More Soft Tissue Surgery Case Challenges for Advanced Practitioners Mini Series

Session 1: Respiratory Distress

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In the first of the three webinars, I present some of the cases that I have seen this year. By working through the cases I will consider the problem-solving involved and present relevant facts. As in previous mini-series, I will present how I approached the case and made decisions and give my opinions, and these should be supported by independent learning of relevant book chapters.

Respiratory emergencies can be difficult to deal with, as both owners and animal will be stressed. Depending upon how critical the patient is the examination may need to be brief initially. It is helpful to take a few moments to look careful at the animal to assess the breathing pattern. In particular it is important to look for signs of upper respiratory tract obstruction, and determine if the animal is likely to need anaesthesia to maintain an airway. However, anaesthesia in a patient with another form of respiratory distress, e.g. pneumonia, may suffer negative consequences if anaesthetized, so it is important to differentiate them. Pleural disease can be recognized by a rapid shallow respiration and reduced heart and lung sounds (pleural fluid or air) or sometimes increased sounds (pleural air). Differentiation of these causes of respiratory distress is important as the animals will benefit from prompt thoracocentesis. Finally, animals with pulmonary or airway disease can be identified by listening for crackles and wheezes.

CASE 1 – pleural haemorrhage following an RTA

Triage

A 1-year-old Cocker spaniel had presented following a road traffic accident at 50mph. it is important to triage critical patients and to remember all body systems. Clearly it is important to prioritise the most life-threatening injuries first. The clinical findings listed below were those recorded at the referring veterinary practice. I have added my conclusions and how I would have treated the dog initially.

Respiratory:

- Physical examination: tachypnoea (180 breaths/minute), shallow breaths, reduced heart and lung sounds.
- Monitoring – pulse oximetry 70%.
- Conclusion – hypoxaemia, probable pleural fluid or air. History of RTA means that lung contusions are also likely to be present.
- Initial plan – oxygenate, thoracic ultrasound if quickly available, thoracocentesis (diagnostic and therapeutic tool)
- Further diagnostic tests – thoracic radiography and ultrasonography, arterial blood analysis if available

Cardiovascular:

- Physical examination: heart rate 126bpm, weak pulses, pale mucous membranes, slow CRT
- Monitoring - blood pressure not detectable
- Conclusion – hypovolaemia shock, cause not yet determined
- Initial plan – oxygenate, investigate cause of shock, intravenous access, fluid therapy, evaluate ECG (cardiac contusions can lead to arrhythmia)
- Further diagnostic tests – blood tests, thoracic and abdominal imaging, monitor ECG

Neurological:

- Physical examination: non-responsive, pupils fixed and responsive, no pupillary light response. No obvious head or vertebral trauma.
- Conclusion – could be neurological injury or due to hypovolaemia
- Initial plan – re-evaluate after treat hypovolaemia
- Further diagnostic tests – if becomes responsive, assess withdrawal and deep pain, spinal imaging if concerned

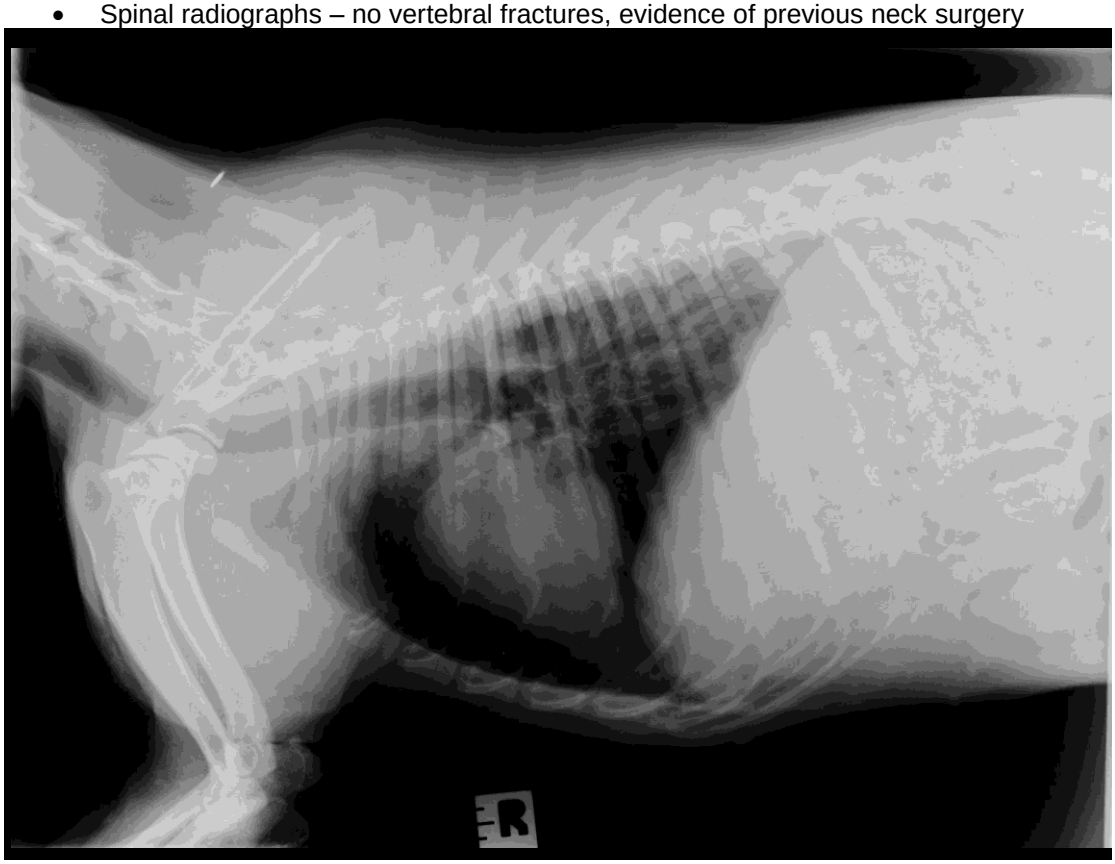
Rectal temperature: 39.0

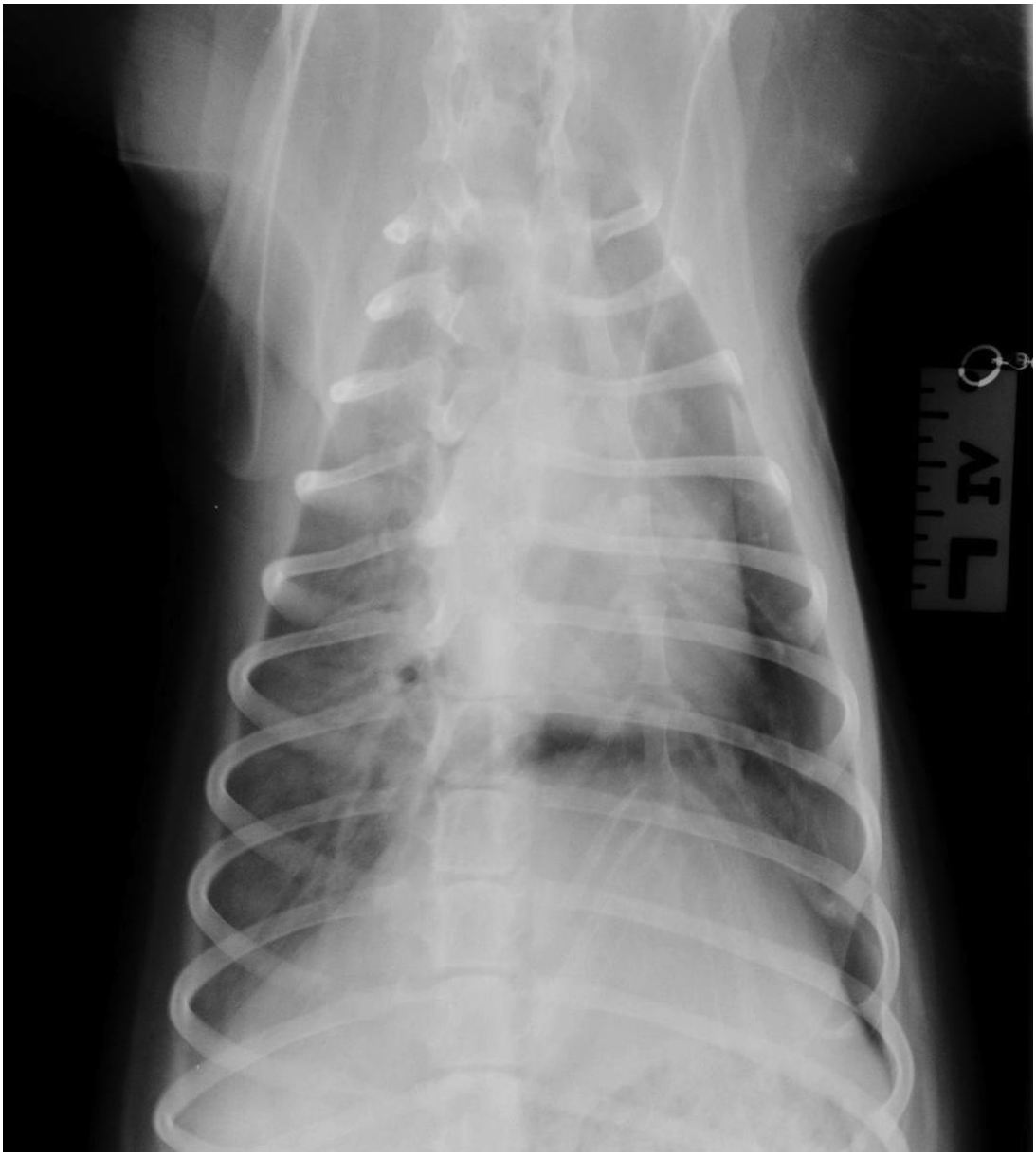
Orthopaedic: no obvious fractures

Skin: minor wounds.

Results of initial investigation

- Thoracocentesis was performed bilaterally and initially yielded 250 ml air and 370ml blood – diagnosis pleural haemorrhage and pneumothorax.
- Peripheral blood PCV 45%
- Thoracic radiography – pneumothorax
- Spinal radiographs – no vertebral fractures, evidence of previous neck surgery





A normal PCV shows that she was not anaemia prior to the injury. The hypoperfusion was likely to be hypovolaemic secondary to blood loss, although other types of shock may have contributed. Traumatic blood loss involves loss of all components of blood, and so PCV will be normal initially despite how much blood is lost. PCV will only fall when redistribution and dilution from fluid therapy occurs. It is therefore important to assess the volume of blood loss. Given an estimated blood volume of 1250ml after initial thoracocentesis of 370ml, she had lost an estimated 30% of blood volume. This is a significant volume and is sufficient to explain both cardiovascular and neurological parameters. After receiving crystalloids her PCV was 34%, but persistent hypovolaemia suggested that this value would drop significantly following restoration of vascular volume.

Triage after referral

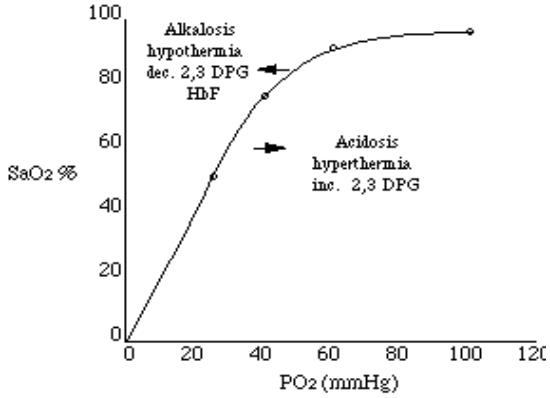
She was referred after having received some crystalloid fluid therapy and methadone analgesia. Concerns on presentation after triage were:

- Persistent marked hypovolaemia. Potential causes:
    - Further haemorrhage into the pleural space
    - Failure to provide sufficient volume fluid therapy
    - Failure to give appropriate fluids

- Decreased lung sounds – suspicious for recurrent pleural space disease. Repeat thoracocentesis – 110ml blood, total blood loss 40% blood volume
- Neurological status – she was conscious but had reduced mentation and no menace or PLR. She was weak on all four limbs but she had deep pain and withdrawal reflexes.

Oxygenation

Oxygen was delivered via a facemask. We give oxygen to most trauma patients. In this dog it was particularly important as ventilation is reduced by pleural space disease, VQ mismatch and reduced oxygenation can occur with pulmonary contusions and blood loss reduces oxygen-carrying capacity. Facemasks do not provide much increase in fractional inspired oxygen (FiO_2 0.35-0.55), especially if a seal is not present around the mask, but it is easy to provide as soon as a patient presents. Flow-by oxygen at > 100ml/kg/min flow rate is an alternative for animals that will not tolerate a facemask and can increase fractional inspired oxygen to 0.24-0.45. Consideration can be given to nasal oxygen delivery (FiO_2 0.40-0.60), although this can take some time to perform if a nasal catheter is to be placed. Oxygenation can be assessed in patients that will tolerate pulse oximetry, and probes can be placed on any mucosal surface. Note that pulse oximetry machines will not over-read so a result of 98-100% shows good oxygenation. A low signal can occur if the probe is not positioned well, so repositioning should be attempted. However a low reading in a dog that clinically is likely to be hypoxaemic should be considered accurate. Pulse oximetry does not give an assessment of ventilation. Pulse oximetry is most useful to show whether oxygenation is good or not. It is insensitive to early hypoxia and small decreases from the normal range are clinically relevant. As pulse oximetry uses a logarithmic curve to equate to partial pressure of oxygen, at a reading of 90-93%, arterial oxygen partial pressure (PaO_2) has already fallen to around 60mmHg. Beyond this, small decreases in SpO_2 equate to massive decreases in PaO_2, which are difficult to quantify, and it is impossible to know when the patient is developing significant hypoxaemia.



The oxygen dissociation curve. Note that at SaO_2 of 75%, PaO_2 is only 45%

Haemorrhage, hypovolaemia and fluid therapy

Typically I will address hypovolaemia first, assuming that the animal does not have life-threatening pleural space disease. In this dog, the volume of fluid in the pleural space was not excessive, and I would anticipate a dog would die of blood loss before dying of pleural space disease given that 30% of blood volume had already been removed from the thorax. Most cases of pleural haemorrhage have mild to moderate dyspnoea from the space occupying effects of the blood, and the risk of death is from effects of blood loss on the cardiovascular system. That said, pneumothorax concurrently with pleural haemorrhage is important to consider, and these patients can die of hypoxaemia due to reduced ventilation. Therefore I will perform diagnostic thoracocentesis before addressing hypovolaemia if this is more life threatening. In some instances, lung laceration can occur from rib fractures or blunt trauma, and pleural air accumulates very rapidly. In these cases, continuous thoracocentesis from one staff member may be necessary while other members of staff address hypovolaemia. In

the worst lacerations, animals require prompt thoracotomy, and continuous thoracocentesis is performed during patient preparation and during the surgical approach to the thoracic cavity.

Initial fluid therapy was with a crystalloid, as it can be obtained more quickly than any blood products. It has the advantage that it will expand both the intravascular and extravascular spaces and will maintain hydration. 75% of the volume will redistribute to the interstitial space so it will rapidly address expand the intravascular space and correct hypovolaemia initially, but colloidal support is needed in addition. Previously, shock rates of 90ml/kg were given to dogs, which failed to account for those animals with lower needs. Care must also be taken not to overhydrate animals with pulmonary contusions as increased vascular permeability can lead to pulmonary oedema. An initial bolus of 5ml/kg was given over 5 minutes, followed by 2 more boluses until signs of hypovolaemia had reduced. In addition to crystalloids, a synthetic colloid (Volulyte) was given – the larger molecular weight of molecules means it stays within the intravascular space to give intravascular space expansion. Colloids are therefore very useful for treating hypovolaemia secondary to haemorrhage. It is hyperoncotic so will draw fluid from the extravascular space so it is important to give enough crystalloid fluid therapy to support this. Originally colloid dose rates were 50ml/kg/d – this has been reduced to 20ml/kg/d, as higher dose rates can be associated with disruption of normal coagulation. In life-threatening situations, where blood products are available, the risk of death from reduced intravascular volume must be considered against the risk of coagulation effects. Colloids can be bloused within the daily reference range.

Blood products were given due to the high volume blood loss – at presentation she had a calculated blood loss of 30%. Dogs can tolerate 10-15% acute blood loss without blood products if they receive intravascular volume resuscitation. Acute blood loss of >20% is likely to require blood transfusion after crystalloid and colloid resuscitation. Various blood products are available. Some practices keep fresh whole blood, but this is only feasible if a practice is using lots of blood products and has plenty of donors. This system was used in universities prior to the availability of the Pet Blood Bank and is no longer necessary. Fresh whole blood can be obtained from donors and is a good option if a practice does not keep packed red blood cells or plasma, but it is time consuming to find and bleed a donor, and in some situations there may not be time or personnel available to do this. Collecting fresh whole blood may also be useful if there is so much haemorrhage that packed cell supplies have been used. Advantages of fresh whole blood are the presence of clotting factors and platelets.

Many large practices and referral centres keep packed red blood cells (pRBC), although even large centres are limited to 2-3 units at a time. In an acute setting, and if a dog has not had a previous transfusion, dogs can receive either DEA 1.1 positive or negative blood. I tend to blood type so that I can use positive blood on a positive dog, in order to keep negative blood available for other dogs in the practice that may be having chronic blood transfusions and cannot have the wrong blood type. Positive blood can be given to a negative dog if there is no other alternative, but antibodies will be made that cause problems if transfusion is needed in the future. pRBC has a PCV of approximately 80% and will restore intravascular volume and provide oxygen carrying capacity, but there are no clotting factors or platelets. Whilst pRBCs can be used without warming, they should be gently warmed (in a waterbath of 37C maximum to avoid damaging cells, not microwaved!) if given in large volumes to avoid hypothermia. As a general rule, 10-15ml/kg of pRBC's will increase PCV by ≈10%. Transfusion is usually started at 0.25ml/kg/h and the patient is monitored for transfusion reaction, then the rest of unit is given at 4 -10 ml/kg/hr, usually over 3-4h.

Plasma was given as it contains clotting factors and proteins that are depleted with haemorrhage, although plasma is not particularly effective at increasing colloidal osmotic pressure in the quantities given in veterinary practice, hence the previous use of a colloid.

A concern with pleural haemorrhage in veterinary practice, given that blood products will either not be available or in limited supply, is that blood loss will exceed the availability of blood products. Therefore in the face of moderate haemorrhage, I start to collect blood so that it can be used for autotransfusion. Autotransfused blood will maintain PCV and oxygen-carrying capacity, but contains no clotting factors, as they rapidly deplete upon haemorrhage.

Cell saving machines can remove potentially harmful contaminants such as fat and free haemoglobin, but few practices have access to this. The presence of activated factors, such as thrombin, can lead to inflammatory response syndromes and coagulopathy. Therefore, although autotransfusion may mean that allogenic blood products are not needed, there are disadvantages of both. In this case 30% blood loss had been estimated and more fluid was present in the thorax. Therefore autotransfusion was going to be needed if haemorrhage didn't stop. Blood can be collected for up to 24 hours after haemorrhage into pleural or peritoneal cavities, but must not be used if there is infection or neoplasia that could be disseminated, e.g. pleural haemorrhage secondary to haemangiosarcoma. In large dogs where 400-500ml + is collected, the blood can be added directly to a blood collection bag and delivered to the dog. The blood collection bad contains the appropriate amount of anticoagulant. For smaller volumes, blood can be collected into syringes containing anticoagulant – this can be removed from blood collection bags but it is available to buy separately and some large practices will keep it. It is important to avoid damaging red blood cells during collection. Blood is drained from the thorax via a large bore needle or via a thoracic drain and excessive suction is avoided. If the blood is injected into a donor bag, it is done through a large needle and under low pressure. Blood in syringes are closed with a sterile cap. It is important that collection and handling is performed using sterile technique to avoid contamination.

Blood products are given via a filtered giving set or a blood filter if blood is in syringes (cat blood transfusions or small volume autotransfusions) to avoid microthrombi entering the circulation. They must not be given concurrently with calcium or glucose containing fluids.

In this case, no further blood was removed from the thorax after the thoracic drains were placed. No further pneumothorax developed. The post-transfusion PCV was 24% and the dog was normovolaemic. Further units of pRBC were available but it was not considered necessary to give them based on the dog's cardiovascular status. Similarly the autotransfused blood was not needed.

Thoracic drainage

In the presence of large volume pleural haemorrhage, and particularly pneumothorax, it is helpful to place thoracic drains to make management of the pleural space easier. The dog had been hit 6 hours prior to referral and thoracocentesis had been performed every 45 minutes at the referring vet, based on the increased tachypnea, and so ongoing air leakage was expected. Many dogs with pneumothorax will only need thoracocentesis 1-3 times in the first day, even if the first drain is of large volume, and most have resoled in 1-2 days. I use drains in animals with rapid recurrence of pneumothorax (i.e. more often than every 2-3 hours in the first 12 hours) or if there are still moderate volumes produced after 12-24 hours. Drains are also useful in dogs with thick or fat thoracic walls where butterfly needle drainage is difficult, or dogs with poor temperament that do not allow thoracocentesis, as a drain can be placed under sedation. I am more likely to use a drain early since the advent of the small gauge drains placed by Seldinger technique (MILA drain) as the risk of complications during placement is much less than for the large bore trocar drains. They can also be placed in conscious or lightly sedated dogs. Given the ease of placing them, anaesthesia if needed is likely to be brief. Drains can be removed when they are no longer necessary and in trauma cases drains may only be needed for 24-48 hours.

Decision making – surgery vs. conservative management

The hardest question as a surgeon faced with pleural space haemorrhage is deciding whether to manage the case conservatively with fluid and blood prodecuts and hope that it stops, of whether to operate. I am always reluctant to operate as the only way to determine pleural space haemorrhage will be by median sternotomy. It is an invasive procedure and haemorrhage is often seen in normal dogs, and so is more of a concern in a dog that is expected to have depleted clotting factors. Furthermore, acute haemorrhage in any location is often difficult to find in hypovolaemic dogs under anaesthesia, as bleeding often stops with low blood pressure. Sometimes surgery is performed to locate a bleeding vessel only to be faced with the prospect of not being able to find it, and in the worst case scenario

hameorrhage starts again when vascular volume is restored. Therefore, given that by the time we had stablised the dog it had been 7 hours since trauma, she was a small dog and there were 3 units of pRBC available and we were able to collect the haemorrhaging blood for autotransfusion, I elected to wait to see if she stopped haemorrhaging, by continuously assessing vascular volume and blood loss via the drains. Fortunately in this case, there was no further heamorrhage and she quickly recovered, and that is typical of the cases I have treated, but a surgeon must be prepared to change the plan to an exploratory surgery if necessary.

CASE 2 – a series of diaphragmatic ruptures

There was a case series many years ago that stated that the highest death rates were seen in animals operated within the first 24 hours or after one year. The latter remains true, as animals with chronic ruptures may suffer from adhesions that can tear on attempted reduction, or may suffer re-expansion oedema of the lungs when normal pleural volume is restored after surgery. However a more recent study has shown no effect on mortality rate if animals are operated on within the first 24 hours. It is likely that the improvement in veterinary practice facilities and standards of critical care have improved, for example recognizing that high volumes of fluids should not be given as there is the risk of pulmonary contusions after vehicular impact.

Following RTA, pulmonary contusions are the most common cause of respiratory distress; diaphragmatic rupture is much less common. Tachypnea and dyspnoea will vary and it is not possible to distinguish between them on physical examination, although some animals with diaphragmatic rupture will have obviously reduced heart and lung sounds. Textbooks often talk of an empty abdomen, but my experience is that only small amounts of the abdominal viscera herniate into the thoracic cavity. The only real surgical emergency scenario is a gastrothorax, where the stomach herniates into the thoracic cavity, where it can dilate like a GDV if the inflow and outflow tracts are occluded. As well as being dyspnoeic, often to the point of cyanosis, these animals will have hypovolaemia due to pressure of the dilated stomach on the caudal vena cava preventing venous return to the heart. The diagnosis is easy to make on radiography, as the stomach is obviously dilated (note the lack of vessels in the gas and it is surrounding by a tissue line). If gastrothorax is suspected and the animal is cyanotic, the time delay and stress of radiography may be fatal. In these situations I perform thoracocentesis with a view to draining air from the stomach via an intercostal thoracocentesis. I do the same in animals where I have made the diagnosis on radiography to treat the hypoxaemia and hypovolaemia it causes. Whilst there is the concern that there may be leakage of gastric contents into the thoracic cavity, it can be lavaged at laparotomy in the same way that the abdomen is lavaged in a dog with GDV that had gastrocentesis, and the risk of pyothorax will be negligible. The stomach has a tendency to dilate again, so gastrocnetesis can be repeated, but the animal should have surgery promptly. The potential risk of gastrothorax means that I will operate on most diaphragmatic ruptures on the day of presentation, given that there is no evidence that a delay is beneficial. However if the degree of dyspnoea does not match the extent of abdominal contents in the thoracic cavity, i.e. marked dyspnoea and minimal herniation, I worry that the dyspnoea is related more to pulmonary contusion, and in those cases I am more likely to delay surgery to allow pulmonary tissues to recover.

Most diaphragmatic ruptures are easy to treat but some scenarios prove more difficult.

Inability to reduce herniated abdominal contents

If the rupture is small, abdominal contents may pass through and become strangulated within the small hole of the rupture. Even tissues that have not dilated or increased in size with inflammation or congestion can be hard to reduce, as pulling on the tissues can lead to tissues tearing rather than being reduced. This is worse if organs have dilated. In these situations I will insert a scissor between the abdominal contents and the diaphragm, at the site of rupture, and cut the diaphragm. I aim to cut in a direction where repair will be easier, avoiding any diaphragmatic hiatuses (e.g. oesophageal, caval) or at the junction of diaphragm and body wall. To make suturing easier I cut from the rupture in a ventral or ventrolateral

direction, unless the rupture is ventral. In my experience it is possible to do this without incising abdominal organs.

Rupture is continuous with a hiatus

In my experience, a rupture is most likely to be continuous with the hiatus where the caudal vena cava traverses the diaphragm. Potential mistakes in repairing these ruptures are closing the hiatus too much, which can lead to reduced venous return, or inadvertently penetrating the vena cava during suturing. I ensure the suture line stops within a few millimetres of the hiatus, or as much as is necessary to avoid compressing the cava. If the suture penetrates the cava, the suture line should be cut and puled out, and the cava will soon stop bleeding following compression. Note that the cava should not be compressed so that it is obstructed. If the suture line is left crossing the caval wall, the suture's presence is likely to disrupt a clot and lead to further haemorrhage.

CASE 3 – a series of BOAS cases

There are different approaches to surgical techniques, particularly for reducing the size of the soft palate, but the fundamental approach is to widen the nares and shorten (+/- reduce thickness of) the soft palate. I remove the mucosa of the lateral laryngeal ventricles, particularly since it has been shown that the mucosa will not invert again even if the nares and palate are operated on, and will therefore continue to contribute to airway obstruction, although some specialists choose not to. Tonsils are removed if they are large and contribute to airway obstruction.

Following surgery it is important to ensure that there has been an acceptable outcome. Many owners accept poor respiratory function in brachycephalic dogs as it is 'normal', but a 'good' brachycephalic dog should be able to run around for at least 3-5 minutes and to go for walks of 20-30 minutes. Similarly, vets should expect to try to achieve this after surgery, and if not, further investigation should be performed. Following discharge from initial surgery, it is important that owners ensure that their dog has improved exercise tolerance.

Aberrant turbinates

Some dogs may fail to show improvement after surgery. Consideration should be given to the possibility of other abnormalities. Normal non-brachycephalic dogs do not have mucosal contact between conchae and turbinates stop growing posteriorly. However many dogs with BOAS will have aberrant turbinates, and it has been suggested that this is due to dorsal rotation of the maxillary and palatine bone. Whilst conchae are small in absolute size compared to other breeds, they are oversized in relation to available nasal cavity volume, i.e. there is a small nasal cavity despite undersized turbinates. CT and endoscopy studies have shown the following abnormalities:

	Pug	French Bulldog	English Bulldog	Total
Rostral aberrant turbinates	90%	56%	36%	72%
Caudal aberrant turbinates	69%	65%	54%	66%
Septum deviation[1]	98%	14%	0%	55%

Pugs (with BOAS) in particular have a high incidence of aberrant turbinates. There is not yet evidence to suggest that turbinectomy should be considered part of the routine treatment of BOAS dogs. However dogs that have ongoing respiratory difficulty or distress despite standard BOAS surgery will benefit from investigation by CT and endoscopy with a view to performing laser turbinectomy.

Laryngeal collapse

Some dogs will have laryngeal collapse diagnosed when they are treated for BOAS. This seems to be more common dogs that are older at presentation for surgery. My suspicion is that many of those dogs had clinical signs when young that were severe enough to warrant

surgery, but the perception that the noise and exercise tolerance were ‘normal’ (for the breed) prevented the owners querying their dog’s health.

Typically dogs with laryngeal collapse on first presentation are treated as for other BOAS dogs, with surgery of the nares, palate and ventricles. Some of these dogs cannot be recovered from anaesthesia or fail to show a good enough response to surgery. Other dogs have no evidence of laryngeal collapse at the time of surgery but develop it over time, and initial good exercise tolerance/ reduced noise after surgery deteriorates over time. In my clinic, I see a lot of dogs with BOAS, yet few return with clinical signs consistent of laryngeal collapse. That does not mean that these cases are rare if owners choose not to present them for investigation.

Laryngeal collapse can be assessed under anaesthesia. It is graded depending upon the degree of collapse (Grade II and III have collapse of corniculate cartilage alone or both corniculate/cuneiform cartilages respectively), but clinical signs do not necessarily correlate with grade. A modified tieback (using crico-arytenoid lateralization) can be performed, although it will not prevent collapse in all cases.

Case Example – unrelated disease causing dyspnoea

A pug had surgery that I had performed 6 months previously. He had reasonable exercise tolerance prior to surgery but had marked upper respiratory noise post-operatively. At surgery he had Grade II laryngeal collapse but recovered well from the procedure. The owners had returned because he had continued noise but he could tolerate walks of 30 minutes – 2 hours, so the owners chose to have no further investigation. On the day of presentation he had become restless, with decreased appetite. His breathing had abruptly changed – he had louder and more squeaky stridorous breathing and open mouth breathing but no cyanosis. He had developed a swelling in the neck. Such an abrupt change in breathing suggests a complication related to brachycephalic conformation e.g. pneumonia (see later) or a new problem. This case highlights how it is easy to become blinkered in brachycephalic dogs and assume every episode of respiratory difficult is related to confirmation. In the pug, the swelling was due to significant lymphadenopathy of lymph nodes of the neck. Ultrasound examination showed how they impinged on the pharynx and was the cause of the respiratory distress. It was therefore important to address the lymphadenopathy on the assumption that the dog would return to its normal function when the swelling reduced. Ultrasound guided aspirates of the lymph nodes were consistent with a neutrophilic lymphadentitis. The owners declined investigation and the lymphadenitis resolved with antibiotic and steroid therapy. No recurrence has been seen and he remains able to exercise well.

Case Example – pneumonia exacerbating BOAS

Some dogs, especially prior to BOAS treatment, may develop aspiration pneumonia. The upper airway noise is exacerbated by pulmonary and airway noise. It is important to consider pneumonia in a brachycephalic dog that has acute worsening of airway noise, especially if the dog has other relevant clinical signs e.g. crackles on thoracic auscultation, lethargy. Whilst radiography is usually recommended when pneumonia is suspected, it is difficult to perform in a brachycephalic dog who is in respiratory distress, without causing risk of death. Radiographs are difficult to interpret in an anaesthetized brachycephalic dog due to small thoracic cavity size, and acquisition in a young dog with rapid respiratory rate can be difficult. Therefore presumptive treatment is given, including antibiotics and nebulization/coupage. It is important to remember that pneumonia is a medical condition. Surgical treatment of the BOAS conformation must be delayed until the dog has fully recovered from pneumonia.