Small Animal Respiratory Surgery
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The following notes include overviews of common surgical conditions of the canine and feline respiratory system. For more details regarding these and other less common conditions, please see Tobias and Johnston Veterinary Surgery: Small Animal.

Surgery of the Nasal Region

Nasal Planum Lesions

The most common tumor of the nasal planum is squamous cell carcinoma. Cats are more predisposed than dogs; however, any animal with little pigment on the planum and high solar exposure will be predisposed to actinic dermatitis, which can progress to squamous cell carcinoma. Squamous cell carcinoma should be suspected in any older, white cat with nonhealing ulcerations or crusting of the planum, pinnae, or temporal region. Diagnosis is by biopsy. Squamous cell carcinoma will invade into local soft tissue and bone; preoperative CT is useful in determining the extent of the disease.

Treatment

If lesions are tiny, the biopsy should be excisional; for larger lesions, margins of at least 1 cm are recommended. The edges of resected tissue should be “painted” with a tissue stain to help the pathologist identify margins. Complete excision can be curative; however, surgery is too late or too limited in many animals. Other treatments include cryotherapy, strontium, photodynamic therapy, and proton radiation.

Intraoperative and postoperative analgesia can be augmented with bilateral local blocks of the infraorbital nerves as they exit the infraorbital foramina. Significant hemorrhage should be expected; the incision can be made with a laser or electrocautery; however, this will damage tissue margins.

In cats, removal of the planum alone is straightforward: all pigmented skin is removed, and a purse string suture of 3-0 nylon is placed along the haired skin margin. The suture is tightened to narrow the stoma slightly; since the area tends to scar down, the purse string should not be tightened excessively. In dogs, the purse string suture technique is not as effective because of subsequent stricture and complete obliteration of the nasal opening. Direct mucosa-to-skin apposition should be attempted; this may require drilling some holes in the dorsal nasal bone, rostral maxilla, and incisive bones for suture placement, since nasal mucosa does not hold suture well. If the tumor is invasive, a rostral maxillectomy will be necessary. The defect is closed with rotating skin flaps, once again attempting to get mucosa-to-skin apposition to reduce stricture formation. Owners should be prepared in advance for the change in appearance to their pet, the need for a long e-collar for 2 weeks minimum, and the risk of stricture or recurrence.
**Nasopharyngeal Polyps**

Nasopharyngeal inflammatory polyps are most commonly diagnosed in younger to middle aged cats and have been reported rarely in dogs. Polyps are thought to arise from the mucosal lining of the Eustachian (auditory) tube or bulla. The cause is unknown. Obstruction of the auditory tube will result in otitis media, which can progress to otitis interna (vestibular signs, head tilt) or otitis externa (irritation, inflammation, drainage).

Clinical signs of nasopharyngeal polyps include nasal discharge, sneezing, stertorous or labored breathing, dysphagia, gagging, voice change, otitis, and Horner’s syndrome. Nasopharyngeal polyps are diagnosed by digital palpation of the region caudal to the hard palate and dorsal to the soft palate (which should be an air-filled cavity) and by cranial retraction of the soft palate with a spay hook or stay suture. Polyps are also visible on skull films and CT. The source of the polyp (the bulla from which it arises) can potentially be discovered on otoscopic examination (fluid or a mass effect under the tympanic membrane), but is best detected with CT or, less effectively, with skull radiographs. With skull radiographs, an open mouth view of the bullae is recommended.

**Treatment**

Treatment is with traction-avulsion, with or without ventral bulla osteotomy. The stalk of the mass is gently (and blindly) grasped with Allis tissue forceps, and steady, rostro-ventral traction is applied until the base of the polyp is released. If completely removed, the final polyp should have a narrow stalk. Evidence of hemorrhage under the tympanic membrane may be seen on otoscopic examination of the ear. Removed tissue should be submitted for biopsy to differentiate polyps from lymphoma, mast cell tumor, and other less common nasopharyngeal conditions.

Cats with otitis media are more likely to have recurrence of the polyp with traction alone. Administration of glucocorticoids (1-2 mg/kg for two weeks, then tapered dosing over an additional 2 weeks) will dramatically reduce the risk of recurrence. Cats with recurrence are treated with traction avulsion and ventral bulla osteotomy.

Horner’s syndrome has been reported in over 80% of cats after bulla osteotomy and can also occur with simple traction of nasopharyngeal and ear polyps. Clinical signs of miosis, ptosis, and third eyelid prolapse usually resolves within a month.

**Oronasal Fistulas, including Cleft Palates**

Oronasal fistulas are congenital or acquired openings that connect the oral and nasal cavities. In young animals, the most common type is a congenital secondary cleft palate, which affects the hard and/or soft palate. Acquired oronasal fistulas most commonly occur after upper canine or canassial tooth extraction. Once acquired fistulas are present for several weeks, they are considered “healed fistulas” because the mucosa between the oral and nasal cavity becomes continuous. Clinical signs of oronasal fistulas include sneezing, nasal discharge, rhinitis, and malodorous breath. Primary cleft palate, which affects the lip and incisive bone, does not usually result in a true oronasal fistula. If affected animals survive to weaning, clinical signs of primary cleft palate are usually cosmetic.

**Treatment**

Treatment depends on the size of the fistula and its chronicity. Acute fistulas from maxillary canine tooth extractions are usually closed with suture in older animals; some may require removal of
underlying bone to appose the soft tissues. In younger animals, fistulas from freshly removed teeth may heal on their own. Necrotic or infected fistulas are left open to drain and granulate. If necrotic fistulas are large, patients are treated with antibiotics and tube feeding until the tissues are healthy enough to permit suturing.

Many surgeons will delay repair of congenital secondary cleft palate until the animal is at least 4 months of age. Older animals will be more tolerant of long anesthetic episodes, and their fistulae may become proportionally smaller as the maxilla grows. Before surgery, animals suspected of having pneumonia should have thoracic radiographs. If pneumonia is present, it is treated before surgery is considered. Animals with acquired oronasal fistula may require advanced imaging (skull radiographs, computed tomography) to detect an underlying cause, particularly if retained tooth roots, neoplasia, or foreign bodies are suspected. In animals that have dehiscence of previous repairs, tissues should be allowed to heal for one month until a second surgery is attempted.

Anesthetic induction is usually routine; a cuffed endotracheal tube is recommended to protect the airway from intraoperative hemorrhage or lavage. Bilateral maxillary nerve blocks are recommended to improve postoperative analgesia. Full maxillary blocks are performed by injecting a local analgesic in a site just rostral to the ramus of the mandible and ventral to the zygomatic arch. Rostral blocks can be performed at the level of the infraorbital foramen. Animals should be prepped for feeding tubes, if necessary.

Most acute, nonhealed fistulas can be repaired with a single pedicle sliding or advancement flap of gingival mucosa and adjacent palatal, gingival, or labial mucosa. Chronic, healed fistulas are often closed with two layers to improve strength and provide immediate reconstruction of nasal and oral cavities. The nasal epithelium is reformed with one or two inverting flaps of surrounding oral mucosa and then covered with a sliding or rotating flap of palatal or labial mucosa. Removal of healthy teeth may be required to prevent damage to the base of the labial pedicle. Elevation of flaps must be performed cautiously to prevent damage to the vascular base of the flap.

Elevated tissues should be handled minimally and gently with stay sutures or fine toothed thumb forceps. Flaps are sutured in place with 3-0 to 5-0 absorbable synthetic material in interrupted or continuous patterns with the knots in the nasal or oral cavity. Synthetic multifilament sutures have been recommended because they are soft, strong, and pliable; however, many surgeons prefer monofilament materials for their handling characteristics. Minor hemorrhage during surgery is controlled with digital pressure. The major palatine artery exits the palatine foramen medial to the 4th upper premolar and travels rostrally within the mucosa. Ligation or bipolar cauteryization of this artery may be required if it is lacerated.

Extensive fistulas can be closed with a skin flap (e.g. temporalis or superficial cervical axial pattern flaps). The skin flap is rotated so that the haired surface is facing the oral cavity. Hair may continue to grow on this surface, but does not cause a clinical problem in patients.

After repair of oronasal fistulas, animals are fed soft foods by mouth or feeding tube for up to 5 weeks, depending on the size of the fistula and strength of repair. Dehiscence is common with large fistulas and usually occurs within 3 to 5 days of the surgery. Traumatic surgical technique, tension on the repair, use of electrocautery during dissection, or previous irradiation of the area may increase the risk of dehiscence. If the area dehisces, it is usually left to heal for month before a second surgery is considered.

Elevation of a Nasal Mucosal Based Flap: Measure the size of the fistula. Divide that distance in half and add a minimum of 3 mm to the resulting amount. This is the minimum width for the palatal mucosa incisions on either side of the fistula. If you don’t have enough width, try another technique. Incise the flaps on either side of the fistula, connecting them rostrally and caudally. Elevate the flaps carefully
Elongated Soft Palate

At rest, the soft palate normally divides the nasopharyngeal and oropharyngeal spaces. During swallowing, the palate is elevated dorsally, covering the caudal aspect of the nasopharynx, to prevent food, water, and saliva from entering the nasal cavity. The soft palate normally covers the rostral tip of the epiglottis at rest. Palates that extend caudal to that point may interfere with respiration.

Elongated soft palate is most commonly reported in brachycephalic dog breeds such as English bulldogs and pugs. Palates may also elongate with increased negative inspiratory pressure secondary to upper respiratory diseases that block the airway, such as laryngeal paralysis in Labrador retrievers or
tracheal collapse in Yorkies. Initially, clinical signs of elongated soft palate include snoring and mild exercise intolerance. As the palate thickens, dogs may vomit or gag. With stress, heat, or overexertion, affected animals can present with cyanosis, collapse, and hyperthermia. Severely affected animals may require emergency intubation or tracheostomy.

Diagnosis of elongated soft palate is based on evaluation of palate length under anesthesia. Brachycephalic dogs suspected to have elongated soft palates should also be evaluated for associated respiratory abnormalities. About 20% of affected dogs and cats will have abnormal nasal turbinates that obstruct the choanae (“nasopharyngeal turbinates”). This abnormality is diagnosed with computed tomography or endoscopy.

Treatment

Before surgery, patients should undergo thoracic radiographs to rule out pulmonary edema, aspiration pneumonia, or heart enlargement and to evaluate the trachea size. If pneumonia is suspected, the animal is treated with antimicrobials such as amoxicillin or Clavamox. A transtracheal wash may be required to obtain samples for culture and sensitivity in animals that do not respond to antimicrobials. Because of associated gastrointestinal disease, drugs that enhance gastric emptying (e.g., metoclopramide), reduce vomiting (e.g., Cerenia), and decrease gastric acid production (e.g., famotidine, omeprazole).

Patients are premedicated with buprenorphine or butorphanol and acepromazine (0.01 mg/kg IV) or another sedative, and preoxygenated by face mask for 5 minutes. Induction should be rapid (e.g. propofol IV) so that the animal can be quickly intubated with a cuffed endotracheal tube. Most clinicians will give an anti-inflammatory dose of glucocorticoids (e.g. dexamethasone SP, 0.05-.1 mg/kg IV) after induction to reduce postoperative swelling. Postoperative nonsteroidal anti-inflammatory drugs should be avoided when glucocorticoids are used.

The animal is positioned in sternal recumbency with the mouth propped open. Palate length is estimated and usually based on the clinician’s judgment. Some veterinarians will trim the palate so that it slightly overlaps with the tip of the epiglottis when in a resting position, while others will remove it at the caudal margin of the tonsils, which is usually farther forward than the tip of the epiglottis, resulting in a shorter palate. Palate resection is performed with a carbon dioxide (CO₂) laser, radiosurgical scalpel, or cut-and-sew technique. Laser resection provides immediate hemostasis and a comfortable recovery. Hemostasis is also good with a radiosurgical scalpel, as long as the veterinarian cuts slowly.

The cut and sew technique often requires long handled scissors, thumb forceps, and needle holders. The endotracheal tube is secured to the lower jaw, and the mouth is propped open on a wire stand, with Gelpi retractors, or by suspending the head on a rope or gauze tie. Stay sutures are placed in the caudal edge of the palate to pull it forward. A portion of the palate width is cut with scissors, and the nasal and oral mucosa of the cut margin is sutured together with 4-0 rapidly absorbable monofilament suture (Monocryl or Biosyn). Transection is continued until the entire palate is cut; the remaining edges are sutured together, and suture ends are cut short once the knots are tied. If stenotic nares are present, they should be corrected under the same anesthetic episode. Everted laryngeal sacculles may resolve in some dogs after the palate is shortened.

Animals are recovered on oxygen. If necessary, animals can be given light sedatives and mild analgesics after surgery. Heavy sedation should be avoided to reduce the risk of aspiration. To prevent panting that could lead to swelling dexmedetomidine can be used to provide a slow, stress free recovery. Reported outcomes for laser and cut-and-sew techniques are similar. Mortality rate is less than 5%, but postoperative vomiting or regurgitation is reported in 18% of animals. Death is usually associated with aspiration pneumonia, pulmonary edema, or airway obstruction from swelling. Animals with postoperative distress should undergo placement of a temporary tracheostomy tube. If the palate
resection is inadequate, clinical signs will likely reoccur. If palate resection is excessive, the animal will reflux water and food through the nose when drinking and may develop coughing and rhinitis.

**Stenotic Nares**

Stenotic nares are most commonly seen as a component of brachycephalic syndrome seen in short nosed dogs and cats. Predisposed breeds include English and French bulldogs, Pugs, Boston terriers, Pekingese, and Cavalier King Charles dogs and Persian and Himalayan cats. Shih tzu may present with severe clinical signs as early as 6 to 8 weeks of age; at that age, they tend not to have other components of brachycephalic syndrome (e.g. elongated soft palate), but those components will develop if the condition is not treated early. The cause of stenotic nares is axial deviation of the dorsolateral nasal cartilage and its associate skin and mucosa (the “wing” or alar fold of the nostril). The negative pressure produced by this airway blockage instigates severe stress on the soft palate, larynx, and trachea and can result in development of tissue swelling and airway collapse. Many animals will also have abnormal conchal development; in fact, the alar folds are actually extensions of the ventral nasal concha, which must be addressed when surgery is performed. About 20% of brachycephalic dogs and cats will have “nasopharyngeal” turbinates that protrude down into the nasopharynx, blocking nasal flow of air. In Europe, nasopharyngeal turbinates are most common in pugs. Repair of stenotic nares in animals with nasopharyngeal turbinates is unlikely to resolve the clinical signs.

Clinical signs of stenotic areas include inspiratory dyspnea, recurrent nasal infections, and sometimes exercise intolerance or poor appetite. Stenotic nares are easily diagnosed on physical examination. Evaluation of concurrent conditions will require anesthesia (e.g., elongated soft palate, laryngeal collapse), radiographs (e.g., hypoplastic trachea), and CT or scoping (e.g., nasopharyngeal turbinates).

**Treatment**

In puppies or cats with small alar folds, stenotic nares can be widened by removal of the alar folds. When performed with a scalpel blade, this is known as the “Trader technique”. In puppies, the ventral half of the fold, with associated ventral nasal concha, is excised with a #11 blade inserted at 40° angle ventrolaterally. Alternatively the cut can be made with a punch biopsy. Bleeding is controlled with digital pressure. The fold can also be removed with a laser, which reduces bleeding; the subsequent white scar will gradually regain pigment over 6 months.

In older dogs and some cats, a wedge of tissue is taken out of the central portion alar fold and rostra extent of the ventral nasal concha with a number 11 blade. The remaining gap is apposed with 4-0 or 5-0 rapidly absorbable suture in an interrupted pattern.

**Surgery of the Larynx**

**Laryngeal Paralysis**

Laryngeal paralysis is a well-recognized syndrome in large breed dogs that results in upper airway obstruction from loss of arytenoid cartilage abduction. It is occasionally reported in cats and small dogs. Acquired laryngeal paralysis is seen in older dogs (median age, 9 years), with males affected more often than females. In the United States, Labrador retrievers are commonly affected. Most commonly,
acquired laryngeal paralysis is considered to be part of a syndrome of generalized peripheral neuropathy. In a study at University of Tennessee, all dogs with acquired laryngeal paralysis had evidence of polyneuropathy on peripheral nerve and muscle biopsies, despite lack of clinical signs of neuromuscular disease in many of the affected animals. Acquired laryngeal paralysis can occur as a result of trauma, iatrogenic (surgical) injury to the recurrent laryngeal nerve or its cranial laryngeal branch, or compression of the recurrent laryngeal nerve by a cranial mediastinal or cervical mass. A congenital form of the condition has been reported in Bouvier des Flandres, Dalmatians, rottweilers, and Siberian huskies, with onset of clinical signs noted before one year of age.

Dogs with laryngeal paralysis usually present with progressive respiratory signs. Initially, owners may note a voice change, inspiratory stridor, and exercise intolerance. Some animals with peripheral neuropathy may have dysphagia or regurgitation from concurrent esophageal dysfunction. Eventually the dogs can develop severe dyspnea, cyanosis, and syncope. Collapse or respiratory distress may be initiated by hot weather, stress, or heavy exercise.

Animals suspected to have laryngeal paralysis should be examined for evidence of other systemic illnesses that occur as a result or cause of laryngeal paralysis. For instance, all patients should have a thorough neurologic examination, thoracic auscultation, and rectal temperature. Basic blood work is performed to look for evidence of systemic illness (e.g. hypothyroidism, hypoadrenocorticism, sepsis from infection) and to evaluation the older patient’s general health status before anesthesia. Thoracic radiographs are evaluated for evidence of aspiration pneumonia, pulmonary edema, intrathoracic masses, or esophageal dilation or for etiologies that could cause collapse or respiratory distress (e.g. primary heart or lung disease).

Diagnosis

Diagnosis of laryngeal paralysis is usually based on examination of the larynx under a light plane of anesthesia. Anesthetics used for laryngeal examination may cause significant depression of laryngeal function. In one study, at least half of healthy dogs had no arytenoid abduction when lightly anesthetized with ketamine/diazepam, acepromazine/propofol, and acepromazine/thiopental. In that study, thiopental alone was considered the best anesthetic agent for laryngeal examination. A combination of acepromazine, butorphanol, and mask induction with isoflurane was also effective. If patients require acepromazine, propofol, or other drugs that reduce arytenoid motion, respiration can be stimulated by administration of doxapram hydrochloride IV (1.1 mg/kg). The animal must be evaluated for paradoxical motion, an abnormal condition where the arytenoid cartilages are drawn inward on inspiration and forced apart by air during exhalation. This motion can be easily confused with normal laryngeal function.

Treatment

Animals that are in distress are immediately stabilized with sedation and oxygen administered by mask or nasal catheters. An IV catheter is placed for administration of fluids and medications. Hyperthermic animals are topically cooled. Glucocorticoids are administered if laryngeal edema is suspected. Patients are monitored with pulse oximetry, when available. Those that do not respond to treatment may require immediate intubation or emergency tracheostomy.

Conservative treatment can be attempted in mildly affected animals, including weight loss, stress reduction, treatment of any pneumonia, and restriction of exercise or exposure to high ambient temperatures. Because laryngeal paralysis is a progressive condition, most animals managed conservatively will require future treatment.
The most common surgical options include unilateral arytenoid lateralization, partial arytenoidectomy, or vocal fold resection. Laryngeal tieback (unilateral arytenoid lateralization) is considered the technique of choice by most surgeons. Unilateral arytenoid lateralization is usually performed through a lateral cervical incision. A suture is passed through the cricoid cartilage and the muscular process of the arytenoid cartilage and is tied gently; this suture will prevent the arytenoid cartilage from being pulled back into the airway. Only one side is “tied back”: bilateraly arytenoid lateralization is much more likely to result in aspiration and death.

Improvement is expected in 90% of animals undergoing unilateral arytenoid lateralization, and 70% are still alive 5 years after the surgery. Complications are reported in 10% to 28% of dogs and include aspiration pneumonia (8% to 33%), coughing and gagging (16%), suture failure or return of clinical signs (4% to 8%), gastric dilatation and volvulus (4%), respiratory distress (2% to 4%), and sudden death (3%). Aspiration pneumonia may occur shortly after surgery or at any time for the remainder of the dog’s life, and is more common in dogs with concurrent megaesophagus. Perioperative administration of metoclopramide reduces the risk of postoperative aspiration pneumonia.

Laryngeal Collapse

Laryngeal collapse represents an advanced stage of brachycephalic airway syndrome. As a result of airway narrowing, increased resistance, and high negative pressure, the arytenoid cartilages lose their rigidity, allow them to deviate medially and block the airway. Initially, the cuneiform processes are displaced toward midline; eventually, the corniculate processes collapse as well. Mild cases respond to correction of associated conditions (stenotic nares, elongated soft palate). Moderately to severely affected animals have a more guarded prognosis. These dogs require placement of a temporary tracheostomy tube and some sort of surgical intervention, such as laryngeal tie back or permanent tracheostomy. About 80% of affected dogs that undergo temporary tracheostomy and unilateral laryngeal tie back (combined cricoarytenoid and thyroidarytenoid lateralizations) do well after surgery, as long as the tracheostomy tube is left in for at least 24 hours after surgery. About 50% of surviving dogs have intermittent episodes of regurgitation.

Vocal Cord Resection

Resection of the vocal folds can be performed to reduce phonation (“debarking”) in dogs or to widen the airway as a treatment for laryngeal paralysis. Debarking does not prevent noise, but it does reduce its volume. Debarked shelties no longer annoy upstairs neighbors; however, the harsh, raspy noise they subsequently produce is not entirely pleasant for owners. Still, it may help to prevent ordinance infractions and subsequent fines. Effectiveness of vocal cord resection is variable: the more tissue that is removed, the less chance there will be any loud noise. If large amounts of tissue are removed without suture apposition of the mucosa, however, animals may develop laryngeal stenosis (“webbing”) and subsequent respiratory distress.

Surgery

Vocal folds can be partially removed through a transoral approach, using instruments and positioning similar to that for dogs with elongated soft palates. The surgery is performed without an endotracheal tube in place, so the animals must be maintained on injectable anesthetic. The vocal fold and associated
muscle are grasped with long handled instruments and cut at the top and bottom, leaving 1-2 mm of mucosa ventrally so that the ventral commissure is not disrupted. This reduces the chance of granulation tissue formation and subsequent webbing and stenosis. Some clinicians also try to remove the vocal process. About 60% of dogs develop scar tissue and are able to produce a muted bark within months to years after the procedure. A more permanent solution is to resect the vocalis muscles and overlying mucosa through a ventral midline approach (cutting through the thyroid cartilage). The mucosal defect is sutured primarily, and the cartilage is reapposed. This approach is challenging in toy breed dogs.

**Surgery of the Trachea**

**Tracheal Collapse**

Collapsing trachea is a progressive disease usually found in small and toy breed dogs such as Yorkshire terriers, poodles, and Pomeranians. Etiology is unknown; for some reason, the tracheal rings lose their organic matrix, and the cartilages are unable to maintain their C shape. Initially the dorsal longitudinal membrane becomes floppy and is pulled into the airway during breathing. The ends of the tracheal cartilage become farther apart and the C shape of the ring eventually flattens, completely occluding the airway, particularly at the thoracic inlet. The mainstem bronchi can also be affected. Other conditions associated with tracheal collapse include lung parenchymal disease, chronic heart disease, and obesity. Tracheal collapse can be exacerbated by pressure of an enlarged left atrium on the left mainstem bronchus in patients with chronic mitral valve disease.

Clinical signs usually start with a cough that can be harsh, dry, or sound like a goose honk. Physical examination findings may be nonspecific. In some dogs, the cough can be initiated with tracheal palpation. In dogs that are stressed or anxious, tachypnea, hyperthermia, and cyanosis may be present. Increased inspiratory and expiratory sounds or cardiac murmur may be present on auscultation. On abdominal palpation, many animals have enlarged livers. Many dogs will have aerobic growth on culture of the tracheal secretions (obtained during brush cytology) but do not consistently have cytologic evidence of tracheal inflammation or infection.

Diagnosis of tracheal collapse is based on radiographs, fluoroscopy, and especially endoscopy. The trachea is usually collapsed in a dorsoventral direction and can be mildly to severely affected. Radiography underestimates the extent of collapse. Endoscopy is particularly useful for evaluating extent of the tracheal collapse and determining whether bronchial collapse is also present. Some animals may have concurrent laryngeal paralysis, so laryngeal function should be examined under light anesthesia before intubation for endoscopy or surgery.

**Treatment**

Treatment depends on the severity of clinical signs and tracheal collapse and whether any concurrent conditions can be improved. Animals that are distressed or cyanotic should be administered oxygen and sedatives as soon as possible. Antitussive agents, such as hydrocodone syrup or butorphanol, are administered to reduce coughing. Injectable or oral steroids are given to reduce inflammation and edema. Bronchodilators (aminophylline, theophylline) improve mucociliary clearance and decrease small airway spasm. Obese animals are placed on a weight reduction diet; in some animals, weight reduction alone can resolve signs. Neck collars are removed, and harnesses are used in animals that are leash walked. Up to 70% of animals have improvement in clinical signs with medical management alone.
**Intraluminal Stents:** Intraluminal stenting is a minimally invasive technique for preventing tracheal collapse. It is usually performed under fluoroscopic or endoscopic guidance with the animal maintained on injectable anesthesia. To determine stent size, the maximal diameter of the trachea is measured on radiographs taken under anesthesia during manual inflation of the trachea and lungs, and the length of the trachea is determined on radiographs and with endoscopy. A stent is chosen that will expand to 1-2 mm greater than maximal tracheal diameter and that preferably will support the entire trachea, or at least bridge trachea beyond the collapsed region. Current intraluminal stents are made of a nickel-titanium alloy (“nitinol”) that has a high radial force to resist dynamic compression, plus excellent flexibility to help the stent retain its shape. They are self-expanding, with a repositionable/reconstrainable delivery system. This allows a partially deployed stent to be retracted back into the delivery system and repositioned during fluoroscopic or endoscopic evaluation. The catheter containing the stent is placed into the trachea under endoscopic guidance, and the stent is gradually released, starting at the tracheal bifurcation and extending cranially. To reduce coughing, the stent should not extend into the region of the cricoid cartilage or into a mainstem bronchus.

Dogs that undergo intraluminal stent placement are always prone to coughing after the procedure: after all, there is a foreign body in their airway. Eventually the stent becomes covered with tracheal epithelium, but that epithelium may not have normal structure or function, and also the bronchial may continue to collapse. All dogs are placed on cough suppressants and glucocorticoids after the procedure, and many may need sedation and antibiotics for two weeks. Glucocorticoids are continued for one month and then are gradually decreased. If the cough reoccurs after the glucocorticoid dose or cough suppressant is decreased, the animal is placed back on a higher or more frequent dose. The cough cycle must be broken immediately in these dogs, even if it means keeping the dogs heavily sedated for days. Coughing can result in breakage of the stent. Because stents cause coughing, they are not used in dogs in which clinical signs are limited to coughing.

Intraluminal stents are expensive and are therefore usually placed in animals that cannot be managed medically. Mortality rates after stent placement are 17% within the first two months; overall, about 31% of dogs die of respiratory problems 5 days to 2.5 years after placement. Dogs can live for more than 4 years after stent placement. Besides continued coughing, complications include stent fracture (>20%), stent migration, tracheal collapse cranial or caudal to the stent, exuberant granulation tissue formation, rectal prolapse, and perineal hernia.

**Extraluminal Tracheal Ring Prostheses:** The trachea can also be supported by surgical placement of extraluminal (peritracheal), C-shaped, synthetic rings above, below, and at the level of collapse. Prostheses are secured to the trachea with interrupted sutures. A cervical approach is used for animals with cervical or thoracic inlet collapse; thoracotomy is required for intrathoracic distal tracheal collapse. Extraluminal prosthesis placement requires dissection of one or both recurrent laryngeal nerves from the trachea, which increases the risk of postoperative laryngeal paralysis. Some animals may require laryngeal tieback after surgery. Some surgeons automatically perform unilateral arytenoid lateralization in order to avoid this complication. Because of the potential complications of extraluminal prosthesis placement and the invasive nature of the procedure, intraluminal stenting is used more commonly.

**Tracheal Injury**

Tracheal injury can occur with cervical bite wounds, penetrating foreign bodies, laceration from stylettes during intubation (usually noted in cats), or necrosis from endotracheal cuff overinflation. Clinical signs include dyspnea, anorexia, lethargy, coughing, stridor, and subcutaneous emphysema. Affected animals
may have pneumomediastinum, pneumoretroperitoneum, or pneumothorax on radiographs. Most cases with perforating tracheal injury respond to conservative management: cage rest, oxygen supplementation, sedatives, and a well-padded cervical bandage. Surgical intervention is recommended if dyspnea worsens or does not respond to oxygen, or if emphysema progresses. Emphysema usually takes about 2 weeks to resolve. Location of the tears can be determined with endoscopy; lacerations are closed through an open, surgical approach with fine absorbable suture material.

**Permanent Tracheostomy**

Permanent tracheostomy may be required in animals with laryngeal masses or severe collapse. It is rarely performed in small animals because of the intensive veterinary care required for weeks after the procedure and life-long management required by owners. The procedure is technically simple: an opening is made in the mid-cervical trachea, and its margins are sutured to the skin edge. Complications occur for several reasons. The stoma will shrink about 50% as it heals; additionally, excessive skin and soft tissue in the area will hang down over the resultant stoma, both of which can lead to airway obstruction. Stomas that are too large can result in tracheal collapse. For the first 4 weeks after the procedure, the animal produces thick, mucoid secretions that can block the airway. The site must be cleaned and the airway humidified, with the schedule dependent on productivity of the animal; in some animals, cleaning initially may be required every 2-4 hours. Animals are therefore kept under observation until the owners can manage stoma care. Because filtration, humidification, and airway protection are bypassed, animals with permanent tracheostomies are prone to respiratory infections, foreign body inhalation, and drowning.

**Surgery of the Thoracic Cavity**

**Diaphragmatic Hernias**

The two primary etiologies for diaphragmatic hernia include trauma and, less frequently, abnormal development of the transverse septum of the diaphragm, known as congenital peritoneopericardial hernia.

**Traumatic Diaphragmatic Hernia**

With traumatic diaphragmatic hernias, the disruption occurs most commonly through the diaphragmatic costal muscle. In most affected animals, the liver herniates into the thoracic cavity. Inflammation or incarceration will subsequently result in pleural effusion, which compounds the ventilatory derangements. Most animals present with dyspnea and exercise intolerance as a result of lung compression by viscera or pleural effusion. Pleural effusion is particularly common with liver entrapment. Traumatic hernia is occasionally an incidental finding. Other clinical signs are associated with the viscera entrapped within the hernia (e.g. vomiting or sepsis from intestinal obstruction) or with concurrent traumatic injuries. Heart sounds are muffled on physical exam, and gut sounds may occasionally be heard in the chest. Diagnosis is most commonly based on plain thoracic films; occasionally, contrast radiographs or ultrasound may be performed.
Treatment

Surgical correction of diaphragmatic hernias is recommended as soon as the patient is stable. Early surgical treatment of stable animals increases the chance of survival; diaphragmatic hernia repair should take precedence over fracture repair in most animals. Diaphragmatic hernias are approached through a ventral midline incision, with mechanical or manual ventilation of the patient initiated before the procedure begins.

Hernial enlargement may be required to “reduce” hernial contents (return them to the abdomen). A caudal median sternotomy can be performed if adhesions prevent organ reduction (for instance, if the liver is adhered to the pericardium). In some patients, liver lobectomy is required. The hernia is closed primarily with 2-0 monofilament absorbable suture in a continuous pattern. The edge of the diaphragm does not need to be “freshened” (trimmed of scar tissue) for healing. Interrupted stay or sutures along the “corner” (center angle) of any L shaped tear can be preplaced to align the diaphragm edges. Closure should begin dorsally at the most difficult region of visualization. Caution should be taken when placing suture bites in the diaphragm near the caudal vena cava. If the muscle has torn off of the costal junction (torn off the ribs), sutures can include the ribs or the rib periosteum. A thoracostomy tube can be placed through the lateral thorax before diaphragmatic closure or transabdominally and incorporated into the diaphragmatic closure. For a transabdominal chest tube, the diaphragm closure is partially completed; the tube is then placed through the remaining diaphragmatic tear with its end extending out the abdominal incision, and the diaphragm closure is completed.

Animals with chronic diaphragmatic hernias may have diaphragm atrophy and fibrosis that prevents diaphragm muscle apposition. Closure options include transversus abdominis flaps, synthetic mesh, or collagenous auto- or xeno-grafts (fascia lata, bovine pericardium, porcine small intestinal submucosa). Omentum can be used over the top of these materials to improve blood supply and support these repairs.

Once the hernia is closed, some, but not all, of the air can be removed from the thorax. Full re-expansion and re-establishment of negative pressure should NOT be performed, however, because of the risk of trauma to pulmonary parenchyma and subsequent re-expansion pulmonary edema. Mortality rate before surgical correction of traumatic diaphragmatic hernia is approximately 15%; postoperative survival rates range from 82% to 94%, with acute complications seen in up to 50% of patients.

Peritoneopericardial Diaphragmatic Hernia.

With this congenital defect, various organs (liver, falciform ligament, omentum, spleen, small intestines) may enter the pericardial sac, resulting in indirect pulmonary compression and subsequent respiratory insufficiency. Some animals may have unremarkable physical exams, while others may be underweight or dyspneic, have muffled heart sounds, or have other midline defects.

Diagnosis is based on changes seen on thoracic radiographs, including an enlarged cardiac silhouette and abnormal intrapericardial tissue density. In some patients ultrasonography or computed tomography (CT) may be necessary for definitive diagnosis.

Treatment

Surgical closure is approached through a ventral midline incision. In most animals, the pericardium is circumferentially adhered to the diaphragmatic defect; therefore, a pneumothorax is not always present during laparotomy. Ventilation is still recommended, however. Occasionally the defect must be enlarged to permit removal of entrapped or enlarged organs. Once the contents are reduced, the defect is closed with a continuous pattern of 2-0 monofilament absorbable suture. Air entrapped within the
pericardium can be removed by transdiaphragmatic pericardiocentesis. A thoracostomy tube may not be required if the pericardial-diaphragmatic seal has not been disrupted.

The biggest debate regarding peritoneopericardial hernia repair is the necessity for surgery in asymptomatic animals. In one study, 2 of 22 asymptomatic cats had progression of clinical signs without surgical treatment (7 additional cats were lost to follow-up). Animals that are having clinical signs (respiratory, gastrointestinal) are usually treated surgically. Postoperative mortality rates range from 5% to 14%.

**Lung Lobectomy**

Lung lobectomy is performed when a portion of the lung is nonfunctional or leaking because of neoplasia, abscess, torsion, trauma, or structural abnormalities (e.g., bulla or blebs). Obviously, the animal must be intubated and manually or mechanically ventilated during the procedure, and a chest tube is usually placed to allow drainage of any accumulated air or fluid after the surgery. Because thoracotomies are painful, the animals may receive epidurals before surgery, intraoperative local nerve blocks, and block by infusion of bupivicaine/lidocaine through the chest tube, along with systemic and oral administration of opioids and nonsteroidal anti-inflammatory drugs, after the surgery.

The most common reason for lung lobectomy is removal of a solitary lung lobe tumor. Unfortunately, lung lobe tumors are most commonly metastatic and therefore found in multiple locations. Metastatic tumors are rarely treated surgically. Single, primary lung lobe tumors are usually bronchogenic or alveolar carcinomas. Survival time after lung lobectomy for neoplasia depends on the histologic cell type, tumor size, presence of metastasis or pleural effusion, and clinical signs. Survival times are better in animals with no clinical signs (545 days vs. 240 days) or lymph node involvement (452 vs. 26 days). Prognosis is best in dogs that have no pleural effusion, clinical signs, or lymph node involvement and have a solitary, well differentiated carcinoma that is <5 cm in diameter: half of those dogs will live 1 to 1.5 years. Cats with well differentiated tumors and no metastasis may live an average of 1.9 years after surgery, versus 2 months for poorly differentiated tumors.

Torsed lung lobes occur most commonly in deep chested large breed dogs, pugs, and Yorkies. Affected dogs and cats will have pleural effusion and lung consolidation on radiographs and ultrasound. After lung lobectomy, survival rates are 50 to 61%, with the best outcome seen in pugs (92% survival).

Lobectomy of consolidated, infected or abscessed lobes provides faster recovery than conservative management. Mortality rates are 20% and correlate with the amount of lung tissue removed: mortality rate is 14% after removal of one lobe and 60% after removal of 3 lobes (the maximum amount that can be removed).

Dogs with spontaneous pneumothorax are explored through a midline approach so that all lung lobes can be evaluated, since lesions are usually found in multiple lobes. The thoracic cavity is filled with warm saline, and the animal is “bagged” (the lungs manually inflated) to find the source of the air leak (look for bubbles). The damaged lobes are removed partially or completely, and a chest tube is placed to monitor for continued air leakage after surgery. In dogs with spontaneous pneumothorax that were treated with median sternotomy and lung lobectomy, recurrence and mortality rates are 3% and 12%, respectively. Animals managed conservatively with a chest tube had a 50% recurrence rate and 53% mortality rate.

**Surgical Approaches**

When the disease is limited to one side of the chest, lung lobectomy is performed more easily through a lateral thoracotomy (usually a 4th, 5th, or 6th intercostal space). The skin, subcutis, and muscles over the
lateral thorax at the desired intercostal site are incised, and the chest is entered by cutting intercostal muscles between the ribs. A chest tube is placed, and the chest is closed by placing sutures around the ribs and pulling them closer together, followed by closure of the individual layers external to the ribs.

A median sternotomy is particularly useful for exploration of the entire thoracic cavity in animals with spontaneous pneumothorax or penetrating foreign bodies (e.g. grass awns) or other conditions that affect both sides of the chest. Lung lobectomies are more challenging to perform through a median sternotomy, however, because the vessels and bronchi are dorsal. With a median sternotomy, the sternum must be split with a saw on midline, basically resulting in a fracture. Animals are much less painful if a portion of the sternum- one or two sternabrae and either the manubrium cranially or the xiphoid caudally- is left intact. This reduces postoperative motion along the sternum. The sternum is repaired with orthopedic wire in a figure eight pattern around the costal-sternal junction to hold the two halves of each sternabra together.

Lung lobectomy is most quickly performed with a thoracoabdominal (“TA”) stapler. With one application, these instruments place 2 or 3 rows of metal staples across the vessels, bronchus, and parenchyma at the base of the lobe. Staplers can even be used to amputate half of a lung lobe; surprisingly, the parenchyma seals easily. If staplers are not available, the pulmonary artery and vein must be carefully dissected along the base of the lobe, and each is triple ligated and transected. The bronchus is then ligated, transected, and, if large, oversewn with fine absorbable suture. Tears in the lung parenchyma can also be oversewn with fine (4-0 or 5-0) absorbable mattress sutures. Any twisting or upward lifting of lung parenchyma with suture needles or forceps will result in larger suture holes or tearing of the parenchyma.

**Chylothorax**

Chylous effusion usually has a milky, white or pinkish-white appearance. Chyle is a protein rich transudate (protein, 2.5 to 4 g/dL) with a cellularity initially rich in lymphocytes and, over time, with nondegenerate neutrophils. Chyle is definitively diagnosed by its triglyceride content, which is higher than that of serum. Chylomicrons can be seen within the fluid, particularly with a Sudan black stain.

Chylothorax develops as a result of increased hydraulic pressure within the cranial vena cava or, less commonly, secondary to trauma. Nontraumatic etiologies may include mediastinal neoplasia (thymoma, lymphoma, heart based tumors), fungal disease (blastomycosis), heartworm disease, jugular vein thrombosis, pericardial effusion, peritoneopericardial diaphragmatic hernias, heart disease (cor triatrium dexter, tetralogy of Fallot, tricuspid dysplasia, double chamber right heart), or thoracic duct abnormalities. If these causes are ruled out, the condition is considered idiopathic.

Chronic chylothorax can be associated with fibrosing pleuritis, especially in cats. Lung lobes of affected animals will fail to inflate after the pleural cavity has been drained. Surgical treatment may reduce fluid production in these animals but cannot resolve the fibrosis.

**Treatment**

Conservative management of chylothorax [intermittent thoracocentesis, a low fat-diet, and use of the nutraceutical rutin] is unlikely to be successful unless chylothorax is caused by trauma. Surgery is therefore the treatment of choice. A wide variety of procedures have been recommended, including thoracic duct ligation, cysterna chyli obliteration, partial pericardiectomy. These three procedures are usually performed simultaneously to give the best chance of a positive outcome, with success rates averaging about 80 to 88% in dogs. Outcomes are much poorer for Afghan hounds. Cats are more likely
to have thickened pleura, which prevents lung expansion, and therefore may continue to have clinical signs, even if effusion resolves.