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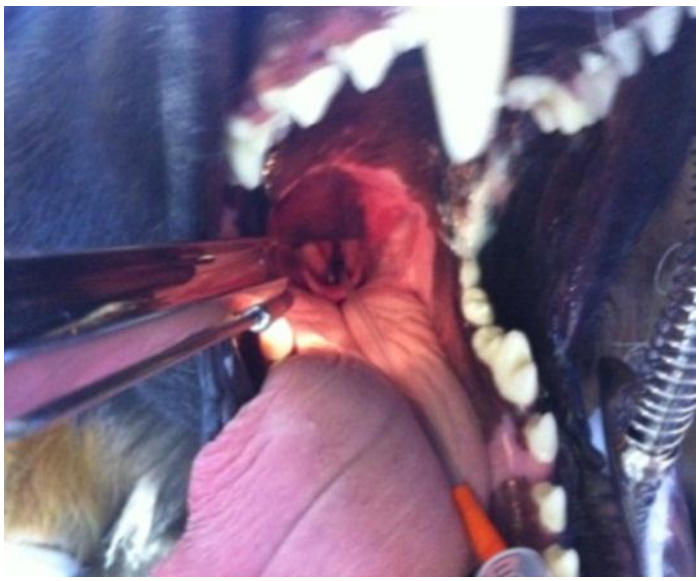
Emergency Surgery Mini Series

Session 1: Surgical Emergencies of the Respiratory System

Aidan McAlinden MVB CertSAS MRCVS DipECVS
Diplomate of the European College of Veterinary Surgery



1. Respiratory Emergencies

Upper Airway Obstruction: Laryngeal Obstruction

Airway Physiology

Air enters and exits the lungs during respiration by moving along pressure gradients generated by changes in the volume of the chest cavity. Inspiration, largely accomplished by contraction of the diaphragm, generates a negative (subatmospheric) pressure within the airways and air moves into the lung until pressures are equalised. The elastic recoil of the lungs following relaxation of the diaphragm drives expiration. This elastic recoil pushes the air from the lungs.

Airflow through the respiratory tract is opposed by the friction of air molecules against the walls of the airways. This airway resistance must be overcome during inspiration and expiration by creation of a greater pressure gradient within the airways than would be necessary in a frictionless environment. In the normal animal, this is carried out efficiently, with very little expenditure of energy. However, if resistance to airflow increases in the airway because of disease or structural design, the respiratory muscles must work harder to maintain normal airflow to the alveolus. The increased effort to breathe is noticed by the owner, who may then seek treatment for the animal. Additional consequences of this increased effort include generation of even greater negative pressures within the airway lumen, and a greater velocity of air moving over airway structures. The importance of both of these effects will become apparent in subsequent sections.

The larynx is composed of five cartilaginous units: the epiglottis, the thyroid, cricoid and paired arytenoid cartilages. These cartilages are arranged to create a four-sided box, with a lid (the epiglottis) that opens and closes the opening from the oropharynx. The box is designed to be a rigid structure that maintains a stable entrance to the lower airway. Muscles surround the laryngeal cartilages, and act to open or close the airway (rima glottis) at appropriate periods to control regulation of airflow through the larynx. Under disease conditions, however, the apparent rigidity of this box can be affected, and airway regulation may be compromised. In these instances, the larynx has a tendency to close during inspiration. Bernoulli and Venturi effects are the key mediators of this dynamic airway collapse.

The **Bernoulli principle** states that when airflow passes over almost any shape that is inclined in the direction of flow, the pressure of air on the top surface will be reduced, while that underneath will be increased. The Bernoulli principle is most commonly demonstrated by the ‘vertical lift’ over wing surfaces that keeps aeroplanes in the air.

Venturi effects are classically described for fluid travelling in a pipe. If a constriction occurs in a pipe, there must be an accelerated flow and a corresponding decrease in pressure at the site of the constriction, if flow through the pipe is to remain constant. At higher

flows, suction (i.e. increased negative pressure) can be generated at the site of constriction. Venturi effects can also be applied to air flow. It has been shown that the increase in air-speed at the site of a constriction is directly proportional to the reduction in cross-sectional area.

Bernoulli effects occur in the larynx as a consequence of airflow over arytenoid surfaces. Laryngeal muscles are usually able to resist the 'lift' generated across the arytenoid surfaces. If these muscles weaken, (e.g. in laryngeal paralysis), the vocal cords can be sucked into the glottis by the reduction in intraluminal pressure. Venturi effects contribute by increasing the relative negative pressure within the glottic lumen, which compounds the tendency for dynamic airway collapse. Both Bernoulli and Venturi effects are dramatically heightened as the speed of airflow through the larynx increases, such as occurs with exercise, panting etc. The intra-laryngeal "suction" generated by Venturi effects probably also contribute to progressive collapse of the laryngeal cartilages in the brachycephalic breeds, and also to the eversion of laryngeal saccules.

Emergency Care Of Laryngeal Obstruction

As summer approaches, the likelihood of a day's surgery being interrupted by a respiratory emergency increases. Prompt and effective action is vital. By ensuring that essential equipment is close-at-hand, and staff are practiced in effective resuscitative techniques, the likelihood of patient survival increases. Although the intensity of resuscitative efforts will be dictated by the degree to which respiration is impaired, action is directed at restoring an effective airway; providing oxygen supplementation; and managing hyperthermia. Attention is then directed at getting the patient stabilised for definitive surgical management. In many cases, this will require safe transportation to a surgical referral centre.

Techniques that may be employed in the treatment of acute laryngeal obstruction are outlined in the following table:

Management of Acute Laryngeal Obstruction
Restoration of Airway
Minimise dynamic collapse (conservative)
Endotracheal intubation
Tracheostomy
Provide oxygen supplementation
Manage hyperthermia
Stabilise potential pulmonary oedema

Minimise dynamic collapse

The influence of increased respiratory rate and effort on airflow and dynamic airway collapse has already been discussed. Even if the animal is only minimally distressed at the time of presentation, every precaution should be taken to maintain calm and minimise excitement or distress in the patient. The anxiety associated with handling, examination and hospitalisation will elevate the respiratory rate, and exacerbate dynamic airway collapse. Place the animal in a cage in a quiet, but readily observable area of the hospital. Provision of a cool breeze by an electric fan or open window is beneficial. If the animal continues to pant, administration of a sedative (e.g. acetylpromazine 0.02 – 0.04 mg/kg, SQ) may calm the patient. Increasing the inspired fraction of oxygen above that of room air will also assist in relieving the signs of respiratory distress. The technique chosen should be simple and effective and not cause undue stress to the patient. Many patients' often resent oxygen masks and continued struggling to maintain the mask in place serves only to heighten anxiety and worsen the dyspnoea. Several techniques are described for provision of long-term oxygen supplementation, including intranasal oxygenation, oxygen tents, and oxygen cages. If dyspnoea or cyanosis continues or progresses in spite of this action, more aggressive intervention is required.

Provision of Oxygen Supplementation

In animals with only moderate dyspnoea, increasing the inspired fraction of oxygen above that of room air may be all that is required to relieve respiratory distress. Again, the technique chosen should be simple and effective and not cause undue stress to the patient. For example, many patients often resent oxygen masks and continued struggling to maintain the mask in place serves only to heighten anxiety and worsen the dyspnoea. Several techniques are described for provision of long-term oxygen supplementation, including intranasal oxygenation, oxygen tents, and oxygen cages.

Endotracheal intubation

If respiratory distress rapidly progresses in spite of more conservative efforts, intravenous anaesthesia and immediate endotracheal intubation is required. Propofol (Rapinovet®) provides superior control over induction of anaesthesia in these distressed animals, and is the agent of choice. Instead of administering a bolus calculated on the animal's body weight, progressive infusion of one-millilitre increments is performed until suitable relaxation is obtained for laryngoscopy and intubation. A range of cuffed, endotracheal tube sizes should be available in case the glottic lumen is obstructed by either a mass or laryngeal collapse. It is important to keep the head elevated during induction and intubation to avoid inhalation of oesophageal content.

Once the animal is intubated, ventilation with 100% oxygen is begun by connection to an anaesthetic circuit. If the animal is not breathing spontaneously, IPPV should be started and

the patient assessed carefully. Other systemic effects associated with a period of hypoxia and dyspnoea should be suspected and appropriate intervention taken.

Once the patient is stable, and breathing spontaneously, there is now time for a more critical evaluation of the animal's condition. Clinical examination (including laryngoscopy), blood tests and radiography are best performed at this time. The owner can also be questioned more thoroughly about the history and events that preceded the respiratory collapse. If necessary, the patient can be kept anaesthetised with a Propofol infusion while these examinations are performed. Careful monitoring is essential during anaesthesia, as the existence of acidosis, cardiac ischaemia and circulating cathecholamines may predispose to the development of ventricular arrhythmias.

Manage hyperthermia

Recognition of hyperthermia is also essential. Rectal temperatures of dogs presenting in acute respiratory distress can be of the magnitude of 40 - 41°C. Hyperthermia occurs because the animal is unable to lose heat by panting. In addition, the increased respiratory effort associated with obstructive dyspnoea generates a terrific amount of body heat, contributing to a vicious circle. Hyperthermia can be life threatening. At the very least, the elevated body temperatures continue to drive the respiratory centre, which contributes to the respiratory crisis. Hyperthermia is often the cause of patients failing to respond satisfactorily to other conservative measures, including oxygen supplementation and sedation. Immediate body cooling is essential. Cold-water showers, dowsing the skin in methylated spirits, or immersion in an ice-bath are effective in most cases. Use of electric fans assist cooling by convection. Total body cooling is usually only necessary for a few minutes. Continued monitoring of rectal temperature is essential to prevent hypothermia.

Stabilise potential pulmonary oedema

Another potential complication that may develop following an episode of acute respiratory collapse is non-cardiogenic oedema. Development of pulmonary oedema following a period of upper respiratory tract obstruction is a well-recognised phenomenon in humans, and is referred to as negative pressure pulmonary oedema. While few reports exist in the veterinary literature, there is sufficient evidence to indicate that this phenomenon also exists in our patients, and may impact adversely on clinical outcome.

The cause of the non-cardiogenic pulmonary oedema that occurs following laryngeal obstruction is believed to be the result of two mechanisms working together to produce transudation of fluid from the pulmonary vessels. First, the hypoxic insults results in a massive sympathetic outflow, causing pronounced peripheral vasoconstriction and systemic hypertension. Blood then accumulates in the pulmonary vasculature, resulting in pulmonary capillary hypertension and subsequent oedema. Secondly, inspiration against a closed airway may cause a tremendous increase in transpulmonary pressure, resulting in a hydrostatic

pressure gradient favouring transudation of fluid from the pulmonary vasculature into the parenchyma.

Clinical signs in these animals range from coughing to severe respiratory distress. Clinical signs can develop rapidly (less the 4.5 hours of insult), and incidence does not necessarily correlate with the duration of obstruction.

Thoracic radiography is most useful in assessing the extent and pattern of pulmonary oedema. The predominant pattern is alveolar, with involvement of the caudodorsal quadrant of the lung field occurring most commonly. The cardiac silhouette is usually normal, providing evidence to rule out cardiac failure as a cause of the pulmonary oedema.

Treatment of pulmonary oedema consists of providing oxygen supplementation, appropriate fluid resuscitation, and diuretics (e.g. furosemide). The use of mechanical ventilation (± positive end-tidal pressure) and positive inotropic support of blood pressure is argued by some to improve outcome. In many cases, rapid responses to treatment are seen, but significant mortality rates are reported in severe cases. In one study, the survival rate following treatment for pulmonary oedema as a result of airway obstruction was only 50%.

Safe Recovery and Transportation of the respiratory patient

Recovery of the patient who was anaesthetised due to escalating respiratory obstruction is often problematic. In many cases, removal of the endotracheal tube is associated with a resumption of increased respiratory effort and dyspnoea. In these patients, provision of a temporary tracheostomy tube is necessary to facilitate recovery, and ensure a stable airway is available until definitive treatment of the laryngeal condition can be performed.

Placement of a tracheostomy tube can be performed quickly and simply, and requires a minimal amount of equipment. In the majority of cases, a tracheostomy can be performed on a stable, anaesthetised and intubated patient. Rarely is it necessary for a tracheostomy tube to be placed in the conscious, cyanotic patient as an emergency cut-down.

A tracheostomy tube should only be considered for short-term maintenance of an airway, and definitive treatment of the laryngeal condition should be actioned as soon as possible. Tracheostomy bypasses the normal protective mechanisms of the lower respiratory tract and bacterial colonisation of the trachea is inevitable. In addition, the presence of the tube interferes with normal ciliary motion and coughing, which further compromises the defenses of the lower respiratory tract. Proper care of the tracheostomy tube is therefore imperative. Aseptic technique and sterile equipment are required in all manipulations of the tracheostomy site to delay development of infection and productive secretions.

OPERATIVE TECHNIQUE:
Temporary (emergency) tracheostomy

Positioning
Dorsal recumbency. The front legs should be secured caudally. The head is extended with a sandbag placed under the neck to elevate the trachea and larynx close to the skin.

Anaesthesia Considerations
Although the need for tracheostomy suggests severe upper airway obstruction is present, surgery can usually be performed on the stable, anaesthetised and intubated patient to facilitate recovery. Rarely is an emergency 'cut-down' procedure in a severely dyspnoeic patient required.

Effects of severe dyspnoea should be assessed (e.g. acidosis, hypercapnia, anoxia, pulmonary oedema, hyperthermia)

Time required *Short (15-30 mins)*	Assistant ***Not essential***

Tray Extras
Tracheostomy tubes are available commercially. Uncuffed tubes are ideal for most instances, unless the patient is to be maintained on a ventilator. If commercial tubes are not available, a 'cut-down' endotracheal tube may be used. The diameter of the tube should not be more than 50% the diameter of the trachea. Use tubing made of inert materials (silicone, nylon) rather than PVC.

Important Surgical Landmarks
Cricoid cartilage, tracheal rings, sternohyoideus muscles

Surgical Technique
Make a ventral midline incision beginning from the base of the larynx (cricoid cartilage) and extending for 4-5 cm. Continue the incision through the subcutaneous tissues and fascia to expose the underlying muscle. Separate the paired sternohyoideus muscle. Counting from the cricoid cartilage, identify the third, fourth and fifth tracheal cartilage rings. Make a transverse incision through the annular ligament between the third and fourth, or fourth and fifth ring. Do not extend the incision more than 50% about the diameter of the trachea. Place a suture of monofilament nylon about the tracheal ring on either side of the tracheostomy incision. Tie loosely and leave the ends long. These sutures may be used to assist opening the tracheostomy incision should the tube need to be replaced in the post-operative period. If one is present, the endotracheal tube can now be removed to permit access to the lumen of the trachea. Suction any blood and/or mucus from the airway. The tracheostomy tube can now be inserted. The anaesthetic circuit may not be connected to the tracheostomy tube to provide continued delivery of oxygen and anaesthetic agents.

Appose the sternohyoideus muscle, subcutaneous tissue and skin about the tube. The tube can be tied about the neck with gauze, or secured to the skin with suture.

Post-operative Care
A tracheostomy tube should only be considered for short-term airway maintenance. Definitive correction of the airway disease should be actioned as quickly as possible.

Close monitoring for airway obstruction or dyspnoea, oxygen supplementation as required, analgesia, antibiotic

Tube maintenance: Aseptic handling of the tube and instrumentation is essential at all times. Aspiration of mucus from airway will be required on a regular basis (15 mins – 2 hourly). Inject 1-2 mls of sterile saline into the tube prior to suctioning the airway to help loosen secretions. Suction the tube and distal trachea with a sterile suction system. If the tube becomes heavily occluded with secretions, it should be replaced with a new sterile tube.

Following removal of the tube, the trachestomy site can be left to heal by second intention. Appropriate cleaning of the wound may be required.

Complications

Complications with tracheostomy tubes will increase exponentially with the duration of use.

Tube dislodgement, obstruction and dyspnoea; coughing/gagging; pneumonia; dysphagia, vomiting/regurgitation; aspiration; tracheal stenosis; tracheocutaneous fistula; laryngeal nerve injury

Lower Respiratory Emergencies:

The Chest Trauma Patient

Incidence

Injury to the structures of the chest should be suspected following any traumatic injury, particularly those involving a fall, blunt injury or road traffic accident. Thoracic injuries may be a 'silent' component of many emergency presentations. Injuries such as fractures and open wounds are often more obvious and can distract from more life-threatening internal injuries. It is therefore vital to suspect internal injury in every trauma patient until ruled out by detailed clinical assessment and complimentary diagnostic investigations. Vital time can be easily wasted treating less serious injuries which can ultimately lead to the demise of the patient especially if the patient becomes distressed or anaesthesia is induced.

Causes of Injury

The nature of the traumatic event may increase our suspicions for the potential of thoracic injury. In a large review of 300 dogs presenting with musculoskeletal injuries, 31% of animals involved in a road traffic accident were found to have concurrent thoracic injuries. This compared to only 2.4% when the musculoskeletal injury occurred through other means (e.g. falling from a height, fracture while running etc.). A similar high rate (52%) of thoracic injury was detected in cats following RTA, but cats generally had a higher rate of injury (38.7%) regardless of the inciting cause compared to the dog.

Penetrating injuries to the chest from dog fights, ballistics or other sources should always be surgically explored (once the patient has been adequately stabilised). The 'ice berg' effect applies to these injuries. Often there is an inconsequential skin wound which frequently hides significant underlying trauma. One study found that 50% of these cases had underlying pulmonary injury.

Pulmonary contusions represent the most common type of pathology seen following thoracic injury (55% or more of all cases). This is often not given the same consideration as widely discussed diaphragmatic hernias or pneumothorax but is just as important. Haemorrhage and oedema of the alveoli leads to regions of the lung being perfused but not ventilated (ventilation:perfusion mismatch) with resultant hypoxia and hypercapnia. The clinical impact will depend on the amount of lung tissue affected by the injury. Haemoptysis, or the presence of bloody froth in the oropharynx is an indicator of severe pulmonary contusion.

Recognition of Injury

In a normal animal Injury to the respiratory system usually causes a change in breathing efficiency – this is noted initially by an increase in effort. As the severity of injury increases,

the animal may begin to show signs of distress due to oxygen depletion; the animal may be unable to breathe effectively if laying or sitting down (orthopnoea), or each respiratory effort may become extremely laboured (dysponea). Noises (stertor/stridor) may become apparent with the increased effort, particularly if there is obstruction to the airway (e.g. collapsed maxillary fractures or laryngeal cartilage fracture). The source of these noises may give some clue to the underlying pathology.

a) Physical Assessment

Clinical signs of respiratory injury are not always obvious; in a recent study 75% of patients that had evidence of thoracic trauma on radiography, blood gas evaluation or ECG had gone undetected on clinical evaluation. This may have significant consequences for an individual – failure to recognise thoracic injury before proceeding with a prolonged general anaesthetic, or leaving the animal unmonitored overnight, for example, may result in death due to the effects of progressive hypoventilation, hypoxia (low oxygen) and hypercapnia (high carbon dioxide).

It is important to have knowledge of normal respiratory physiology to understand the changes that occur during injury or disease and thus helping recognise subtle abnormalities. In a normal animal chest wall movements are almost undetectable during respiration. During inspiration the ribs are pulled cranially and laterally by the external intercostal muscles and the diaphragm contracts. Negative pressure is created within the pleural space which expands the closely coupled lungs permitting air to flow into the lungs. The recoil of the elastic structures within the lungs allows passive expiration. Progressive respiratory distress due to oxygen depletion will lead to increased work of inspiration. The secondary muscles of respiration (scalenus and sternomastoideus) are recruited to help elevate the chest wall and pull the sternum cranially. The alae nasi muscles also become active and can be noted by subtle flaring of the nostrils. Expiration becomes active with contraction of the internal intercostal and abdominal muscles leading to so called ‘abdominal breathing’.

Therefore careful assessment of respiratory movement often provides the first clues about underlying thoracic problems. Standing back and observing the animal from a distance thus avoiding stress may aid detection of altered thoracic wall movement. The respiratory pattern may be restrictive in nature (shallow and rapid) with ‘space filling’ conditions like effusions, diaphragmatic rupture and pneumothorax due to uncoupling of the lung and chest wall. With contusions and pulmonary injury, the breathing tends to become more laboured, with a greater abdominal component evident. Subtle changes in posture may be evident. Animals may decide to stand /sit instead of lying down, they may extend their head and neck to relieve upper respiratory tract resistance or they may abduct their elbows to permit maximal excursion of the chest wall. In animals with a tension pneumothorax a ‘barrel shaped’ contour of the chest wall can sometimes be appreciated. Other signs that may be noted include exercise intolerance, tachypnoea, tachycardia, cyanosis and open-mouth breathing. The latter is a very concerning sign in the cat – IT MEANS HANDLE WITH CARE!! Other signs may be

more referable to the causative disease, and include fever, depression, vomiting/diarrhoea, anorexia, ascites, peripheral oedema, orthopaedic trauma, cardiac murmur or arrhythmia.

Thoracic auscultation with a stethoscope allows air sounds to be assessed over the whole area of the chest. Dullness, an absence of breath sounds or displacement of the cardiac beat suggests the presence of effusion, or interfacing tissue (diaphragmatic hernia); these signs may be gravity dependant. Harsh sounds, crackles or wheezes may suggest the presence of fluid in the smaller airways, and indicate pulmonary contusion. In rare occasions borborygmus may be detected in diaphragmatic hernia cases with intestine displaced into the thorax. Percussion can be useful in large breed dogs. Increased resonance of the chest may be detected in animals with pneumothorax and a fluid line may be detectable in animals with pleural effusion. It is useful to compare the left and right hemi-thoraces during percussion.

b) Monitoring Techniques

Physical evaluation alone is insensitive for the detection of many thoracic injuries. In order to improve detection of subtle and/or progressive injuries, a clinician may rely on various objective monitoring aids: blood gas machines, pulse oximetry, thoracocentesis and electrocardiography (ECG).

Pulse oximetry: Pulse oximetry is a simple, and readily available technology. It can provide instantaneous and continuous recording of oxygen status, and provide early detection of hypoxia. The probes are usually well tolerated by the critical patients, and should form the first-line monitoring technique for all trauma patients for early detection of hypoxia. However, it is important to recognise the limitations of pulse oximetry. The value displayed on the screen is simply a percentage oxygen saturation of haemoglobin (should be >92% on room air); thus if the patient is anaemic, has low haemoglobin concentrations, or has poor peripheral circulation (due to shock, hypovolaemia etc) then the actual oxygen delivery to tissues can still be sub-optimal, even though the SpO2 is recording a high value.

ECG: Cardiac arrhythmias may occur in chest trauma cases secondary to reduced myocardial perfusion and ischemia and a common in patients with diaphragmatic hernia. ECG monitoring can aid the detection of these and provide evidence for concurrent myocardial damage.

Blood Gas measurement: Blood gas measurement provides the most accurate and comprehensive evaluation of respiratory function – i.e. ventilation efficiency and perfusion, a measure of gaseous exchange at alveolar level. It requires specialised equipment, and the collection of arterial blood samples (though central venous samples can also be used).

Arterial blood sampling is normally performed from the dorsal pedal artery, which courses on the medial aspect of the hock; this may be catheterised if repeat sampling is required.

An animal's ability to ventilate effectively is determined by the PaCO2 value – if it is low, the patient is probably hyperventilating. With shallow respiratory movements due to pain, or altered pulmonary function, the CO2 may be high. Because CO2 is an important 'buffering' agent for balancing the build-up of metabolic acids in the blood stream (lactic acid etc), the pH value of the blood gas can also provide some important information on the patient's systemic status.

Estimation of the effectiveness of alveolar perfusion/gaseous exchange is measured by the PaO2 value. In patients breathing room air, a value less than 80mmHg is concerning and indicates a need for oxygen supplementation.

Thoracocentesis: If altered respiratory efficiency is suspected (based on disturbed respiratory efforts, or auscultative findings), thoracocentesis can provide a rapid and effective technique to rule out the presence of a pleural effusion or pneumothorax. If performed properly, thoracocentesis can allow these concerns to be swiftly eliminated from the diagnostic list; if confirmed, therapeutic relief is achieved in the same step.

c) Imaging Assessment

Radiography: Radiography may be helpful in the investigation of thoracic trauma but does NOT form part of the emergency assessment and treatment. Radiography should only be attempted AFTER stabilisation. Accurate positioning can be very difficult in the dyspnoeic patient, and poorly positioned radiographs are of little diagnostic value. Excessive and inappropriate handling can be very stressful, and extreme care is required with dyspnoeic patients. If a patient struggles DO NOT PERSIST. If chemical restraint is required' general anaesthesia is often safest. The ventro-dorsal view is contra-indicated due to the risk of patient de-compensation. Oxygen supplementation should be considered at all times – a flow-by technique is the least stressful.

Initial imaging should be performed with the patient in sternal recumbency only. This view is the most sensitive for pleural effusion, and thus will provide information to proceed with further support or treatment. It will also allow assessment of the severity of any effusion, and may provide some guidance as to whether imaging in lateral recumbency will be tolerated by the patient. (Standing lateral views can be obtained if appropriate radiation safety measures are in place). Rib fractures can be difficult to detect, but if seen will provide some indicator of the potential for underlying bruising to the lung tissue.

Radiographic evidence of pulmonary contusions is often not immediately apparent after an injury – it may take 6-12 hrs for these changes (increased interstitial density; air bronchograms etc) to become evident.

Ultrasonography: Ultrasonography can be invaluable for the identification of neoplastic lesions, diaphragmatic hernia, or for collection of pleural fluid when only small quantities exist. Ultrasonic examination is a minimally stressful technique, and can usually be performed with the patient in its most comfortable position (standing, or sternal recumbency). The presence of a fluid effusion in the pleural cavity provides an excellent acoustic media, and good images can usually be obtained.

Clinical Interventions

Provision of Oxygen Supplementation

In animals with only moderate dyspnoea, increasing the inspired fraction of oxygen above that of room air (21%) may be all that is required to relieve respiratory distress. The technique chosen should be simple and effective and well tolerated. Many patients often resent oxygen masks and continued struggling to maintain the mask in place serves only to heighten anxiety and worsens the dyspnoea. Several techniques are described for provision of long-term oxygen supplementation, including intranasal oxygenation, oxygen tents, and oxygen cages.

INTRANASAL OXYGEN: If the patient will tolerate placement of the catheter, nasal cannulation is an excellent method for providing long-term oxygen therapy. An intranasal catheter will increase the inspired fraction of oxygen to about 40% at flow rates of 100 ml/kg/minute, and is considerably less wasteful of oxygen than other methods. Gastric distention can occur with excessively high flow rates so a regulated oxygen source must be used (e.g. anaesthetic machine). Humidification of the oxygen is imperative to prevent dehydration of the airways.

Nasal cannulation can be performed in the conscious patient, with any standard polyurethane nasogastric feeding tube. The tube is inserted into either nasal cavity in a similar fashion to nasogastric intubation along the ventral meatus. In this instance, however, the tip of the tube terminates in the mid-nasal cavity (pre-measure to the level of the medial canthus). Prior instillation of local anaesthetic (Opthaine®) into the nostril, and lubricating the tube with xylocaine gel, will improve patient compliance. Once the tube is inserted, it is secured to the perinasal skin with suture or superglue. An Elizabethan collar may be necessary to prevent interference with the tube by the patient. As an alternative, nasal prongs can be employed.

OXYGEN TENT: A personal oxygen tent can be created from an Elizabethan collar and cling film. Oxygen is supplied via a tube which enters the Elizabethan collar at the back. High flow rates (> 10 L/min) are required to prevent carbon dioxide building up in the tent, but humidification is not required. Specifically designed collars are also available.

INCUBATOR/OXYGEN CAGE: Oxygen cages are considerable more wasteful of oxygen but can allow high (>40% FiO_2) atmospheric rates of oxygen to be maintained. However, they restrict direct access to the patient for examination. If the door is opened, the benefit of oxygen supplementation is lost. Precise control of inspired oxygen fraction is therefore difficult to achieve. Care is required to ensure excessive temperature or humidity does not develop within the cage.

OXYGEN MASKS / FLOW BY: If tolerated this is an excellent method for short-term supplementation. High inspired oxygen (~50% FiO_2) to be achieved but does require flow rates of 5-10 L/min and is wasteful. The mask should fit snugly but not airtight to prevent carbon dioxide build up. If the patient does not tolerate the mask, simple flow by oxygen can be used by placing the mask in front of the patient.

Analgesia:

Most animals with thoracic trauma experience significant pain particularly if the thoracic wall is compromised (e.g. rib fractures, flail chest etc.) or if a thoracostomy tube is in place. Not only is pain a welfare consideration but thoracic wall pain also results in hypoventilation, depressed cough, promotion of atelectasis and thus predisposition to infection. Opioids are safe to use in thoracic trauma patients and should not result in significant respiratory compromise when used at appropriate clinical doses. As always, pre-emptive multimodal analgesia employing intercostal nerve blocks, wound analgesic catheters, intra-pleural block and/or non steroidal anti-inflammatory drugs (if cardiovascular system stable) will be most effective.

Thoracocentesis:

Thoracocentesis is easily performed, with care. Equipment required includes a small butterfly catheter (or an 'over-the-needle' intravenous catheter (20g) attached to extension tubing), a three-way stopcock, and syringe. The site of thoracocentesis will be dictated by physical examination or imaging studies, particularly if a unilateral effusion is present but is generally performed at the 6^{th}, 7^{th} or 8^{th} intercostal space. A more dorsal position is chosen for a pneumothorax and a ventral position for effusions. In general, the mediastinum is a permeable membrane, and it is usually possible to drain both sides of the chest from a single puncture site. The animal should receive oxygen supplementation throughout. Following infiltration of local anaesthetic at the site, the needle is inserted CRANIAL to the edge of the rib. Once the pleura is penetrated, the needle should be re-directed parallel to the thoracic wall before aspirating the contents of the pleural space.

Chest Drain placement and management

Thoracostomy tubes are indicated when greater than two thoracocenteses are required within a 24 hour period or if fluid / air rapidly re-accumulates following drainage. Tubes are available from several commercial suppliers. They consist of a tube made of PVC or silicone, together with a metal stylet to facilitate placement. They should have an end hole and at least three side holes. Extra fenestrations may be added if required but should not exceed one third of the tube diameter. Tubes come in a range of sizes. The correct size for an individual patient may vary on the type of effusion present, but a usual rule of thumb is to place a tube approximately equal to the diameter of the main stem bronchus or half – two thirds the width of the intercostal space (see guidelines in table below). The tube should be of sufficient length to reach as far cranial as the second rib. A wide bore may be required for thicker inflammatory secretions. Other equipment that will be required includes a scalpel blade, three-way tap, appropriate adaptors from the tube to the three way tap, syringe, gate clamp and suture material. Some method of providing adequate security of the various connections to the chest drain is also essential to prevent dislodgement.

Patient size	Recommended tube size
Cats / dogs < 7 kg	14-16 F
Dogs 7-15 kg	18-22 F
Dogs 16-30 kg	22-28 F
Dogs > 30 kg	28-36 F

Complications of chest drains

When appropriate care is taken with the placement of chest drains, it is unusual for significant complications to occur. Potentially, a chest drain trocar can bruise/perforate the underlying lung tissue, causing pneumothorax and/or haemorrhage. Because there is usually air of fluid present within the pleural cavity, this can act as a reasonable buffer to prevent such injury from occurring.

Following placement of the chest drain, the most common complications include pain, stoma infection/irritation, subcutaneous emphysema, and poor ‘seal’ of the stoma due to an inadequate subcutaneous tunnel (particularly in lean animals, or due to poor placement). Catastrophic pneumothorax may occur if the patient interferes with the tube, due to dislodgement of sealing bungs, or by biting through the tube.

Chest drains can be very uncomfortable for some patients (especially cats), either due to direct pleural irritation, impingement on intercostal nerves, or other reasons. Appropriate use of opiate analgesia may be necessary. Regional anaesthesia may be of value in other patients.

Prevention of stoma infection requires careful attention to asepsis during placement and handling of the drain. The use of protective dressings to prevent contact contamination of the area during hospitalisation should also be considered. Latex or polyurethrane drains are likely to induce a minor cellular reaction if a drain is left in place for over 5 days due to chemical irritation of the tissues. This is usually self-limiting, but may be the cause of some discomfort for the patient.

Subcutaneous emphysema can develop if there is an insufficient seal about the surface of the drain, either as a result of air leaking directly from the pleural space (with a pneumothorax) or by direct extension from the skin surface. This is unusual, but may occur when a drain has been left in situ for a prolonged period (with progressive necrosis of intercostal muscles about the pleural wall stoma), or if an inadequate subcutaneous tunnel was performed at the time of tube placement.

The importance of creating a good subcutaneous tunnel at the time of chest tube placement cannot be understated. In very lean animals, it can be difficult to achieve a good seal, however. Without adequate tunnelling, there is a potential for atmospheric air to track along the chest drain during respiratory movements. In addition, if there is a poor ‘seal about the skin/tube interface as a result of poor tunnelling, during aspiration of the chest drain large volumes of air may be obtained giving the impression of pneumothorax, when in fact this air is simply being drawn from the atmosphere. This complication can sometimes be difficult to differentiate – although if >1000ml of air has been removed from a dog, with no signs of dyspnoea or change in respiration pre- and post- drainage, then it is likely that the air being withdrawn was not within the pleural space.

Pleural Effusion in the Dog and Cat

Development of pleural effusion in the dog or cat can represent a common emergency for the general practitioner. There are several potential causes for pleural effusion, including accumulation of air or fluid within the chest. Types of pleural effusion are outlined in the following table:

Causes of Pleural Effusion
Hydrothorax
Pneumothorax
Haemothorax
Chylothorax
Pyothorax

Specific Causes of Pleural Effusion

1. Inflammatory Exudate ('Pyothorax')

Pyothorax is an accumulation of purulent exudate in the pleural cavity. The majority of pyothorax conditions will have a septic aetiology, with bacterial/fungal inoculation of the pleural cavity occurring as a result of penetrating wounds, migrating foreign bodies, haematogenous spread, or by direct extension of local disease.

Pyothorax can occur in any animal, at any time. There is no breed, sex or age predilection. However, younger male cats may be more prone than other cats (due to fighting and bite wounds to the chest).

Clinical Signs

Affected animals are presented due to signs referable to the pleural effusion. Septic signs may also be seen in these animals, including pyrexia, anorexia, weight loss and severe malaise. There may be a delay of several weeks between the injury which established the infection, and the onset of obvious clinical signs, so historical data may be unhelpful.

Gross inspection of thoracic fluid, combined with routine fluid analysis is useful diagnostic for pyothorax. The fluid is purulent in appearance (red/creamy/amber in colour, with marked turbidity due to high cell numbers). Heavy 'granules' may be evident within the fluid with Nocardia or Actinomyces infection. The fluid will have a high protein content, and cytological examination will reveal large numbers of neutrophils. The neutrophils may be degenerate, or non-degenerate, depending on the causative agent.

Fluid samples should be submitted for specialist laboratory analysis, including anaerobic and aerobic culture. However, it is not uncommon for a negative culture to be obtained, even when obvious features of infection are present clinically.

Treatment

Successful treatment of pyothorax requires prompt and aggressive management. Systemic stabilisation of the septic patient is essential, using intravenous fluids, antibiotics and nutritional support. This is combined with aggressive drainage of what is essentially a large 'abscess' within the pleural cavity. Effective management can only be obtained with a chest drain.

Following initial placement of the chest drain, the pleural cavity is copiously lavaged performed with warmed, sterile saline. No more than 10 ml/kg of fluid should be instilled into the thoracic cavity at any time. Irrigation is continued until the drainage fluid become clear. The initial irrigation of the pleural cavity is performed under general anaesthesia, as this will allow higher volumes of lavage to be circulated through the chest without distress to the patient. As outlined above, initial stabilisation of the patient may be required with needle thoracocentesis and oxygen supplementation before anaesthesia is contemplated.

Following this initial lavage, subsequent irrigations of the chest cavity can be performed in the conscious patient. Irrigation is performed 2-4 times daily, depending on the severity of the condition. The addition of an antibiotic to the lavage fluid offers no advantage over systemic treatment. The choice of a systemic antibiotic will ideally be based on culture results, but in the event of a negative culture, the choice is often empirical. In most instances, a broad spectrum antibiotic with anaerobic cover, is effective. Treatment must be continued for at least 4-6 weeks to prevent relapse of infection.

With a pyothorax, there is a possibility that pockets of fluid/infection may become walled off from the rest of the chest due to thickening of mediastinal folds. A chest drain may also become entrapped within such a pocket. Two or more chest drains may need to placed initially, and repositioning of the patient during drainage may also be required to facilitate complete evacuation of all lavage fluid. A common finding during treatment is to instil a large volume of saline, yet only obtain small amount back! In this instance, radiographs are useful to determine the position of the tip of the chest drain. It is not unusual for a drain to end up in the dorsal thoracic cavity as a result of respiratory movements. In this position, it is not surprising that it can be relatively ineffective in draining fluid from the standing animal. Heparin added to the lavage fluid (1500 units/100ml of lavage) is also considered by some to be helpful in reducing fibrinous pockets from developing, but this has not been rigorously assessed.

Surgical intervention is indicated if there is no improvement in the animal's condition within 3-4 days of treatment, or when there is an identifiable cause for the infection on initial investigations. Exploration is best performed via median sternotomy, as this allows both sides

of the chest to be explored with ease. However, surgical manipulation of intrathoracic organs is more difficult via this approach. Surgical exploration allows necrotic tissue, foreign material, or localised abscesses to be removed. A thorough irrigation of the thoracic cavity should be performed prior to closure.

Prognosis

A reasonable prognosis is expected with prompt and aggressive management. Recent papers suggest that complete resolution of infection occurs in over 75% of patients that are managed with a combination of pleural drainage and antibiotics.

Fibrosing pleuritis may limit lung expansion and be a cause of peristent dyspnoea or exercise intolerance. Decortication of the lungs may be required in this instance.

2. Pneumothorax

Pneumothorax occurs when there is an accumulation of air within the pleural cavity. A pneumothorax may be classified as open or closed, depending on whether there is patent communication between the pleural cavity and the external environment. A tension pneumothorax occurs when a damaged section of tissue acts as a one-way valve, effectively allowing air into the pleural cavity during inspiration, but preventing its escape. A tension pneumothorax will lead to rapid respiratory failure as the lungs are quickly prevented from expanding effectively due to the high intrapleural pressures. A pneumothorax may also occur as a result of a traumatic incident (blunt injury, penetrating injury), or can occur spontaneously.

Traumatic Pneumothorax

Traumatic causes represent the most common cause of pneumothorax in the dog and cat. Blunt trauma (e.g. kicks, falls, road traffic accidents etc) can cause rupture of the lung tissue or bronchial tree due to pressure overload or shearing forces [a closed pneumothorax]. Less commonly, direct puncture of the lung may occur due to penetrating injuries such as stake wounds, rib fractures, or ballistic injuries. Thoracic injury is common in both the dog and cat following road traffic incidents, with about a third of all cases have some degree of thoracic involvement. In two separate studies, pneumothorax occurred in between 36 and 50% of these cases.

Spontaneous Pneumothorax

Spontaneous pneumothorax occurs in purportedly healthy dogs, without any evidence of antecedent trauma. The pneumothorax may occur as a primary event (i.e. without any evidence of underlying pulmonary disease) or may be secondary to disease of the pulmonary parenchyma (e.g. pneumonia, neoplasia, parasitic disease. Perhaps the most common cause of spontaneous pneumothorax in the dog is due to rupture of sub-pleural blebs, cysts or bullae, (also termed bullous emphysema). The reason for development of sub-pleural blebs in lung parenchyma is unknown, but they occur with high frequency in tall male humans. Cigarette smoking is an important risk factor. Spontaneous pneumothorax appears to occur more commonly in the large breed, deep-chested dog suggesting a similar predisposition for sub-pleural bleb formation.

Diagnosis

Clinical signs of pneumothorax depend on the amount of air present within the pleural cavity, and the presence of underlying pulmonary disease. For example, in the road traffic injury patient, concomitant pulmonary contusions may have a significant impact on an animal's ability to tolerate a smaller amount of pneumothorax than an animal without this

additional complication. Other physical injuries (e.g. fractures) may cause pain, leading to an increased respiratory rate and exacerbation of dyspnoea.

Thoracic auscultation usually reveals dull, muffled lung and heart sounds, especially dorsally. The chest will be hyper-resonant to percussion. Respirations will be shallow and rapid. With tension pneumothorax, respiration becomes ineffective, the chest is barrel-shaped and in maximal extension. The animal will be very distressed, cyanotic, and close to death without immediate relief of the pneumothorax.

Radiography

Bilateral pneumothorax is usually present, as air diffuses easily across the mediastinum. The sternal projection is the most sensitive view for detection of even a minor pneumothorax. The vascular pattern of the lung should extend to the lateral recesses of the diaphragm. Careful inspection of the radiograph, using a hot-light if necessary, may be required in some cases. When larger amounts of air are present, the lung lobes will be collapsed and atelectic. In a lateral projection, the heart will appear elevated from the sternum.

Radiographs should be carefully inspected for evidence of associated pulmonary disease (e.g. rib fracture, pulmonary contusion, neoplasia). Pulmonary blebs are only rarely visible.

Management

If the animal has an open pneumothorax, it is important to immediately cover the open thoracic wound with a sterile pad, and thus convert the chest to a closed system. Needle thoracocentesis can then be performed to evacuate air from the chest. Oxygen supplementation may be required, particularly if there is underlying pulmonary injury. The patient should be monitored carefully, to assess for recurrence of the pneumothorax. Needle thoracocentesis may need to be repeated if there is continual accumulation of air.

Placement of a chest drain should be considered if more than two thoracocenteses are required within a 24-hour period, or if air rapidly reaccumulates within the chest. Continuous drainage may be indicated for animals with large traumatic defects, though this is very rarely necessary. This may enable quicker resolution of the injury as the chest is kept inflated, and an effective fibrin seal can form without being regularly displaced. Heimlich valves may be considered as an alternative to continuous drainage in some animals. However, they should only be used in medium to large breed dogs. Smaller animals are unable to generate sufficient expiratory pressure. The animal must also be monitored constantly, as the presence of fluid within the valve chamber will completely negate their function.

Surgical intervention is rarely required in animals with traumatic pneumothorax, and spontaneous resolution is the norm. Intermittent drainage may be required for 3-5 days, while the pulmonary injury heals.

Conversely, spontaneous pneumothorax rarely resolves without surgery. In two large studies, the pneumothorax in over 80% of animals did not respond to a combination of needle thoracocentesis or thoracic drainage, and surgical intervention was required. Surgical management was found to provide a lower recurrence rate than conservative care, the animals had a short hospital stay. However, surgical management is not straightforward. A large proportion of animals (75%) will have bilateral pulmonary disease, and this may necessitate the need for multiple complete or partial lobe resections. Exploration of the chest via a median sternotomy is recommended due to this high rate of bilateral involvement. The use of stapling equipment can greatly facilitate lung resection, which can otherwise be difficult from this approach.

A mortality rate of up to 30% is described for animals with spontaneous pneumothorax following medical or surgical therapy. Recurrence in the surviving animals may occur immediately many months after apparently successful management. Some authors also advocate the use of pleural abrasion intra-operatively in order to cause pleurodesis. They suggest that this may prevent recurrence of pneumothorax in animals with generalised pulmonary involvement.

Diaphragmatic Hernia

Introduction

Diaphragmatic hernia is a relatively common condition in dogs and cats that usually (~ 85% cases) occurs following blunt trauma such road traffic accidents, kicks, falls or fights. Although most animals present almost immediately with signs referable to the traumatic incident, a proportion of animals (~ 20%) will present many weeks to months after injury, despite having been apparently normal during the intervening period. Congenital forms of the disease also occur (~ 5-10%), but many of these animals will die immediately after birth due to severe respiratory compromise.

Pathogenesis

Diaphragmatic rupture normally results from indirect injury to the diaphragm. Blunt trauma produces a sudden increase in intra-abdominal pressure with the glottis open. This results in a significant elevation of the pleuro-peritoneal pressure gradient, rupture of the diaphragm and herniation of abdominal contents. Although direct injury is a common cause in people that sustain a stab wound, this is an uncommon mechanism in animals but may occur following inadvertent extension of a coeliotomy or sternotomy wound or incorrect placement of a thoracostomy tube.

Herniation of abdominal contents into the pleural space results is loss of sub-atmospheric pleural pressure thus equalising the pleural and peritoneal pressures. Diaphragmatic function is compromised therefore abdominal and thoracic wall muscles must take over its function. Pain and mechanical factors associated with concurrent rib fractures, flail chest and body wall contusions limit their ability to perform this with resultant hypoventilation. Atelectasis of lung lobes by compression from herniated organs results in ventilation:perfusion mismatch and hypoxia. This can further be compounded if there is shock and hypovolaemia reducing effective tissue perfusion.

Signalment

Because road traffic accidents constitute the most common cause of traumatic diaphragmatic hernia, animals that typically present with the condition reflects a group with a tendency to roam and a relative naivety of traffic i.e. young, adult male dogs or cats. However, a wide variety of breeds and ages can be affected, and diaphragmatic hernia should be considered in all animals that present with acute respiratory distress.

Presentation and Clinical Signs

Animals with traumatic diaphragmatic hernia are usually presented after a traumatic incident. Cardiovascular compromise, due to shock, blood loss and pain, is the most significant concern and requires immediate attention. Assessment of mucous membrane colour, pulse rate, rhythm and pressure, and respiratory status should occur immediately. The

violence of the trauma suggests that other soft tissue injuries may be present, including liver lobe laceration, bladder rupture, myocardial or pulmonary contusions. Respiratory signs predominate with dyspnoea being reported in approximately 40 % of patients. Affected animals tend to have a restrictive breathing pattern typified by rapid shallow breaths. They may adopt a sitting or standing position with the head and neck extended and elbows abducted. Other aspects of the physical examination that may raise an index of suspicion for a diaphragmatic hernia include absent or muffled heart sounds, distortion of the apex beat, boborygmus audible on thoracic auscultation (infrequent and unreliable finding), and a 'wasp waist' appearance due to absence of abdominal contents.

Most animals will tolerate disruption of the diaphragm without significant respiratory distress. The occurrence of progressive dyspnoea or cyanosis suggests the presence of significant concurrent injuries that are compromising the animal's ability to compensate adequately. Concurrent injuries that require attention include myocardial or pulmonary contusions, fractured ribs, flail chest and pain. Provision of intravenous fluids, colloids or hypertonic saline to stabilise the cardiovascular parameters, in combination with oxygen therapy and pain relief should be the first priority.

Animals with more chronic traumatic diaphragmatic hernia, or congenital peritoneopericardial diaphragmatic hernia, may present with a wider variety of clinical signs, which may reflect a degree of respiratory compromise (dyspnoea, exercise intolerance), or may be attributable to the herniated organ (e.g. chronic vomiting, hepatic disease, ascites). The clinical status can deteriorate quite acutely in some of these animals, and will require an astute clinician to identify the cause (e.g. a liver lobe that becomes acutely entrapped in an old diaphragmatic rent, leading to significant pleural effusion and respiratory compromise).

Diagnosis

Definitive diagnosis of traumatic pleuroperitoneal diaphragmatic hernia requires radiography or ultrasonography. Radiography should be performed in a way that minimises stress to the patient to prevent de-compensation. The first radiograph should be a dorso-ventral view to establish the affected side and determine which lateral radiograph should be obtained (i.e. the animal should only be positioned with the affected side down). Consideration should also be given to horizontal beam radiography if required safety measures are in place. Reliable interpretation of radiographs is often compromised if significant pleural effusion is present, thus obscuring soft tissue detail. In this instance, thoracocentesis may be necessary. Alternatively, ultrasound examination provides excellent visualisation of the diaphragmatic line and the pleural cavity, and is particularly helpful for animals with very subtle herniations (e.g. partial hepatic herniation only). The presence of

fluid in the thorax will considerably improve the acoustic resolution of the ultrasound images. Radiographic signs of diaphragmatic hernia include loss of the diaphragmatic line, loss of the cardiac silhouette, presence of gas-filled viscus within the thoracic cavity, and cranial displacement of the gastric fundus or splenic shadow.

Abnormalities in laboratory tests with diaphragmatic hernia usually reflect compromise of the displaced organs, and may include increases in liver, biliary or pancreatic enzyme activity. The fluid obtained by thoracocentesis is usually serosanguineous, and analysis is consistent with a modified transudate.

Stabilisation and repair

Pre-operative stabilisation

Immediate surgical repair of the acutely ruptured diaphragm is usually contraindicated, unless there is life-threatening haemorrhage from the liver or other vessels, or the stomach is herniated and distended in the thoracic cavity.

Mortality rates for surgery performed within the first 24 hours are significantly higher than if the animal is adequately stabilised first. Correction of cardiovascular shock with adequate intravenous fluid resuscitation is therefore the main priority.

If the animal is dyspnoeic, immediate oxygen supplementation via a face mask, oxygen cage or nasal tube should be provided and attention given to the cause of the dyspnoea. Sitting the animal in sternal recumbency may improve pulmonary dynamics. Significant pleural effusion should be drained by thoracocentesis.

Pain from abdominal or thoracic bruising, rib fractures or other orthopaedic injuries should be managed with effective opiate analgesia and splintage.

Pulmonary contusions will require careful titration of intravenous fluids (to prevent worsening of pulmonary oedema), oxygen supplementation, antibiotics and analgesia. Severe contusions may require ventilatory support if significant ventilation:perfusion deficits are present.

Arrhythmias due to myocardial contusion and ischaemia are a significant concern, are seen in ~12 % of patients, and can be associated with a higher mortality. They are frequently refractory to most anti-arrhythmic drugs; ideal treatment consists of oxygen supplementation, analgesia and fluid support.

Continuous monitoring of the patient between the time of diagnosis and definitive surgical treatment is essential because acute de-compensation may occur.

Anaesthesia considerations

A variety of anaesthetic agents may be used to induce anaesthesia in these patients. However, doses used in the trauma patient are likely to be significantly reduced. A smooth intravenous induction and rapid endotracheal intubation should be the objective of any protocol employed; mask induction should be avoided in animals with respiratory compromise. Providing oxygen supplementation prior to induction will improve myocardial oxygenation and minimise compromise during induction and intubation. Maintenance of anaesthesia is performed with an inhalation agent such as halothane (or isoflurane if myocardial arrhythmias are present). Nitrous oxide should not be used.

Surgical access for repair of the diaphragmatic hernia is best achieved via a ventral midline cranial coeliotomy. Extension of the incision into a median sternotomy may be useful in some cases where extensive herniation, organ entrapment or adhesions exist. Patient preparation must be performed taking this possibility into account. Careful monitoring of the animal is essential when the patient is placed into ventral recumbency, as significant respiratory compromise may be seen. Intermittent positive pressure ventilation should be performed if respiratory difficulties are seen.

As soon as the abdominal cavity is entered, anaesthetic management for an ‘open chest’ should be started. Controlled ventilation via intermittent positive pressure ventilation is obligatory, and will require a dedicated anaesthetic assistant. Good observation and communication between the surgeon and anaesthetist will prevent excessive over-inflation of the lungs (which may lead to expansion pulmonary oedema in chronic cases), and will allow the surgeon to perform manipulations (e.g. dissection of adhesions, suturing) within the thoracic cavity between inspiration.

Once the diaphragm is closed, ‘closed chest’ respiratory dynamics may resume and the animal may start to ventilate for itself, with assistance (by IPPV) if required. Depending on the chronicity of the case, it is usually not necessary to evacuate all of the air from the chest immediately after surgery. Indeed, it may be unsafe to do so, with re-expansion pulmonary oedema being a significant concern. Placement of a temporary chest drain will allow intermittent thoracocentesis in the post-operative period. Criteria for ‘how much air to remove’ are vague; I typically rely on pulse oximetry and ventilatory effort to gauge progress. Careful monitoring of mucous membrane colour, respiratory effort and rate in the post-operative period is essential. Oxygen supplementation via a nasal oxygen catheter should be considered in all cases. Provision of effective pain relief is also important.

Surgical management

Surgical management of most diaphragmatic hernias is relatively straightforward. However, complications can occur if inappropriate care is taken when repositioning organs, or when reconstructing the defect in the diaphragm.

A midline cranial coeliotomy is the preferred technique for the repair of most diaphragmatic hernias. This approach can be extended into the sternum if necessary to maximise the exposure. Resection of the falciform ligament and associated adipose tissue and use of a self retaining retractor such as a Balfour significantly improves visualisation and access.

Every care should be taken during replacement of herniated organs into the abdominal cavity. The liver is the organ most frequently herniated with the remainder of the content depending upon the size of the defect coupled with the proximity and mobility of other organs. Careful assessment for injury or tissue ischaemia that may have occurred as a result of the traumatic incident or though vascular compromise as a result of incarceration should be made. Damaged and displaced parenchymal organs such as the liver and spleen are often considerably more friable than normal, and have a tendency to fragment and bleed during handling. Adhesions between abdominal and thoracic organs may be a significant concern, particularly in hernias that are more than a few days old. Early fibrous adhesions (3-8 days) can usually be broken down easily with gentle manipulation. More chronic adhesions require greater care, as damage to pleural surfaces may occur, resulting in pneumothorax or haemorrhage. Careful sharp dissection of the adhesions should be performed wherever possible. If the adhesion is too large, then a partial lobectomy of lung from the adherent organ should be considered. It is possible to enlarge the hernial rent with a radial incision directed ventrally (away from vena cava and phrenic nerves) to facilitate atraumatic reduction.

> Major care is needed in the anaesthetic and surgical management of the patient with a chronic diaphragmatic hernia or peritoneopericardial hernia as we have encountered a number of patients where the replacement of chronically entrapped organs (in particular the liver) leads to sudden and disastrous de-compensation. This appears to be due to the sudden outflow of toxic metabolites from the entrapped liver entering the circulation, lead to sudden massive drop in blood pressure, due to massive peripheral vasodilation. Very close monitoring and rapid intervention with adrenaline to reverse these changes by trained anaesthetic staff is essential to reverse this otherwise fatal occurrence.

Assessment of the diaphragmatic rent can usually be performed once all of the organs have been replaced in the abdominal cavity. Most tears involve the costal muscles with central tendon and crural muscle tears being uncommon. The diaphragm may rupture circumferentially (40%) or radially (40%), or may be a combination of the two. The distribution of right and left sided tears is equal but is important to note that there may be multiple tears in up to 15% of cases making through examination of the diaphragm essential. Access to the diaphragm is facilitated by wrapping abdominal contents in moistened laparotomy swabs and by gently retracting the liver with protected ribbon / malleable retractors. Suturing should begin in the dorsal recesses of the wound, and advance ventrally. Care is taken to avoid placement of sutures close to the vena cava which could compromise vessel integrity or reduce venous return. Judicious placement of one or two stay sutures can be useful in defects with complex shapes. The diaphragm can be repaired with a simple continuous or interrupted pattern using an absorbable material with a prolonged duration of tensile strength retention (e.g. polydiaxinone) or alternatively a monofilament non-absorbable material. Continuous patterns are ideal because it minimises the number of sharp suture ears that may irritate local viscera and is rapidly placed. At all times, ensure suture bites are adequate to minimise the potential for dehiscence. Resection of scarred margins should be avoided as it increases the size of the defect, and risks dehiscence of the repair through excessive tension. If the diaphragm has torn circumferentially from the ribs, incorporate a rib in the repair for additional strength.

The use of synthetic (e.g. polypropylene mesh) or autogenous materials (e.g. omental flap) is rarely required but may be considered if the defect is too large to close primarily, or there is significant tension on the repair. This may occur particularly in chronic traumatic cases due to induration of the wound edges, or with large congenital peritoneopericardial diaphragmatic hernias. Omentum should always be interposed between the pleural cavity and mesh to prevent lung trauma. The use of a vascularised muscular pedicle graft from the transverse rectus abdominal muscle has also been reported.

Various options are available to evacuate free air from the pleural space. A thoracostomy tube may be placed under direct visualisation prior to diaphragmatic repair or alternatively an infant feeding tube may be placed through the diaphragm and exited through the abdominal wall to permit short-term post-operative drainage. Finally in cases in which further management of the pleural space is not anticipated in the post-operative period, simple needle thoracocentesis may be performed through the diaphragm following closure. Successful evacuation of the pleural space of free air will be assured when the diaphragm changes from a flaccid structure to a taught concave shape. It is **not** appropriate to use increased lung ventilation / expansion as the diaphragm is closed as a method of evacuating free air as this seriously risks iatrogenic damage to the lung parenchyma.

Closure of the abdominal wound may also be a problem in animals where the herniation has been long-standing and contraction or atrophy of the abdominal muscles may reduce

abdominal domain. This can lead to difficulty in closing the linea alba when all organs have been re-positioned. Abdominal closure needs to be performed with extreme care to ensure that the repair is sufficiently strong to tolerate the transiently increased tension on the wound, and to prevent inappropriate entrapment of viscera in the suture line. These animals may experience greater post-operative levels of discomfort which needs to be considered in the analgesic plan.

Post-operative care

Continuous monitoring of vital signs (pulse rate, respiratory rate and pattern, mucous membrane colour, capillary refill time and temperature) is essential. Objective measures such as oxygen saturation, blood pressure and ECG are very useful additions to the clinical assessments and can aid decision making. Oxygen therapy should be considered especially if there is ongoing dyspnoea or if oxygen saturation falls below 90%. Judicious and aseptic thoracostomy tube management is important if one is placed. Care must be taken to gently and gradually evacuate the pleural space in chronic hernias with long standing atelectasis to avoid re-expansion pulmonary oedema. Multimodal analgesia is an important part of the post-operative care plan and usually involves systemic opiates, a NSAID (if appropriate), intercostal nerve blocks (performed at the end of surgery) and possibly intra-pleural local anaesthesia.

Prognosis

Successful management of the animal with a traumatic diaphragmatic hernia requires application of a variety of resuscitative, diagnostic and peri-operative skills. The prognosis for recovery is good, but reported mortality rates range from 12-48%. Mortality rates are higher for animals operated within the first 24 hours or over a year after injury. With appropriate care, however, good success should be expected in more than 75% of animals. Possible complications include: pneumothorax, haemothorax and continued pleural effusion due to injury or damage to intra-cavitary organs (lungs/liver/spleen) that was not recognised at the time of surgery; infection; re-expansion pulmonary oedema; and re-herniation due to dehiscence of the diaphragm wound. Re-herniation has been reported up to 5 months after the original repair and is in the order of 5% in one study.

Epiglottic retroversion

Epiglottic retroversion is a rare cause of upper airway obstruction in dogs with only a few cases reported. The disorder can result in inspiratory stridor and life threatening dyspnoea. It is characterised by episodic epiglottic retroflexion during inspiration and obstruction of the rima glottides. The more stressed the patient becomes the greater the degree of retroversion and the more severe the dyspnoea. The condition therefore develops into a vicious circle. Repeated obstructive episodes may lead to upper airway swelling and

pulmonary oedema. Temporary epiglottopexy was deemed to be an appropriate first step in the management of these patients based on two reported cases (Flanders et al. 2009). Temporary epiglottopexy has the advantage of being reversible should postoperative complications occur. If well tolerated it can be converted to a permanent fixation after 24 hours (Flanders et al. 2009).

This has not been universally successful in our hands, and indeed failure of the 'permanent' epiglottopexy over time, has anecdotally been reported by others. In all cases the epiglottopexy wound has healed but there has been stretching of surrounding mucosa, permitting recurrent retroversion. It is suspected that the sutures may also be at risk of pulling through the mucosa due to repetitive tensile forces, following excessive panting during the postoperative period.

Having failed to achieve a good outcome after two attempts in our first case, permanent fixation was not considered possible. The patient was not considered a good candidate for permanent tracheostomy given his temperament. Subtotal epiglottectomy was thus performed as a salvage procedure. There was concern that epiglottectomy would risk dysphagia or aspiration during eating. This did not occur and has not been reported following experimental epiglottectomy in cats, nor following epiglottectomy in humans.

We do not yet understand the aetiology of this condition. Interestingly, the two previously reported cases both had hypothyroidism and one additionally had epilepsy (Flanders et al. 2009). Our patient did not have thyroid function testing performed due to financial limitations and lack of history to support underlying endocrine disease. It is also speculated that the primary cause could relate to a myopathy affecting the hyoepiglotticus muscles or neuropathy involving the hypoglossal or glossopharyngeal nerves but this remains a theory. It would likely require electromyography of the hyoepiglotticus muscles to investigate this further which would be no simple task.

Total epiglottectomy, most commonly using a carbon dioxide laser, is performed in humans for treatment of supraglottic malignancies and severe cases of laryngomalacia. To the authors' knowledge our case was the first peer-reviewed report of subtotal epiglottectomy in the dog. No postoperative complications were observed in this patient. Following the successful outcome in this patient, subtotal epiglottectomy may be considered a reasonable treatment option for management of severe cases of ER, particularly those that recur following epiglottopexy.