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Emergency and Critical Care Nursing Mini Series

Session 3: Emergency Surgical Nursing

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Introduction

Gastric dilatation and volvulus (GDV), commonly known as bloat, is an acute life threatening condition, which is characterized by the malposition of the stomach when it rapidly fills with air and rotates. Gastric dilatation (the expanding of the stomach) and gastric volvulus (the twisting of the stomach without expansion) can occur separately, however when they occur simultaneously the disease process to the body results in death if left untreated. Despite the first report of GDV in 1906, researchers have been unsuccessful in identifying a cause. Mortality rates range from 15–33%. While GDV can occur in many species (including cats and primates), deep chested and giant breed dogs are most commonly affected. Prompt recognition, rapid treatment and surgery are required to increase the chance of survival.

Risk Factor

Approximately 22% of giant breed dogs and 24% of large breed dogs will develop GDV in their lifetime.

The Great Dane has the highest incidence (42.4%). The other most common breeds affected breeds are the Weimaraner, Saint Bernard, Gordon setter, Irish setter and the standard poodle. As these breeds get older, the risk of developing GDV also increases.

Some behaviour studies have suggested that fearful or anxious dogs may have an increased risk to developing GDV. It is speculated that the gastrointestinal tract may be adversely affected during times of stress, especially in the fearful dogs, which may lead to an increase risk to developing GDV.

Though there is no exact cause for GDV, many proposed risk factors could contribute. Exercise after a large meal, especially a meal of highly processed food or water, may be a risk factor. Dogs that were fed only one type of food appear to have an increase in risk, while dogs that were fed table scraps or canned food appear to have a lower risk. Despite the myriad of reports suggesting that it is food related, there has not been one definitive report showing a link between GDV and the types of food. Dogs that are male, geriatric, eating only one meal a day, eating too quickly or having a raised food dish may all increase the risk to developing GDV. One published report showed that large breed dogs that ate quickly out of a raised food dish had a 20% increase in risk and giant breeds had an increase of 50%.

Dogs that have developed GDV have been found to have increased gastrin concentrations. Gastrin is a hormone produced in the stomach that increases the release of gastric juice. In digestion, gastrin constricts the pyloric sphincter, causes oesophageal spasming and slows the rate of gastric emptying. This can lead to aerophagia and decreases the chance of vomiting with gastric dilation.

Other risk factors include a decrease in oesophageal sphincter tone, myoelectric dysfunction and dogs that experience a delay in gastric emptying.

Pathophysiology

Most commonly the stomach rotates 90–270 degrees in a clockwise motion (viewed from behind). The fundus will generally shift to lie against the ventral abdominal wall while the pylorus will move along the ventral abdominal floor, eventually sitting dorsally and on the left side. This will cause the body of the stomach to shift right. Because the spleen is attached to the stomach by the gastrosplenic ligaments and vessels, the spleen will also follow the stomach and will become displaced, if not twisted as well.

Both air and fluid become entrapped within the stomach causing it to become enlarged. The gas may have become trapped because of aerophagia, but it may also be formed from bacterial formation. As the stomach fills with air, the caudal vena cava becomes compressed, leading to a decrease in venous return from the heart. A distended stomach can cause up to a 75% decrease in arterial flow to the gastric mucosa. Depending on the degree of rotation of the stomach, a partial or complete blockage of the portal vein may also occur. This may cause the liver or pancreas to become ischemic to some degree. With the rotation of the spleen it too may become ischemic**.**

As mentioned typically the patient presenting with a GDV will be a large breed, deep chested dog; however, it is important not to make this your only guideline. GDV has been documented in smaller deep chested breeds such as Dachshunds or dogs prone to over-eating such as Spaniels and Labradors. It has also been seen in puppies that have over-eaten. It can also occur in cats and other species though it is less common.

Pre-Surgical Treatment

Patients presenting with GDV usually have unmistakable signs. Signs include non-productive retching, abdominal distension, abdominal pain, anorexia and restlessness. The stomach may be tympanic. Most patients will present in shock.

Treatment of the shock is the first priority. Because the patient is in shock and usually has severe abdominal pain, intravenous (IV) catheter placement, IV fluid therapy and pain medication should be started before diagnostics. Two large-bore short peripheral catheters should be placed to maximize fluid resuscitation efforts. Short, large diameter catheters allow for higher fluid flow. Oxygen should be administered (generally given by flow-by) because many GDV patients are in respiratory distress due to the enlarged size of the stomach pressing on the diaphragm. All vitals and parameters should be monitored including pulse, respiration rate, blood pressure, mucous membrane colour and ECG. Many GDV patients experience ventricular arrhythmias.

Fluid resuscitation is performed in stages to end-point parameters of improved perfusion, normal heart rate and normal blood pressure. Isotonic replacement crystalloids (e.g. Hartmann's) are always administered with incremental doses. Synthetic colloids (hetastarch) or HBOC (Oxyglobin®) are administered at incremental doses of 5-10 ml/kg (up to 20 ml/kg with synthetic colloids and 30 ml/kg

with Oxyglobin®). These solutions promote colloid osmotic pressure during fluid resuscitation. In addition, HBOC carry oxygen to tissues with compromised blood flow and have a mild vasoconstricting effect which may be desired during SIRS states. If crystalloids are used alone, the bolus doses are increased to 20-30 ml/kg.

The rapidly deteriorating hypovolemic patient without significant haemorrhage can benefit from the infusion of hypertonic saline (4 ml/kg 7% solution) with synthetic colloid added in an effort to augment exogenous fluid infusion with interstitial fluid redistribution. As these patients are frequently large dogs, the use of hypertonic saline may enable rapid improvement of cardiovascular signs compared to isotonic crystalloids.

Analgesia should be administered immediately because it will, not only help alleviate the acute abdominal pain, but also it will help to reduce the stress for the patient.

Radiographs are not to be taken until fluids and temporary decompression has been initiated, unless euthanasia is an option over surgery. Radiographs should not delay surgical preparation. Abdominal x-rays are not performed routinely in our institution. Indication to go to surgery is given if orogastric decompression is not possible. However, if oral decompression is possible, a right lateral abdominal radiograph will show if a volvulus is present and will also indicate surgical treatment.

Laboratory database

Obtaining pre-fluid blood samples for PCV/TS, electrolytes, venous gas, azostick and glucose, platelet count, activated clotting time, and saving samples for coagulation profile, serum chemistries, complete blood count and urinalysis are recommended.

Pre-fluid values provide a baseline from which subsequent values are compared to and monitored. In addition, any significant abnormalities are addressed prior to surgical intervention.

Any clinical evidence of coagulation abnormalities in addition to laboratory abnormalities requires appropriate treatment prior to surgery; frozen plasma if DIC or coagulation factor defect is suspected.

Initial bloodwork should be obtained prior to starting IV fluids (obtain blood at the same time catheters are placed) to ensure that values are those of initial presentation. With any patient suspected of ischemic disease, a blood lactate level should be obtained. Lactate acid build-up occurs when the body is unable to perform aerobic metabolism. During the anerobic process, lactate will start to build up indicating a worsening of illness. During GDV, blood supply to the stomach is decreased if not completely stopped (an ischemic event). This causes the body to switch to an anerobic process, thus causing a build-up of lactate. Blood lactate values under 2mmol/L are normal. In the case of gastric dilatation volvulus, levels greater than 6mmol/L are associated with increased gastric necrosis.
In 1999 one study of 102 dogs with GDV found that only 58% of dogs survived with a blood lactate greater than 6mmol/L, while 99% of dogs survived with levels less than 6mmol/L. However a retrospective study showed this should not be a reason for euthanasia. The results of a second retrospective study in 2011 indicated that an initial presenting plasma lactate concentration >6.0

mmol/L is not predictive of macroscopic gastric wall necrosis or survival in dogs presenting with GDV. A decrease in plasma lactate concentrations >50% within 12 hours may be a good indicator for survival. Other bloodwork that should be run includes a complete blood count, packed cell volume, total protein, coagulation profile, serum chemistry profile and electrolytes. Though it seems excessive, it is important to know whether any pre-existing disease exists and how decompensated the patient is. Since all GDV patients require surgery it is equally important to make sure that all electrolyte or other blood work abnormalities be rectified before the animal is placed under general anaesthesia. Since disseminated intravascular coagulopathy (DIC) can occur whenever there is major change in the vascular system, all GDV patients are considered at risk, which is why it is important to check a coagulation profile.

Decompression

Once GDV has been diagnosed and therapy for shock has been initiated, gastric decompression should be performed. Gastric decompression helps to improve cardiac output and blood pressure by alleviating pressure on the vena cava and portal vein. There are two methods that can be used to decompress the stomach: orogastric and gastrocentesis. Placement of an orogastric tube, which allows for the most decompression of the stomach. This method, however, generally causes more stress to the patient, and some patients may not tolerate it at all.

A tube should be measured and marked from the point of the nose to the last rib. A roll of tape should be placed in the mouth just behind the incisors with someone shutting the animal's mouth on the roll. The tube is then advanced with slightly firm pressure and a twisting motion. Be careful not to apply too much force on the tube because you could cause an oesophageal or gastric tear. Once you are in the stomach, contents should flow out of the tube into a bucket below the patient. If an orogastric tube cannot be placed, then a gastrocentesis should be performed, or gastrocentesis may be performed initially since it is often quicker, to remove some of the gas from the stomach. Since GDV patients are prone to DIC, it is important to check coagulation factors prior to starting this procedure. A gastrocentesis is done by inserting a 14 to 18 gauge needle or over-the-needle catheter into the stomach. The patient should be lying in left lateral recumbency to allow for the gastrocentesis to take place on the right side. The area should be clipped and prepped. Once the gastrocentesis has been performed and air has been removed, orogastric decompression can be attempted again to remove more of the air and contents.

Prior to surgery, broad-spectrum antibiotics are usually given because of the risk of gastric necrosis and perforation.

Lidocaine treatment

Lidocaine is used in our institution for various effects in a GDV patient. Lidocaine is an effective pain relieving substance, it is a viable option for treatment of ischemic arrhythmias and it is potentially helpful in the prevention of reperfusion injuries.

Blood pressure and ECG

Hypotension is usually present in the critical GDV patient. If significant haemorrhage and/or DIC are present, hypovolemic resuscitation may be warranted until exploration. This entails careful endpoint resuscitation techniques using crystalloids and colloids/HBOC to a MAP of approximately 80 mmHg (systolic around 100 mmHg). The goal is to initiate some reperfusion without disturbing any clots that have formed until haemostasis is achieved surgically. A constant rate infusion (CRI) of hetastarch can be administered after resuscitation of the hypotensive animal at a rate of 0.8 ml/kg/hr to help maintain blood pressure until cardiovascularly stable.

Normal to increased blood pressures is assessed with respect to intravascular volume status. Adequate or increased blood pressure may be the result of a compensatory response to hypovolaemia, and aggressive fluid resuscitation is still indicated.

Any auscultable or ECG arrhythmia should be treated with oxygen therapy. Any acid-base and electrolyte (potassium, calcium and magnesium) abnormalities should be corrected. When improvement of perfusion does not occur, antiarrhythmics are administered. The most common arrhythmia treated is a ventricular tachycardia, and lidocaine is administered IV up to 4 mg/kg slow bolus. If this improves the rhythm, then a 50 mcg/kg/min CRI is started. Constant ECG is required.

If the blood pressure is not responsive to fluid resuscitation, dobutamine infusion (5-10 mcg/kg/min) may be required and underlying causes of nonresponsive shock investigated. Dopamine infusion (5-15 mcg/kg/min) may also be necessary. Clinical experience has shown, that animals in need of catecholamines to maintain blood pressure have decreased survival rates compared to animals that do not need catecholamines.

Oxygenation

As a result of circulatory compromise these patients may present hypoxic and also due to low blood pressure/poor perfusion have reduced tissue oxygen delivery, therefore increasing the fraction of inspired oxygen (FiO2) will be beneficial.

Flow-by techniques are often used during the initial stages but this, particularly for larger patients, is usually only a short-term option. A useful technique is the placement of nasal oxygen catheters, which can be utilised both pre- and post-surgery.
Humidification of the oxygen is important if using nasal oxygen catheters for a period of longer than 3-4hrs since this technique will bypass a portion of the patient's upper airway. GDV patients will frequently have compromised ventilation due to the over-distension of the stomach. Sternal recumbency offers the best opportunity for a patient to ventilate itself properly BUT these patients are normally extremely uncomfortable and should be allowed to assume the position most comfortable for them.

Intraoperative/Anaesthesia considerations

Pre-anaesthetic protocols vary and the individual patient should be assessed to determine what is appropriate. Phenothiazine tranquilizers (acepromazine) usually avoided as they can cause vasodilation and hypotension. Ideally, a benzodiazepine (diazepam) should be included in the pre-anaesthetic protocol because it serves as a muscle relaxant and can help reduce anxiety by slowing down the central nervous system. Other pre-anaesthetic drugs include: fentanyl and alfaxan or propofol. Propofol can cause vasodilation and respiratory distress and should be limited to those animals that have been stabilized. Once intubated, the patient should be monitored very closely, which should include ECG, blood pressure, pulse oximetry and capnography. Mechanical ventilation may be required in order to maintain adequate oxygenation.

It is imperative that intraoperative blood pressure be maintained at a mean arterial pressure of 80 mmHg. 'Surgical rate' fluids generally include using a crystalloid at 5-10ml/kg/hr. Using a CRI of analgesics may allow for a decrease in inhalant anaesthesia and help increase blood pressure. However, because some medications can cause respiratory depression it is important to have mechanical or assisted ventilation ready if needed. If the patient is hypovolemic despite efforts (be sure that the patient is on an appropriate plane of anaesthesia), vasoactive medications should be started. Commonly dobutamine (5–20mcg/kg/min) and/or dopamine (5–10mcg/kg/min) are administered as a CRI to help improve cardiac contractility. If inotropic support alone does not improve blood pressure, then a pressor agent such as norepinephrine (at 0.05–0.4 mcg/kg/min) can be used.

Post Operative considerations

Continuous or intermittent monitoring of the vital signs will detect development of hypotension and/or dysrhythmias that may require immediate therapy. Monitoring PCV/TS, glucose, BUN, albumin, electrolytes, acid/base status, and lactate levels may uncover organ decompensation. Intravenous analgesia and antibiotic administration is continued until oral feedings and medications are tolerated. The use of promotility agents such as metoclopramide and cisapride may improve gastric emptying more rapidly than without. Supplemental oxygen therapy is also recommended in the post-operative period.

Maintaining gastric decompression post operatively is recommended in the critical GDV patient. Gastrostomy tube placement allows large volume decompression and removal of large clots that can occur with large resections. Nasogastric tubes are appropriate when gastric resection is not required. Nasogastric tubes are preferably placed intraoperatively with proper placement assured by palpation. Small volume infusion of electrolyte/glucose/glycine containing fluids feeds the gastric mucosal cells, which rely on intraluminal contents for nutrition. Placed appropriately, intestinal feeding tubes provide immediate intestinal feeding postoperatively. It allows home care if gastric feeding is not possible once the animal is ready to be discharged. It also reduces the cost of parenteral nutrition because caloric requirements can usually be supplied within a few days.

Monitoring nasogastric tube suction volumes assists in more accurately determining volumes lost. When suction volumes decrease, this may indicate when refeeding may be initiated. Infusion of a balanced electrolyte/carbohydrate solution promotes gastric mucosal healing and feeding. Regular assessment of the patients abdomen size is also recommended to assess for rebloating.

GDV patients can be particularly challenging and require intensive nursing care but it is these aspects of their care which make a successful outcome so rewarding.

The GDV patient is one of the most challenging but perhaps the most rewarding patient to Nurse. By close observation and monitoring, good communication with the Veterinary Surgeon and taking the time to give TLC, all of the things that make us veterinary nurses we can have a really positive impact on the outcome of these cases.

HAEMOABDOMEN

Haemoabdomen (or haemoperitoneum) is defined as the presence of free blood within the peritoneal space. The true incidence of haemoabdomen is probably underestimated, however, it remains a common finding in small animal emergency practice. The degree of severity of intra-abdominal haemorrhage can vary widely, requiring a dynamic approach and careful consideration of the individual patient and their clinical picture.

In contrast to human medicine, where a large number of interventional trials have been designed to determine the best approach to these patients, few veterinary studies have been published: there remains, therefore, a degree of controversy regarding the ideal treatment of clinical veterinary patients with haemoabdomen.

Aetiology

Causes of haemoabdomen can essentially be reduced to 2 categories: traumatic and non-traumatic. Blunt force trauma due to road traffic collisions is probably responsible for the majority of traumatic haemoabdomens, although penetrating injury can often be a frequent finding. Non-traumatic causes of haemoabdomen are most frequently due to rupture of intra-abdominal neoplasms, although vascular trauma due to ischaemia or traction, as well as systemic coagulopathies are not uncommon aetiologies.

Triage and Primary Survey

Whilst a succinct ‘capsule’ history is being obtained from the owner, an initial primary triage survey should establish whether the animal is in imminent danger of failure of one of the major body systems (respiratory, cardiovascular and neurological): if so, the animal should immediately be conveyed to the treatment area and resuscitative efforts begun. Following the primary survey, a more thorough secondary survey of the major body systems (together with abdominal palpation and determination of rectal temperature) should allow a more comprehensive assessment of the state of cardiopulmonary compromise of the animal.

Major body systems assessment: the cardiovascular system

Careful evaluation of the cardiovascular system as part of the secondary survey should allow the degree of hypoperfusion to be determined. This is probably most easily categorised as compensatory or decompensatory.

Patients with mild hypoperfusion will still be compensating for the intra-abdominal haemorrhage by peripheral vasoconstriction (resulting in pale/normal mucous membranes with rapid capillary refill {CRT}) and increased cardiac rate and contractility (resulting in hyperdynamic pulses). Respiratory rate may be increased in these animals and they are generally alert. The intra-abdominal haemorrhage in these patients is likely to be low volume or chronic in nature, although the early presentation of an animal with a major bleed should not be discounted at this stage.

Patients in a moderate-severe hypoperfusion state are likely to represent animals with a greater volume of intra-abdominal haemorrhage, either acutely or chronically. Such patients will present with pale mucous membranes, prolonged CRT, progressive loss of palpable peripheral pulses with weakened femoral pulses, greater tachycardia and reduced mentation. As the volume of blood loss progresses, femoral pulses weaken and peripheral pulses are lost, mucous membranes become white with no discernible CRT and the patient becomes stuporous; timely intervention is required at this point to avoid sudden death. Re-establishment of an effective circulating volume as soon as possible after signs of decompensation appear is likely to meet with a more favourable outcome.

Fluid resuscitation in the haemoabdomen patient

Experience from both experimental models of haemorrhagic shock and from human clinical trials has questioned the appropriateness of the ‘traditional’ use of large, untitrated volumes of crystalloid fluids. Instead, 2 techniques for ‘low volume resuscitation’ have become widespread in recent years: the first involves withholding any intravenous fluid therapy until at a trauma centre with the option of immediate transfer to theatre and as such, is probably not appropriate for most clinical situations in small animals. The second technique aims to titrate intravenous fluid therapy to a level where circulatory support of vital organs such as the brain and kidneys should be maintained. In practice, this involves administration of small volume boluses of intravenous fluids with the target of maintaining mean arterial pressure (MAP) in the 60-70mmHg range (or systolic arterial pressure in the 90-100mmHg range).

Seemingly inexhaustible levels of debate and investigation have been dedicated to attempting to determine the most appropriate resuscitation fluid to use: whilst most human studies compare albumin (as a colloid), rather than a hydroxyethyl starch, to a balanced isotonic crystalloid, the overriding conclusion from these studies is that fluid choice is probably of little relevance in the acute phase. Recently, aggressive use of high levels of blood products has been employed in human military settings, although the application of point-of-care coagulation testing has allowed a more measured approach to good effect.

Diagnostic approach to the haemoabdomen patient

Upon presentation, procurement of a ‘minimum database’ (classically consisting of PCV, total solids, glucose and urea) is often a good approach in any patient presenting with a major body system disorder. Extension of this to include serum lactate, central venous blood gas and examination of a blood smear would also be useful, if possible. Obtaining samples for complete blood count and serum biochemistry prior to any treatment could also be argued, but the utility of any such results in furthering diagnosis, particularly in an emergency setting, should be considered carefully.

Testing for coagulopathies is indicated for most patients presenting with haemoabdomen.

Whilst historical information and certain physical examination findings can raise suspicions for haemoabdomen, diagnostic imaging is often indicated for animals presenting with acute signs of hypoperfusion. In general, these should be delayed until the patient is stable, although certain procedures may be possible during the resuscitation process without compromising patient safety.

Thoracic radiographs may well be required later if there are concerns regarding metastatic neoplasia, and plain/positive contrast studies of the urinary tract may also be indicated, especially with a history of blunt abdominal trauma. However, the rapid assessment of the abdomen with diagnostic ultrasound is undoubtedly the modality of choice, if available. The need for rapid ultrasonographic assessment of the abdomen without recourse to a specialist team led in human medicine to the development of the Focused Abdominal Sonography for Trauma (FAST) protocol; this technique has also been described in dogs and found to have 96% sensitivity and 100% specificity for the detection of free abdominal fluid (but not specifically for haemoabdomen) following road traffic injury.

Acquisition of non-clotting whole blood via abdominocentesis is pathognomonic for haemoabdomen. Serial abdominocentesis demonstrating changes in the PCV of the free abdominal fluid can raise suspicions of on-going haemorrhage and the need for surgical intervention.

Control of intra-abdominal haemorrhage

If clinical findings (such as changing PCV of the abdominal fluid, abdominal fluid chemistry suspicious for visceral rupture, inability to stabilise cardiovascular parameters) indicate likely on-going haemorrhage, then further therapeutic intervention is required.

In the presence of coagulopathy, judicious use of blood products such as fresh frozen plasma, packed red blood cells or whole blood may be indicated, as well as Vitamin K if hepatic failure or anti-coagulant rodenticide toxicosis are suspected. Abdominal counterpressure bandaging may be used to reduce intra-abdominal blood flow and provide a tamponading effect: this technique does have several contra-indications and complications associated, however, and no clinical studies evaluating its use have been published in veterinary patients.

Animals that fail to stabilise their cardiovascular parameters after these measures have been taken are candidates for emergency surgical intervention. It should be remembered, however, that these are physiologically fragile patients and a considered approach to their anaesthesia is obligatory, as is prior patient and surgeon preparation.

Diaphragmatic Rupture

Rupture of the diaphragm is most commonly seen with blunt thoracic trauma, with 77-85% of all cases of diaphragmatic rupture being traumatic, congenital pleuroperitoneal diaphragmatic ruptures are very occasionally seen. Affected animals are presented with different clinical signs starting from no respiratory distress to catastrophic life endangering dyspnoea depending on the amount of herniated abdominal organ material.

The abrupt increase in intra-abdominal pressure accompanying forceful blows to the abdominal wall causes the lungs to rapidly deflate (if the glottis is open), producing a large pleuroperitoneal pressure gradient. Alternately, the pressure gradient that occurs between the thorax and the abdomen may cause the diaphragm to tear.

The tears occur at the weakest points of the diaphragm, generally the muscular portions. Location and size of the tear or tears depend on the position of the animal at the time of impact and the location of the viscera.

Traumatic diaphragmatic hernias are often associated with significant respiratory distress; however, chronic diaphragmatic hernias in asymptomatic animals are not uncommon.

Animals with recent traumatic diaphragmatic hernias frequently are in shock when they present for treatment; therefore, clinical signs may include pale or cyanotic mucous membranes, tachypnea, tachycardia, and/or oliguria. Cardiac arrhythmias are common and associated with significant morbidity.

Other clinical signs depend on which organs have herniated and may be attributed to the gastrointestinal, respiratory, or cardiovascular system. The liver is the most commonly herniated organ, a condition that often is associated with hydrothorax caused by entrapment and venous occlusion.

Causes of Respiratory Compromise

- Loss of functional residual capacity (mass effect from herniated organs and or pleural effusion / pneumothorax)

- Pulmonary contusions
- Atelectasis of the lung lobes
- Rib fractures
- Flail chest
- The effects of shock
- The effects of pain

Myocardial contusion often present and may decrease cardiac output. When myocardial injury is concomitant with impaired ventilation, tissue hypoxia can result. Pain resulting from chest and abdominal contusion and accompanying injuries causes voluntary restriction of thoracic excursion and can therefore further compromise ventilatory capability.

Diagnosis

Definitive diagnosis of pleuroperitoneal diaphragmatic hernia usually is made by radiography or ultrasonography. If significant pleural effusion is present, thoracocentesis may be necessary before diagnostic diagnostic radiographs are performed. Radiographic signs of diaphragmatic hernia may include loss of the diaphragmatic line, loss of the cardiac silhouette, dorsal or lateral displacement of lung fields, presence of gas or a barium-filled stomach or intestines in the thoracic cavity, pleural effusion, and/or failure to observe the stomach or liver in the abdomen. It may be difficult to diagnose diaphragmatic hernias radiographically if only a small portion of the liver is herniated. Ultrasound examination of the diaphragmatic silhouette may help when herniation is not obvious radiographically (i.e., hepatic herniation, pleural effusion). Ultrasonography may be particularly difficult if severe pulmonary contusions are present which make the lung appear ultrasonographically similar to liver, if only omentum is herniated, or if adhesions between the liver and lung are present.
Also, care should be taken not to mistake a normal mirror-image artifact (usually seen as apparent liver parenchyma on the thoracic side of the diaphragmatic line) for herniated liver.

Positive contrast coeliography occasionally may be helpful. Pre-warmed water-soluble iodinated contrast agent is injected into the peritoneal cavity at a dosage of 1.1 ml/kg (the dose is doubled if ascites is present), the patient is gently rolled from side to side or the pelvis is elevated, and films are taken immediately after the injection and manipulation. Criteria used in evaluating these images should include the presence of contrast medium in the pleural cavity, absence of a normal liver lobe outline in the abdomen, and incomplete visualization of the abdominal surface of the diaphragm.

Positive-contrast celiograms should be interpreted cautiously, because omental and fibrous adhesions may seal the defect, resulting in false negative studies.

Pre-operative considerations

Oxygen supplementation
If the patient is dyspnoeic, oxygen should be provided by face mask, nasal insufflation, or an oxygen cage/incubator as all cases of diaphragmatic rupture are likely to have a VQ mismatch, a minimum FiO2 of 50% should ideally be administered. Oxygen supplementation must not induce undue stress

that can result in a deterioration of the animal's condition. Positioning the animal in sternal recumbency with the forelimbs elevated may help ventilation. If moderate or severe pleural effusion is present, thoracocentesis should be performed.

Cyanosis is a late sign of the need for oxygen, and any signs suggestive of hypoxia should be treated promptly to prevent this happening i.e., nasal flaring, dyspnoea, reduced mentation and signs of oxygen hunger such as abducted elbows, extended head and neck, and open-mouthed breathing. Patients that fail to respond to oxygen supplementation may have severe ventilation perfusion mismatching as a consequence of atelectasis or pulmonary contusions.

Fluid therapy

Adequate volume replacement is essential. However, vascular support must be delivered with the knowledge that these patients often have concurrent pathology such as atelectasis and pulmonary contusions that can be exacerbated by over aggressive fluid administration.

Antibiosis

Prophylactic antibiotics should be given before induction of anaesthesia in animals with devitalized tissue, e.g. due to hepatic herniation or significant lung atelectasis. Massive release of toxins into the circulation may occur with hepatic strangulation or vascular compromise.

Pre-anaesthetic considerations

Supplementing oxygen before induction improves myocardial oxygenation. Because of the animal's already compromised ventilation, drugs with minimal respiratory depressant effects should be used. Injectable anaesthetics allowing rapid intubation are preferred. Inhalation anaesthetics should be used for maintenance of anaesthesia.

Timing of surgery

Surgery is best performed after a period of patient stabilisation. Approximately 15% of patients will die prior to surgery. If surgery is performed within the first 24 hours of presentation, mortality rates are highest (33%) reflecting acute cardiorespiratory deterioration in these unstable, shocked and compromised patients. In general, patients are best managed by a period of stabilisation (24-72 hrs) to improve their respiratory function and tissue oxygenation, to correct fluid deficits and to diagnose and manage other potentially life-threatening complications such as cardiac arrhythmias. However, surgery should be performed as soon as the patient is stable and should not be delayed if a patient is deteriorating despite supportive care. However, some cases cannot be left for a period of stabilisation and will require immediate surgical intervention because of the risk of acute decompensation. These include:

- Diaphragmatic hernia with intrathoracic gastric dilatation or GDV: if the stomach or proximal small intestine are herniated, the risk of pyloric outflow and oesophageal cardial obstruction are high and cases may present with a tension gastrothorax as a result of GD or worse, GDV within the thoracic cavity. As normal a stomach tube may be passed in an attempt to relieve the pressure within the stomach;

- Rupture of the gastrointestinal tract;
- Rupture of the biliary tract;
- Ongoing life threatening intrabdominal or intrathoracic haemorrhage;
- Tension pneumothorax secondary to lung damage.

Intra-operative considerations

Intermittent positive pressure ventilation should be performed, and high inspiratory pressures should be avoided to help prevent re-expansion pulmonary oedema. The lungs should be allowed to expand slowly after surgery.

Surgical approach

A ventral midline abdominal approach is used most commonly. The incision should extend from the xiphoid to a point no further cranial than the umbilicus. In rare cases a caudal median sternotomy or paracostal incision may be required to allow management of intrathoracic pathology.

Goals of surgery

- Identify the position of the hernia;
- Reduce hernia contents;
- Assess abdominal viscera for viability;
- Assess thoracic viscera for injury;
- Repair diaphragmatic defect: tension free repair of viable tissue;
- Remove air and fluid from the thorax.

Re-establish negative intrathoracic pressure

There are a number of methods that can be used to re-establish negative intrathoracic pressure:

- Thoracostomy tube placement
- Transdiaphragmatic thoracocentesis
- Transthoracic needle thoracocentesis.

Thoracic drain placement

Thoracic drains are required if there is a risk of ongoing air or fluid accumulationif there is concern over the integrity or viability of the thoracic organs or if there was a moderate or large pleural effusion at surgery. It is far better to place a thoracic drain at this stage and not use it than to have to place one postoperatively. Tubes ideally should be placed under direct visualisation prior to closing the diaphragmatic defect.

Lung overinflation

The traditional technique of overinflating the lungs prior to final suture placement in order to reinflate atelectatic areas of lung and to evacuate air from the thorax is contraindicated and most probably contributed to the high mortality rates in the early reports of diaphragmatic hernia management. This practice can lead to pulmonary re-expansion injury leading to acute alveolar flooding. In this syndrome, increased permeability of the alveolar membrane leads to rapid pooling of fluid in the alveolar space and respiratory collapse. This is seen within a few hours of re-expansion and is usually progressive and fatal. The aetiology is uncertain and could relate to membrane injury secondary to endotoxaemia, hypoxia or reperfusion injury but what is clear is that its development is directly linked to rapid re-inflation and over-inflation of lung. It is far safer to slowly re-establish negative pressure in the thorax and to allow atelectatic areas of lung to re-inflate over time.

Recovery and complications

Patients should be monitored postoperatively for hypoventilation, and oxygen should be provided if necessary.

Most cases that survive surgery but die do so in the immediate postoperative period as the result of acute respiratory collapse. This may be secondary to re-expansion pulmonary injuries or pneumothorax due to previously undiagnosed lung injuries that become apparent as the lungs re-expand or due to thoracostomy tube complications. Patients need to be carefully monitored postoperatively to ensure that their respiratory status is not deteriorating and should continue to receive oxygen supplementation well into the recovery period.

If there is any deterioration, diagnostic thoracocentesis and radiography early in the course of the problem to identify the cause is the safest option. Animals with empty abdomen syndrome may show signs of respiratory distress as a result of raised intra-abdominal pressure. Ventricular arrhythmias are also common. Less frequently, complications associated with the organs that have herniated are encountered. Gastrointestinal tract perforation and haemorrhage from splenectomy and partial hepatectomy are potential complications.Post-operatively pancreatitis is another potential post-operative complication as the pancreas may have been traumatised either at the time of the original injury or subsequently during reduction of the hernial contents.

Emergency Wound Management

Introduction
Most of the wounds we see in our patients are as a result of trauma. Animals that have experienced trauma can have a wide variety of injuries affecting various body systems. When faced with a wounded patient in an emergency situation, it is important to appreciate that the same trauma that caused the obvious wound may also have caused unseen life-threatening internal injuries. While it is all too easy to be distracted by an impressive wound, we need to concentrate on the body as a whole and focus on detecting issues with the major body systems (MBS) initially.

Telephone Triage
When obtaining information on the telephone, it may be necessary to calm the owner prior to trying to obtain concise accurate information. The owner's perception of the problem should be interpreted with caution. If in any doubt about the need for the animal to be seen, it is safest to advise the owner to attend.

Advice may need to be given on transportation of the animal, following trauma: if an animal is unable to walk it may need to be carried, it is preferable to a trauma victim to be carried on a board or something rigid rather than a blanket etc. In the case of active bleeding, direct pressure onto a clean cloth is probably safer than tourniquets. Where penetrating injuries have occurred, it is safest to leave the object in-situ. Always warn the owner that the animal may be aggressive due to pain.

Clinic Triage and Assessment
Patients require rapid and accurate triage and initial stabilisation, followed by ongoing clinical monitoring. Preliminary examination should focus on the respiratory, cardiovascular and central nervous systems; these systems take initial priority, as dysfunction of one of them is most likely to be the cause of death in a trauma patient. Examination and assessment usually follows an "ABCD" protocol; airway, breathing, circulation and disability. Where a problem is detected with the MBS, immediate stabilisation measures are taken, prior to completing the rest of the examination. Initial stabilisation addresses concerns of oxygenation and tissue perfusion.

Examination of MBS at this stage relies on a quick focussed assessment of clinical parameters that give us the maximum of information. Airway and breathing are assessed by observation, auscultation and palpation. Circulation is assessed by heart rate, pulse quality, mucous membrane colour, capillary refill time, and the presence of bleeding. The most common circulatory issue present in trauma patients is hypovolaemic shock. The CNS is rapidly assessed in terms of demeanour,

responsiveness and alertness; bear in mind that poor oxygen delivery to the brain due to issues of respiration or circulation will affect demeanour.

Initial Emergency Wound Management

While life-threatening conditions are the priority, temporary and emergency management wounds should not be neglected. Emergency management should prevent any additional injury, minimise contamination and control systemic implications of the wound.

Bleeding should be controlled first. Apply direct pressure with sterile gauze swabs, or by bandaging. Pressure can be applied to brachial or femoral arteries if profuse arterial haemorrhage is present. A form of tourniquet can be applied above the wound if it is on a limb. Narrow elastic tourniquets such as Penrose drains put significant pressure on neurovascular structures and should only be used for up to 5 minutes. Bands 5-10cm wide can be used for up to 30 minutes. Blood pressure cuffs can be placed proximal to the wound and inflated to 20-30cm H20 higher than arterial pressure- these can be left in place for up to 6 hours. Ultimately ligation may be needed for larger vessels, and the limb then relies on collateral circulation.

Injuries caused by thermal or caustic burns will require emergency management at this stage. Chemical contaminants should be washed thoroughly from the coat, skin and eyes. Thermal injuries caused by burns or scalds should be cooled for an extended period of time under running water.

To prevent desiccation and further contamination of the wound, a sterile water soluble gel, or saline soaked gauze swab can be placed on the wound, and covered with a sterile towel or soft padded bandage; this protects the wound from the hospital environment

Once the patient is stable, a secondary survey can be performed; this is a full physical examination of the patient, and at this stage the wound and the surrounding areas can be evaluated to assess damage to other structures. Survey imaging of the thorax and abdomen may be required to assess for any penetrating injury. With wounds affecting limbs radiography may be required to assess the impact of any trauma on bones and joints. Damage to underlying neurovascular structures should be assessed. A management plan should take into account the wound's location, size, damage to local structures and the amount of tissue loss.

We are commonly presented with patients that have sustained wounds due to a wide variety of aetiologies in veterinary practice, and the patterns of trauma to tissues will likewise vary. An appreciation of the type of insult can give an idea of the resulting wound environment and the resulting impediments to healing, along with anticipated complications. The mechanism of injury should also alert the clinical team to the possibility of other unseen injuries.
Other than just the level of contamination in a wound, the presence of foreign material, and vascular damage will also have a profound effect on wound healing. Which of these factors are present can be anticipated from the original insult;

- thermal burn- large amounts of necrotic tissue, but little contamination or foreign material
- bite wound- crushed tissue and damaged vasculature with deep inoculation of bacteria
- shearing injury- extensive tissue loss, large amounts of contamination, large amounts of foreign material
- laceration- contamination, possibly foreign material, small amounts of necrotic tissue.

To optimise conditions for wound healing, steps should be taken to remove these impediments. Aseptic technique should be used to prevent introducing additional contamination from the hospital environment. The wound is packed with water soluble gel, or sterile saline soaked swabs, prior to a wide clip being performed. The surrounding skin can be aseptically prepared prior to lavage of the wound, aiming to loosen foreign material and necrotic tissue, and reduce bacterial numbers.

Debridement of the wound removes dead or damaged tissue, commonly used techniques include sharp debridement (surgical excision), mechanical debridement (eg wet-to-dry dressings) and hydrosurgery.
The presence of penetrating wounds over the abdomen is an indication for exploratory laparotomy once the patient has been stabilised.

Skin Preparation:

Aiming to prevent further contamination of the wound, especially with potentially resistant bacteria from the hospital environment.

- WEAR GLOVES
- Pack large wounds with sterile swabs or water soluble gel.
- Use sharp clean clipper blades (no missing teeth)
- Wetted scissors can be used for skin margins
- After clipping, remove swabs or gel from the wound, and replace with fresh prior to skin preparation.

Wound Lavage

Lavage reduces the number of bacteria present, and helps to loosen necrotic tissue and débris. Lavage solutions containing antibacterials or detergents should be avoided; they can cause cell damage, slow wound healing and may result in bacterial resistance.

The pressure for lavage solution needs to exceed the adhesive and cohesive forces of the contaminant, yet avoid pushing débris into the tissues and causing damage to vital tissues. The suggested force is 5–10 psi. In practice this can be achieved by using a bag of fluid with an 18–20 gauge needle fitted to the end of an attached giving set. The volume of lavage solution is equally important. For small, superficial wounds, 0.5–1 l is generally used; for larger wounds several litres of sterile lavage solution may be needed.

Wound Debridement

Any traumatic wound will require the débridement of devitalised tissues and foreign material in order to prevent infection and necrosis and to promote optimal wound healing. Débridement may be performed using a number of different methods.

Sharp débridement involves the use of a scalpel blade or scissors and may be carried out carefully in stages in order to preserve as much healthy tissue as possible. Subcutaneous tissue, fat, skin, fascia and muscle can generally be freely débrided. Tendons, vessels, nerves and bone should be débrided much more conservatively.

Mechanical débridement involves the use of dressings, irrigation or hydrosurgery. Wet to dry dressings are commonly used in veterinary practice but their use requires sedation or anaesthesia as removal is painful.

Autolytic débridement involves the use of wound dressings and solutions, e.g. hydrogels, and is not recommended in infected wounds.

Following débridement, a decision needs to be made about wound closure. Options include primary closure, delayed primary closure, secondary closure or secondary intention. If doubts exist over remaining contamination and necrotic tissue, a period of open wound management is indicated.