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Respiratory Surgery Online 'Mini Series'

Session 1: Upper Airway Surgical Conditions

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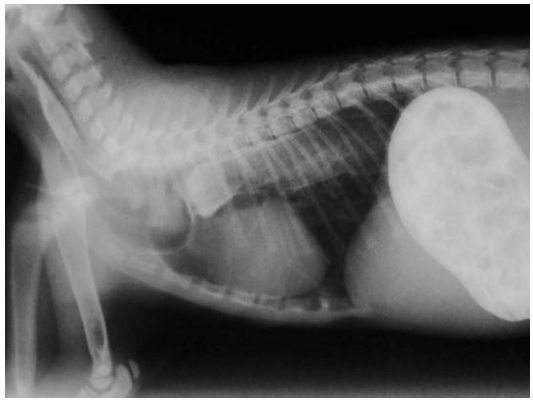

Upper Airway Surgical Conditions

1. 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.

2. Brachycephalic Upper Airway Obstruction Syndrome

Introduction

Brachycephalic upper airway obstructive syndrome is associated with a collection of anatomical deformities that are seen in the Brachycephalic animal. The condition is typically seen in brachycephalic breeds of the dog and cat with the English bulldog, Pekingese, pug dog and Persian being more commonly affected.

The defects are due to a mismatch between skeletal growth (which is dwarf-like due to chondrodysplasia), and the normal soft tissues that would normally develop. Abnormalities that may be seen in animals with BUAOS include:

- Stenotic Nares
- Elongated soft palate
- Hyperplastic tonsils
- Everted laryngeal saccules
- Laryngeal collapse
- Hypoplastic trachea

The Brachycephalic animal may have one, or a combination of these conditions, with variable effect in airway dynamics. Mildly affected animals will have increased upper airway noise (snorting), especially with exercise. Severely affected animals have severe inspiratory stertor and/or stridor, cyanosis, and collapse after exercise. Exercise, hyperthermia, or any stress may exacerbate the clinical signs. These dogs are very prone to hyperthermia even in a cool environment since their most important method of thermo-regulation, panting, is compromised.

The laryngeal and tonsillar changes are considered by many to be secondary changes that develop over a period of time, as a result of altered airway dynamics due to obstruction of the upper airway. Once the laryngeal changes have become established, the animal's respiratory disease becomes very difficult to reverse. Early correction of anatomical abnormalities such as stenotic nares and elongated soft palate may result in a significant improvement of airway dynamics, and may prevent subsequent deterioration during the animal's lifetime.

Stenotic nares and elongated soft palate have a significant influence on airway dynamics, and the most readily corrected anomaly present. They result in an increased respiratory effort, and significantly increase the amount of negative pressure generated within the airway lumen. As a result, they play a significant role in promoting dynamic airway collapse and

causing permanent structural deformity of airway anatomy. Skeletal stenosis of the nasopharynx and nasal chambers also exists, but nothing can be done to correct this.

Diagnosis

Diagnosis is made on the basis of breed, history and clinical examination. Ancillary diagnostic aids include radiography and laryngoscopy. Radiography can be used to assess the length of the soft palate, the available nasopharyngeal airspace and for evidence of tracheal hypoplasia. Definitive diagnosis is made on laryngoscopy that allows for direct visual assessment of the tonsils, soft palate, pharyngeal mucosa and the larynx. The palate is regarded as overlong if it occupies part of the glottic opening and in many cases may hang within the rima glottidis. The overlong palate is usually inflamed and thickened, as may be the pharyngeal mucosa. The larynx should be examined for evidence of laryngeal collapse or eventration of the laryngeal ventricles.

Anaesthesia

Anaesthesia of animals with upper respiratory tract disease should be performed cautiously. The periods of greatest danger are during anaesthetic induction and recovery.

Sedation/Pre-medication should be performed with low-doses of drugs to prevent excessive muscle relaxation and drowsiness. Morphine should be avoided as it may cause vomiting. Pethidine and acetylpromazine are an effective combination. The patient should be monitored constantly following sedation to prevent a respiratory crisis from being overlooked.

Pre-oxygenation prior to induction will guard the patient against hypoxia during intubation.

Induction should be smooth, and allow for rapid intubation. Propofol, or ketamine/diazepam are effective agents. Propofol should be administered slowly to prevent apnoea.

A laryngoscope, and a range of endotracheal tubes should be available. The animal may have laryngeal collapse or tracheal hypoplasia, and will not accommodate a tube size that would be normally used at this body weight.

On recovery, the animal should be kept intubated as long as possible with oxygen supplementation provided. Position the patient in sternal recumbency, and keep the head elevated at all times.

a. Stenotic Nares

The mobile portion of the external nares is comprised of 3 cartilages, the dorsal and ventral parietal cartilage and the accessory cartilage. The alar fold (also called the wing of the nostril or the lateral cartilage) is the nasal structure that forms the lateral border of the nostril. This fold is collapsed medially in dogs with stenotic nares. During inspiration, the alar fold may collapse further, causing complete occlusion of the nostril.

Correction of stenotic nares is a straightforward, and relatively benign surgical procedure. There is a strong argument for its use as a prophylaxis in juvenile animals even if clinical signs are not currently noted.

As a therapeutic technique (in animals presenting with signs of respiratory distress), the naroplasty procedure will almost always be performed in conjunction with some other plastic procedure, usually resection of an overlong soft palate. Conversely, performing a soft palate resection alone without addressing the nostrils would be doing a serious injustice to the animal.

There are several techniques for resection of the stenotic nares. Two different techniques are demonstrated in the video.

The animal is positioned in ventral recumbency with the head at the end of the operating table. In the first procedure, an ellipse of skin is removed form the commissure of the nostril. The tip of the alar fold is then sutured into a more lateral location that opens the nostril.

In the second technique, a wedge of cartilage and epithelium is removed from the lateral portion of the dorsal parietal cartilage and overlying skin. Haemorrhage is controlled with direct pressure. The skin defect is closed with simple interrupted sutures of 2M (3/0) silk or Vicryl these sutures are allowed to slough out.

b. Elongated Soft Palate

The patient should be positioned in sternal recumbency with a gag placed between the maxillary and dental arcades. Use zinc oxide tape to secure the head to the table to prevent movement during surgery. The caudal margin of the tonsillar crypt is used as a landmark for resection. The palate should not be resected rostral to this point. Excessive removal of palatine tissue will result in nasal regurgitation and is almost impossible to reverse.

The oral mucosal surfaces are disinfected with a very dilute solution of povidone-iodine. The caudal border of the soft palate is grasped with De Bakey tissue forceps, and drawn rostrally. Two right angle (tonsillectomy) clamps are then placed from both lateral margins of the soft palate at the proposed level of resection. The redundant palatine tissue is resected

and the palate and clamps oversewn with a simple continuous suture of 3/0 Vicryl. The suture is tightened as the clamp is withdrawn and the suture line is then oversewn again. The patient should be observed for haemorrhage for a minimum of 5 minutes. Postoperative oedema is usually minimal with this technique, but the patient should, as always, be carefully monitored during anaesthetic recovery.

c. Tonsillectomy

This is usually performed simultaneously with soft palate resection. A tonsillectomy clamp is placed over the tonsillar pedicle and the tonsil amputated. The clamp is oversewn with 2M (3/0) Vicryl, the clamp is withdrawn and the suture tied.

d. Everted Laryngeal Ventricles

The increased inspiratory effort secondary to stenotic nares and/or elongated soft palate frequently results in eversion and oedema of the laryngeal saccules (the tissue lining the laryngeal ventricles). This condition further complicates the airway obstruction and should be corrected. The everted saccules appear as round, oedematous structures that obscure the vocal folds.

The endotracheal tube must be removed to visualise and remove the saccules. The saccule is grasped with an Allis tissue forceps and removed by cutting the base of the tissue with Metzenbaum scissors. The opposite saccule is removed in a similar fashion. The vocal folds may be removed at this time if they appear to be contributing to the airway obstruction.

Haemorrhage is controlled by promptly replacing the endotracheal tube so that the cuff is at the level of the larynx, then inflating the cuff to put direct pressure on the laryngeal tissues. The trachea should be gently suctioned if haemorrhage was severe.

e. Laryngeal collapse

Laryngeal collapse occurs due to a progressive loss of structural integrity of the laryngeal structure as a result of chronic upper airway obstruction. For further details, see the subsequent section on laryngeal disease.

Partial laryngectomy (partial arytenoidectomy) has been recommended for these dogs but was successful in only half of the animals in one study. In the author's experience, permanent tracheostomy provides the most effective solution, but requires considerable owner compliance for stoma cleaning and care. Minor amendments to the animal's lifestyle may also be required.

For permanent tracheostomy, the patient is positioned as for a tube tracheostomy and 3 cartilaginous rings are excised. Keep the incision quite cranial in the trachea, where it is most

superficial. Tracheal ring numbers 3 - 5 , or 2 – 4 are best. A section of skin is excised to match the tracheal defect. It is commonly described that the strap muscles (sternothyroideus mm.) dorsal to the trachea should be sutured beneath the trachea to keep it pushed closer to the surface. Provided the cranial segment of the trachea is used, this step may not be necessary. The skin is then sutured directly to the tracheal mucosa with undyed 4/0 monocryl or Vicryl. Any skin deficits are closed routinely.

The stoma is very productive for the first 2-3 weeks, and will require vigilant care. The stoma can become completely encrusted, and airway occlusion can occur. Once the sutures are removed, encrustation usually quickly resolves. The tracheal mucosa also undergoes a squamous metaplasia, and becomes less productive.

3. Laryngeal Paralysis & Collapse

Disease conditions that cause either physical or functional obstruction of the larynx are relatively common in the dog. Frequently, the pathology affecting the larynx develops slowly. As a consequence, dogs with laryngeal disease may display relatively minor clinical signs, the significance of which may be overlooked by their owners. However, it is also possible for animals to present as an acute emergency with severe cyanosis and respiratory distress induced by a period of excitement, exercise or hot weather. Successful management of these conditions requires immediate and effective resuscitation, followed by prompt alleviation of the obstructive process.

Laryngeal diseases that may be seen in the canine patient include laryngeal paralysis, laryngeal collapse, and laryngeal neoplasia. In the cat, neoplastic disease is the usual cause of laryngeal obstruction, although paralysis is perhaps more common than suspected in this species. Although most laryngeal diseases will present with similar clinical signs, the patient's signalment may give some clue to the most likely underlying disease (e.g. brachycephalic dogs are more likely to have laryngeal collapse, whilst paralysis is more common in the middle to larger breed non-brachycephalic dogs.)

Laryngeal paralysis occurs when the vocal folds are unable to abduct (open) in response to exercise or respiratory demands. The dorsal cricoayrtenoid muscle is the only intrinsic laryngeal muscle involved in vocal fold abduction; the recurrent laryngeal nerve innervates this muscle. There are several possible causes of laryngeal paralysis including congenital disease (in Bouvier de Flanders, Bull Terriers and Dalmatians especially), traumatic and neoplastic infiltration (e.g. from a mediastinal mass). However, idiopathic laryngeal paralysis is probably the most common laryngeal condition that will be encountered by the small animal clinician. Idiopathic laryngeal paralysis is usually seen in the middle-aged and older (median 9.5 years) large breed dog. However, it is important to remember that laryngeal paralysis can

also occur in the smaller breed dog and even the cat. It is likely that differences in lifestyle and respiratory dynamics limit the clinical expression of the disease in these smaller animals.

Laryngeal collapse occurs due to a progressive loss of structural integrity of the laryngeal structure as a result of chronic upper airway obstruction. The effects of increased negative airway pressures, and Venturi forces (see later) progressively fatigue the cartilages, until permanent deformation occurs. Early (Grade 1) laryngeal collapse may be limited to soft tissue structures only, with eversion of the laryngeal saccules and oedema of the mucosa. More serious laryngeal collapse include medial deviation of the aryepiglottic folds and cuneiform processes (Grade 2), and ultimately collapse of the corniculate processes of the arytenoid and occlusion of the rima glottidis (Grade 3).

Cancer of the larynx is rare in the dog and cat. This is in contrast with the human, in which laryngeal cancer is common (and related to smoking). Reported tumours in the dog include rhabdomyoma, osteosarcoma, chondrosarcoma, undifferentiated carcinoma, fibrosarcoma, mast cell tumour, squamous cell carcinoma and adenocarcinoma. Most tumours are locally invasive and very aggressive, with significant metastatic potential.

Clinical signs

Laryngeal disease may cause the following clinical signs: exercise intolerance, inspiratory stridor, coughing and gagging, change or loss of voice. Many of the clinical signs of laryngeal disease that may be apparent (e.g. a soft cough, exercise intolerance), are often attributed to ‘old age’ or ‘heart disease’, particularly as they will develop insidiously. However, there are two audible features that are very characteristic of laryngeal disease - a soft ‘ineffectual’ cough, and inspiratory stridor. The clinician who is tuned into these noises is unlikely to overlook a diagnosis of laryngeal disease.

Many dogs with laryngeal disease are asymptomatic at rest. However, rapid decompensation of the respiratory status can occur if the dog becomes excited, is exercised more intensively than usual, or is unable to find a cool area on a hot day. When this happens, the dog can suddenly develop respiratory distress, with rapid escalation into a life-threatening crisis if appropriate action is not taken immediately. Recognising why this rapid deterioration occurs requires an appreciation of the normal contribution of the larynx to respiratory physiology, as well as an understanding of airway resistance, and Bernoulli and Venturi effects.

Diagnosis

Clinical recognition of laryngeal disease is usually very straightforward for the clinician who is experienced with the disease. The acoustic ‘footprint’ of laryngeal disease is very characteristic, and the sensitivity of physical examination alone has been shown to be more than 90% for clinicians experienced with the disease. Features of examination that are useful include direct auscultation of the larynx to aid localisation of the stridor to the larynx. In less

severe cases, listening to the larynx before and after a short period of exercise may accentuate the stridor that is present.

Definitive diagnosis of laryngeal disease requires visualisation of the laryngeal structure, and correlation of movement of the vocal folds during respiration. This can be performed in the conscious dog with an ultrasound probe (10MHz linear transducer) placed directly on the cricothyroid membrane, or more commonly, by direct visualisation of the larynx under a light plane of anaesthesia (laryngoscopy). Because of the high sensitivity of physical examination alone, laryngoscopy is usually unnecessary as an isolated procedure particularly if the dog is severely affected and the expertise for definitive surgical management is not available within the practice. Referral of the dog to a specialist surgical centre for further diagnostic investigation and management should be considered.

When laryngoscopy is performed, care must be taken to only assess laryngeal function with the animal under a light plane of anaesthetic. Care must also be taken to correlate abductive movements of the larynx with inspiratory effort. It is possible to be deluded by passive paradoxical movements of the paralysed vocal folds, which do not correlate with inspiration. The appearance of the larynx can be graded accordingly:

Severity	Laryngeal Paralysis	Laryngeal Collapse
Grade 0:	Normal larynx	Normal Larynx
Grade 1:	Adducted left vocal fold, but some residual motility apparent; right vocal fold appears normal	Musocal oedema and everted saccules
Grade 2:	Paralysed left vocal fold, ‘normal’ appearing right vocal fold	Medial displacement of aryepiglottic folds and cuneiform process of arytenoid cartilage
Grade 3:	Paralysed left vocal fold, adducted right vocal, but some residual motility remains	Complete medial collapse of corniculate process
Grade 4:	Bilateral vocal fold paralysis	

NOTE: Most dogs with clinically apparent disease are Grade 3 or 4. Grades 1 and 2 are rarely associated with clinically significant signs, although features of laryngeal dysfunction (e.g. soft cough) may be recognised by the astute clinician.

The other important aspect in the clinical diagnosis of laryngeal disease is the recognition of other concomitant or complicating disease factors. For examples, population studies have

shown that sub-clinical laryngeal paralysis is remarkably common in the dog, and the same is probably true for laryngeal collapse. Transition from sub-clinical to clinical disease may occur if the animal's ability to compensate for the underlying laryngeal pathology is affected by secondary disease (e.g. aspiration pneumonia) or another unrelated disease (e.g. hypothyroidism, hyperadrenocorticism, obesity). Most dogs with laryngeal paralysis are elderly, and may have features of other endocrine or systemic disease on examination or blood work. The challenge for the clinician is to recognise the significance of these other disease processes on the presenting condition. In most cases, definitive surgical management of the laryngeal disease is the treatment of choice. In others, control of the underlying systemic disease will allow alleviation of the laryngeal compromise and thus delay the need for definitive surgical management of the laryngeal disease.

Routine diagnostic work-up of the patient with laryngeal disease should therefore include a full neurological examination, complete blood count, biochemistry profile and urinalysis, and thoracic radiography. Pursuit of any abnormalities detected in this diagnostic investigation will depend on the severity of the laryngeal disease, and the considered role of the abnormality on the presenting condition.

Surgical management of Laryngeal Disease

Laryngeal Paralysis:

Definitive surgical management of laryngeal paralysis is directed at permanently securing the vocal fold(s) in an abducted position. Surgeons differ in their preference of suture location and placement, but clinical function appears to be similar regardless of technique. Most surgeons only secure a single vocal fold (usually the left), though others advocate a bilateral laryngoplasty. There is little evidence in the literature to definitively appraise the difference in clinical outcome between unilateral and bilateral procedures, although complications appear more frequent with the latter.

Laryngoplasty should only be performed by a surgeon who is experienced with the technique. The consequences of a failed procedure can be devastating for the patient, and there are limited salvage options available. In experienced hands, however, good success rates and reported, with few unexpected complications. The most significant complication is aspiration pneumonia. This is more likely to occur in those animals with megaoesophagus or dysphagia prior to surgery, and every precaution should be taken to recognise these clinical features prior to surgery so that the owner may be warned of the greater prospect of complications. Careful feeding in the immediate post-operative period should be performed until normal swallowing ability is assured.

Laryngeal collapse:

Reversal of the structural changes to the laryngeal cartilages in laryngeal collapse is impossible, and management efforts are therefore directed at lowering the negative airway pressures within the glottic lumen that may be causing dynamic collapse of the weakened structure. Resection of stenotic nares, hyperplastic tonsils, elongated soft palate and everted laryngeal saccules may provide improvements in respiratory status. Correction of obesity is also an important component in the management of this disease. In severe cases, a permanent tracheostomy will provide effective long-term control in carefully selected patients.

Laryngeal neoplasia:

Benign laryngeal tumours can usually be successfully removed with marginal mucosal resections. The treatment of malignant laryngeal tumour is usually unrewarding, and the prognosis very poor.

Conclusion

Laryngeal disease is a common sub-clinical condition in many dogs, and surprisingly, a number of cats. Signs are often attributed to ‘old-age’ and are frequently overlooked by the owner. Rapid escalation into a respiratory crisis can occur, particularly as summer approaches and ambient temperatures rise. Concomitant systemic disease can also contribute to exposing sub-clinical laryngeal disease. Prompt and effective management of most laryngeal diseases can be associated with excellent resolution of clinical signs, and restoration of an acceptable quality of life.