassed, placement of a tracheostomy tube results in inspiration of cool, dry air. The tracheobronchial tree responds by increasing mucus production which collects within the tube lumen. The patient should be nebulised through the tracheostomy tube using hypertonic saline for five minutes every four to six hours. Hypertonic saline encourages hydration of secretions which can become dried out and therefore harder for the mucociliary escalator to clear. Some authors advocate exchange of the tube at regular intervals although this author prefers to exchange only when necessary. Tube lumen should be suctioned every four to six hours using a sterile suction catheter or a sterile rigid dog catheter with the tip cut off. Ensure you pre-oxygenate for five minutes prior to suctioning. To exchange the tube, make sure you have all the kit ready BEFORE you start including propofol (brachycephalic necks can be challenging!) in case you need to induce. Wearing sterile gloves, pull the stay sutures apart and replace the tube, securing with umbilical tape.

Remember patients requiring a tracheostomy tube can die peracutely if the tube becomes obstructed and therefore require constant monitoring. Following removal, the stoma is left open to granulate rather than closed surgically to prevent the development of subcutaneous emphysema.

Laryngeal Paralysis:

The larynx is formed from five cartilages; epiglottis, paired arytenoids, cricoid and thyroid cartilages and innervated by the cranial and caudal laryngeal nerves which originate from the vagus. The majority of the musculature works to close the rima glottis. The main muscle responsible for abduction of the arytenoid cartilages is the cricoarytenoideus dorsalis. The larynx has three main functions:

1) During swallowing, the larynx and the hyoid apparatus are pulled cranially, bringing the rima glottis under the epiglottis to block the laryngeal opening

2) Decrease airway resistance by abduction of the arytenoid cartilages

3) Voice production by changing the tension on the vocal cords.

Both congenital and acquired forms are described. In the congenital form, progressive degeneration of the neurons within the nucleus ambiguous leads to subsequent Wallerian degeneration of the laryngeal nerves. Recognised breeds include Bouvier des Flandres, EBT, Huskies, Dalmatians, Rottweilers and the Pyrenean Mountain dog. Clinical signs are usually seen before one year of age. In the acquired form several aetiologies exist including geriatric onset laryngeal polyneuropathy (most common), polyneuropathy secondary to hypothyroidism, iatrogenic damage or trauma to the laryngeal nerves, cervical or mediastinal mass affecting the recurrent laryngeal nerves and polyradiculoneuritis. The median age of presenting dogs is nine years.

Clinical signs can include progressive inspiratory stridor, progressive exercise and/or heat intolerance, cough or gag associated with eating or drinking, changes in phonation, cyanosis or syncope and polyneuropathy signs. Patients can also present as an emergency with clinical signs such as significant inspiratory stridor and dyspnoea secondary to laryngeal oedema and paradoxical medial arytenoid movement on inspiration, hyperthermia and expiratory dyspnoea due to non-cardiogenic pulmonary oedema and aspiration pneumonia/ pneumonitis. Management is similar to that previously described.

Diagnosis of laryngeal paralysis is made by direct laryngoscopy. A high clinical suspicion for laryngeal paralysis can usually be made on the basis of signalment, history and clinical signs. Anaesthesia CAN OFTEN destabilise these patients. DO NOT INDUCE IF YOU ARE NOT PLANNING TO OPERATE. There is much debate over the “best” premedication and anaesthesia protocol. The author’s preference is for a low dose ACP and methadone premedication and a very slow propofol induction. An assistant should call out inspiration and expiration so you can determine “paradoxical” arytenoid movement which can confuse diagnosis (i.e. arytenoids appearing to move as they are sucked together on inspiration rather than abducting).

Thoracic radiographs (to look for signs of aspiration pneumonia or a cranial mediastinal mass) +/- an extubated cervical lateral projection should be performed under anaesthesia. Surgery aims to widen the rima glottis to reduce resistance to airflow on inspiration.

Unilateral arytenoid lateralisation is currently the treatment of choice in the UK. Sutures are placed between the muscular process of the arytenoid cartilage and the caudal aspect of the cricoid cartilage via a lateral approach to the larynx to abduct the arytenoid cartilage laterally thus increasing the diameter of the rima glottis. Other treatments e.g. trans-oral partial laryngectomy are also reported.

Prognosis for these patients is generally good following surgery depending on the underlying aetiology. Approximately 90% of patients improve after surgery. Bookbinder et al (2016) reported that dogs undergoing surgical correction of their laryngeal paralysis had a 2.6-fold reduction in the hazard of death. Approximately half of that cohort had or developed concurrent polyneuropathy signs at follow up. Wilson et al (2016) reported aspiration pneumonia occurred in 18.6%, 31.8%, and 31.8% of dogs at the 1-, 3-, and 4-year follow-up periods,

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