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Focus on Emergency Surgery Mini Series

Session Three: A to Z of Respiratory Emergencies

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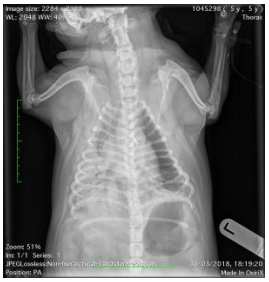

A to Z of emergency respiratory surgery

Introduction:

The first webinar in this series addressed the triage and management of commonly encountered traumatic injuries. Today's webinar will look at management of some of the more commonly encountered conditions affecting the respiratory system which require surgical management or are associated with surgical conditions.

A is for aspiration pneumonia

Aspiration pneumonia is not infrequently seen in patients with either brachycephalic obstructive airway syndrome or laryngeal paralysis. The severity of the inflammation caused by aspiration depends on both the composition and volume of the aspirate. The inhalation of irritants such as oropharyngeal or gastric contents into the airways can cause lung injury and is referred to as aspiration pneumonitis. Pulmonary damage may result from exposure to gastric fluid with a pH of 2.5, aspiration volume, gastric fluid containing colloidal antacid compounds, and large particulate matter. Gastric acid-mediated injury is initially an aseptic injury, but eventually reduces airway defence mechanisms, predisposing the lung to secondary infection via bacterial colonization. Changes in surfactant also lead to increased surface tension and predisposes to atelectasis. The term aspiration pneumonia (AP) refers to the development of a presumed secondary infection after the initial injury.

The main differential for aspiration pneumonia in patients with respiratory distress is non cardiogenic pulmonary oedema or acute respiratory distress syndrome (ARDS) or leads to loss of endothelial integrity. The reason for the loss of vascular and alveolar integrity has not been fully defined. The pathogenesis of ALI/ARDS is extremely complex and involves numerous inflammatory mediators as well as cellular responses. Loss of integrity leads to the uncontrolled flow of fluid and protein into the interstitial space, exceeding the capacity of the lymphatic system. Therefore, fluid begins to accumulate in the pulmonary interstitium and the peribronchovascular cuffs. There is also damage to the alveolar epithelium, and fluid and proteins leak from the interstitium into the alveoli, causing alveolar flooding.

Clinical signs include cough, lethargy, hyporexia and tachypnoea. Some patients may be pyrexic. Diagnosis is usually made based on a combination of clinical suspicion, clinical signs and radiographic findings. Aspiration usually affects the most gravity dependent lobes – right middle lung lobe followed by the right and left cranial lung lobes. Radiographic findings include interstitial to an alveolar pattern within the affected lobe or lobes. This is in comparison to the more diffuse alveolar pattern seen in patients with ARDS.

Treatment is directed towards supportive care. If the reflux/ aspiration event is witnessed, an attempt can be made to suction the pharynx and cervical trachea but often this is not feasible. Treatment includes:

- Provide oxygen supplementation
- Gastroprotectants
- Inhaled bronchodilators e.g. salbutamol.
- Nebulisation with hypertonic saline and adrenaline
- Physiotherapy
- Anti-microbial therapy
- Severely affected cases can require mechanical ventilation for a period of days. This can be very costly and usually requires referral to a specialist centre.

B is for brachycephalic obstructive airway syndrome

Brachycephalic obstructive airway syndrome is an umbrella term for a number of anatomical abnormalities affecting the skull and tissues of the upper respiratory tract which result in an increased resistance to airflow. Whilst we tend to "lump" all brachycephalic dogs under the same heading, there are a number of different abnormalities which can be identified and not all dogs will have the same combination of abnormalities. Thus, not all brachycephalic dogs present with the same type or severity of clinical signs.

Abnormalities are spilt in to primary and secondary abnormalities. Primary abnormalities are those which are identified early in the course of the disease process and may be present before significant clinical signs are identified. These include stenotic nares, aberrant nasal turbinates and increased nasal mucosal contact points, elongation and thickening of the soft palate, macroglossia, small cross-sectional area of the rima glottis and tracheal hypoplasia. Secondary changes occur as a consequence of long-standing increases in inspiratory pressures and include laryngeal collapse (including eversion of the laryngeal saccules) and eversion of the tonsils.

History and clinical signs:

Broadly speaking, there are three main time points where these dogs are presented for assessment:

1) In dogs of less than six months of age. Usually these dogs have either significant regurgitation or tracheal hypoplasia.
2) In dogs of six months of age to several years of age.
3) In older dogs (over five years of age) which can present with more advanced laryngeal collapse.

It has been demonstrated by several studies that owner perceptions of the severity of their pet's clinical signs, and the effect on quality of life of those pets, can be unreliable (Packer, O'Neill et al. 2019). This can be further exacerbated by the very strong pet-owner bond seen in these breeds. However, not all brachycephalic dogs are affected by BOAS and thus each dog must be assessed on a case by case basis.

Clinical signs in dogs affected by BOAS are well documented. These include inspiratory stertor, exercise intolerance, heat intolerance, sleep apnoea/derangement, regurgitation, gastro-oesophageal reflux, syncope and cyanosis. Stridor is often documented in cases where there is significant laryngeal collapse. It is important to take a thorough history as it has been documented that some owners may consider some abnormalities (e.g. regurgitation or reduced exercise tolerance) to be normal for the breed.

Diagnosis:

The diagnosis of BOAS is usually made based on a combination of history, clinical signs and examination. These are, however, all to some degree subjective and we, as a profession, have struggled with identifying an objective and non-invasive method for determining both which dogs require surgical intervention and also for assessing response to surgery. In whole-body barometric plethysmography (WBBP), the dog resides in a Perspex chamber and respiration causes barometric pressure oscillations proportional to tidal volume. Flow traces have been shown to differ between both non-brachycephalic and brachycephalic dogs and also between clinically affected and non-clinically affected brachycephalic dogs (Liu, Adams et al. 2016).

Due to the need for specialised equipment and the time it takes to accurately collect such information, WBBP is currently used as a research tool in most institutes. However, the data collected has been used to validate a three-minute exercise tolerance test which can be performed in the clinic setting. The dog is auscultated before and after being exercised at a moderate trot for three minutes. If no noise and mild stertor is auscultated, the dog is considered clinically unaffected (mild is only audible with a stethoscope). If moderate and severe noise is auscultated, the dog is deemed to be clinically affected. Obviously sensible precautions must be taken based on the severity of the dog's clinical signs and the ambient temperature as to whether it is deemed appropriate to perform such a test.

The sensitivity of clinical examination alone for BOAS diagnosis is reported to be 56.7% pre-exercise test, 70% after a 5-minute walk test and 93.3% after a 3-minute trot test (Riggs, Liu et al. 2019). The sensitivity of using the presence of laryngeal stridor as a predictor of laryngeal collapse was improved after an exercise test (70%) compared with before the exercise test (60%). Specificity of laryngeal stridor for laryngeal collapse was 100% (pre-exercise and post-exercise).

Physical examination will permit assessment of nares diameter but will not allow assessment of the length of the soft palate nor the stage of laryngeal collapse. This must be performed under general anaesthesia. Direct laryngoscopy requires a good light source. When assessing the length of the soft palate, the tongue must be in a neutral position and not pulled rostrally (as this will artefactually increase soft palate length). The tip of the epiglottis should touch the caudal most aspect of the soft palate.

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

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

In humans, the function of the saccule is unknown but is thought to be involved in lubrication of the vocal folds. At this time, this author recommends removal of the laryngeal saccules if everted although a recent publication by Hughes (Hughes, Kaye et al. 2018) reported an increased post-operative complication rate in BOAS dogs post-surgery where laryngeal saccules were removed.

The role of imaging:

The use of both radiography and computed tomography (CT) is reported in the investigation of BOAS. Thoracic radiography is usually performed both to allow assessment of tracheal diameter and also to look for evidence of aspiration pneumonia by the identification of air bronchograms (figure five). This may be subclinical in some dogs. A ratio based on the tracheal diameter at the level of the thoracic inlet and the depth of the thoracic inlet is reported but the association between the severity of presenting clinical signs and the ratio are not clear cut. There is also poor agreement in the ratio calculated depending on measuring method, cut-off value and observer. This is likely due to the fact that the majority of dogs presenting with BOAS have more than one anatomical abnormality and thus it is hard to isolate the effect of one specific abnormality. Tracheal hypoplasia in some brachycephalic dogs might partially or completely resolve with growth to mature body size (Clarke et al 2011).

Grand (Grand and Bureau 2011) reported that dogs with severe brachycephalic airway syndrome had significantly thicker soft palates compared to absent/minimal brachycephalic airway syndrome and control dogs when assessed using CT. A extubated lateral view of the skull can be performed to allow for subjective assessment of soft palate thickness if CT is not available.

CT can also be used to assess the skull and nasal cavity in brachycephalic patients (figure seven). Heindenreich (Heidenreich, Gradner et al. 2016) reported that the smallest nasopharyngeal cross-sectional area is located dorsal to the caudal end of the soft palate in both Pugs and French Bulldogs. Using CT of the skull, Oechtering (Oechtering, Pohl et al. 2016) reported that rostral aberrant turbinates were common in Pugs (90.9%) but less frequent in French (56.4%) and English (36.4%) Bulldogs. Caudal aberrant turbinates obstructing the nasopharyngeal meatus were commonly found in all breeds (66.7%).

Surgical management:

There remains no definitive answer as to the best surgical approach for these patients. Weight loss can have a very positive effect on these dogs and can be recommended prior to surgery if the severity of clinical signs allows for such a delay. The surgical literature is predominantly retrospective and for the most part utilises subjective outcome measures. Despite the fact that the combination of abnormalities identified in each individual dog varies, the majority of dogs undergo similar surgical intervention:

1) **Alarplasty**

2) **Palatoplasty** – two main techniques exist for palatoplasty. Traditional palatoplasty involves a stepwise "cut and sew" technique. Use of a CO2 laser and bipolar sealant devices has also been described in the veterinary literature. Folded flap palatoplasty was described by Findji et al in 2008 (Findji and Dupré 2008). Objective comparison of the two techniques has not been performed and therefore one technique cannot be truly said to be "better" than the other based on the current evidence.

3) **Tonsillectomy** – most commonly performed using a harmonic scalpel or a bipolar sealant device.

4) **Laryngeal sacculectomy**

5) **Laser-assisted turbinectomy (LATE)** – Removal of the obstructing parts of the chonchae using a diode laser is described (Oechtering, Pohl et al. 2016). Liu (Liu, Genain et al. 2019) reported use of WBBP as part of an established protocol to assess the effectiveness of LATE in dogs which had already undergone conventional multi-level surgery (CMS). The median BOAS index of dogs that were operated on decreased from 67% post-CMS to 42% after LATE. Regrowth of turbinates requiring further LATE surgery is reported in 15% of dogs (Oechtering, Pohl et al. 2016)

6) **Management of higher grades of laryngeal collapse –** partial arytenoidectomy and unilateral arytenoid lateralisation have both been reported.

7) **Permanent tracheostomy** is reported as a salvage procedure for dogs where other surgical techniques have failed. Major complications were reported in 80% of dogs in a recent small case series.

Post-operative management:

Recovery of brachycephalic dogs from anaesthesia and surgical intervention can be very challenging and infrequently, dogs can die. A slow and controlled recovery is much preferred and intubation should be maintained for as long as it is tolerated. Once extubated, try to keep the head elevated on a sandbag etc in case of gastro-oesophageal reflux. The period between extubation and full consciousness is often the most difficult period, particularly if airflow through the nasal cavity is reduced (either due to blood clots or due to turbinate conformation). Keeping the oral cavity propped open, either physically by holding the mouth slightly open or by placement of a roll of tape in to the mouth, may improve airflow particularly in dogs with significant macroglossia if it is tolerated. Nebulisation using adrenaline (diluted in saline) can be utilised to reduce oedema in dogs which are demonstrating increased inspiratory effort on recovery.

The author generally avoids the use of non-steroidal anti-inflammatories in the post-operative period as the administration of steroids is often required but opinion does vary on this. Furthermore, some surgeons give steroids routinely whilst the author prefers to administer steroids on an as required basis. Opioids are administered following surgery based on the Glasgow pain scale. Sedation is often useful post-operatively; given with the aim of preventing patient anxiety which can lead to the development of a deleterious respiratory pattern. Dexmedetomidine is the drug of choice in the author's institute; either as an intravenous bolus or more commonly as a constant rate infusion.

Acepromazine can also be used. Food and water are usually withheld for 12-24 hours post-operatively and when food is re-introduced, small balls of moist food are offered by hand under supervision. The author routinely administers omeprazole for 14 days post-operatively unless side effects (vomiting and diarrhoea) are encountered. Eye lubricant is applied frequently.

Complications of surgery include haemorrhage, infection and wound healing complications (particularly the nares). However, the three most challenging complications we face when dealing with BOAS patients are the development of pharyngeal/laryngeal swelling, aspiration pneumonia and persistent oesophagitis. Temporary tracheostomy tube placement is required in 5-10% of dogs undergoing conventional multi-level surgery and subjectively, this rate may be higher if the surgeon is inexperienced as surgery time is likely to be increased, as is tissue handling. If temporary tracheostomy is required, it is usually within the first 24 hours of surgery and therefore it is NOT recommended that dogs be discharged the same day as surgery unless there are specific extenuating circumstances.

Care of temporary tracheostomy tubes is time consuming and requires an experienced team. Obstruction of the tube in these dogs can rapidly lead to death and thus 24-hour observation is required. The face and tracheostomy site are nebulised every four hours using either diluted adrenaline or saline. The lumen of the tracheostomy tube should be suctioned using a sterile urinary catheter based on clinical assessment. The author routinely changes the tube once or twice daily. Placement of stay sutures (figure 10) (the ends of which should be labelled UP and DOWN once the tube has been placed) will greatly facilitate the ease with which the tube can be changed in the conscious patient.

Fenner (Fenner, Quinn et al.) reported that corrective surgery for BOAS was associated with a marked incidence of postoperative regurgitation. Younger dogs and those with a history of regurgitation were predisposed to postoperative regurgitation. Regurgitation can predispose dogs to the development of aspiration pneumonia.

Outcome:

It is important to remember that whilst surgery can alleviate some of the clinical signs associated with BOAS and improve quality of life, surgery cannot restore normal upper respiratory anatomy in these dogs and owners should be made aware of this.

L is for laryngeal paralysis:

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

The larynx has three main functions:

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

2) Decrease airway resistance by abduction of the arytenoid cartilages

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

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

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

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

Thoracic radiographs (to look for signs of aspiration pneumonia or a cranial mediastinal mass) +/- an extubated cervical lateral projection should be performed under anaesthesia. Surgery aims to widen the rima glottis to reduce resistance to airflow on inspiration. Unilateral arytenoid lateralisation is currently the treatment of choice in the UK. Sutures are placed between the muscular process of the arytenoid cartilage and the caudal aspect of the cricoid cartilage via a lateral approach to the larynx to abduct the arytenoid cartilage laterally thus increasing the diameter of the rima glottis. Other treatments e.g. trans-oral partial laryngectomy are also reported but are very uncommon in the UK.

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

L is for lung lobe torsion

Lung lobe torsion is an uncommon condition that has been reported in dogs and cats. Large breed dogs with deep, narrow chests have a higher incidence of lung torsion and a tendency for the torsion to occur in the right middle lung lobe or left cranial lobe. In these dogs, lung lobe torsion can be spontaneous or occur secondary to other conditions, such as chylothorax. In Pugs, the condition is thought to be spontaneous in origin and most commonly affects the left cranial lobe. The cause of the condition is poorly understood but may result from partial collapse of the lung lobe, permitting increased mobility.

Lung lobe torsion leads to venous, lymphatic, and bronchial obstruction in the face of continued arterial blood flow. This results in oedema, haemorrhage, and necrosis. Congestion and consolidation of lobe occurs as fluid moves into the interstitial tissue and airways. Pulmonary venous hypertension and decreased lymphatic drainage lead to haemorrhagic pleural effusion. A chylous effusion is sometimes seen.

Clinical signs include dyspnoea or tachypnoea, lethargy, and cough, occasionally with haemoptysis. Anorexia with weight loss and occasional vomiting can occur in long-standing cases (1 to 3 weeks). Physical examination may reveal pyrexia, pale mucous membranes, pain on abdominal palpation and decreased cardiopulmonary sounds regionally. Classic radiographic signs include a vesicular pattern within the affected lobe which may progress to an alveolar pattern/consolidation as time progresses. There is often a pleural effusion leading to pleural fissure lines.

Surgical management involves lung lobectomy via an intercostal thoracotomy. Surgical approach will be discussed in more detail during the webinar.

P is for pericardioperitoneal diaphragmatic hernia (PPDH)

PPDH occurs due to abnormal development of the transverse septum results either in a gap in the ventral portion of the diaphragm or unusually thin ventral diaphragmatic tissue that ruptures. The result is herniation of abdominal viscera into the pericardial sac. The defect is usually present at birth, although affected animals may remain asymptomatic and undiagnosed for many years. Traumatically acquired PPDH are rare in animals. Organs that frequently herniate into the pericardial sac include the liver, falciform ligament, omentum, spleen, small intestine, and rarely the stomach. PPDH may occur with other congenital abnormalities, including sternal defects, cranial midline abdominal wall hernia, umbilical hernia, abnormal swirling of the hair in the sternal region, intracardiac defects, and pulmonary vascular disease.

PPDH can be asymptomatic in up to 50% of cases. Patients can acutely deteriorate as with patients with diaphragmatic rupture if organs move through in to the pericardial sac. This can lead to cardiac tamponade. Clinical signs include:

Respiratory signs: dyspnoea, tachypnoea, coughing, or wheezing.

Gastrointestinal signs: anorexia, polyphagia, vomiting, or diarrhoea.

Nonspecific signs: weight loss, abdominal pain, ascites, exercise intolerance, shock, and collapse

The diagnosis can be made easily on plain radiography. The cardiac silhouette has a globoid appearance and gas shadows from the intestines may be seen within the pericardial sac.

Surgical management is via ventral celiotomy although if adhesions are present, a median sternotomy is rarely required. Post-operative management and potential complications are similar to that encountered during management of diaphragmatic rupture. Dissection of the pericardial sac may lead to damage to the phrenic nerve which, if damaged bilaterally, can lead to dyspnoea.

S is for spontaneous pneumothorax

In our previous webinar we discussed diagnosis and management of traumatic pneumothorax. These patients usually present with a history of acute onset dyspnoea but with no or a minimal history of trauma. Owners often report cough, retching and dyspnoea. Clinical signs include a restrictive (shallow) breathing pattern, hypoventilation and reduced lung sounds on auscultation.

The two most common aetiologies for the development of a spontaneous pneumothorax are rupture of a pulmonary bulla (or bleb) or a tracking pulmonary foreign body. Rupture of neoplastic lesions is infrequently encountered. Cystic and bullous lesions in the lungs are characterized by a thin-walled cavity within the lung parenchyma. Lung bullae and blebs (pseudocysts) are similar to cysts but have no epithelial lining. Bullae are large air spaces that develop within the lung parenchyma, and blebs are small accumulations of air between the parenchyma and visceral pleura. These cavities develop from traumatic rupture and coalescence of alveoli and are frequently secondary to obstructive lung disease.

A high clinical suspicion for pneumothorax can be made based on clinical signs but the diagnosis is usually confirmed on either plain radiography or using computed tomography (CT). Even CT has a relatively low sensitivity and specificity for quantification of the number of bullae, in particular that which has ruptured and is the source of the leak. The primary use of CT is to rule out other causes for the pneumothorax.

A thoracostomy tube (chest drain) should be placed under general anaesthesia (as discussed in our previous webinar) for drainage of the thorax. Repeating radiographs or CT following drainage of the thorax will increase the chances of identifying pathology. In humans, management of spontaneous pneumothorax is usually conservative in the first instance encompassing use of continuous thoracic drainage. However, the recurrence rate is reported to be high and a recurrence rate of up to 50% is reported when conservative management in employed. Surgical management is therefore usually recommended and involves median sternotomy to permit exploration of both sides of the thorax. The thorax is flooded with saline and each lung lobe, in turn, immersed in saline as the anaesthetist inflates the lung to 20mmgHg. Lesions within atelectic lobes may not leak and thus it is important to ensure the whole lung is fully inflated. Affected portions of the lung are resected, usually using a stapling device (thoracoabdominal or GIA). There is a limit as to how much lung can be removed. Acute restriction of more than 60% of pulmonary artery outflow is fatal in dogs because it can induce acute pulmonary hypertension. This finding correlates well with the observation that dogs can survive a 50% loss of total lung volume but will die after a 75% loss. Excision of an entire left lung is tolerated if the right lung is healthy because the left lung represents only 42% of the lung mass. Because the right lung is larger than the left, an acute right pneumonectomy removes more than 50% of the lung and is likely to be fatal.

Recurrence following surgery is reported to be 13% in the largest and most recent case series. This may either be due to development of further bullae or due to rupture of a previously unidentified bulla.

In case where pneumothorax is caused by an inhaled tracking foreign body, management is again via median sternotomy and surgical resection of the lesion.

T is for tracheal trauma

Tracheal trauma can occur secondary to RTA etc or can be iatrogenic due to endotracheal intubation, particularly in cats. Clinical signs may take several days to develop and can include dyspnoea, lethargy, anorexia and subcutaneous emphysema. The majority of tracheal tears will respond to conservative management (cage rest, oxygen supplementation and sedation). However, some cases will continue to deteriorate in the face of appropriate treatment and may require surgical intervention.

Tracheal avulsion is infrequently recognised and usually occurs due to blunt force trauma occurring when the neck is hyperextended. The most common location is close to the carina. Some cats may die at the time of the trauma but the most common time for presentation is two to three weeks post-surgery. These cases require complex surgical intervention and referral to a more experienced surgeon should be considered.

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