Brachycephalic airway obstructive syndrome (BAOS)

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Brachycephalic airway obstructive syndrome (BAOS) refers to multilevel upper airway obstruction and secondary soft tissue collapse in brachycephalic breeds. A combination of primary anatomical abnormalities and secondary changes can progress to life-threatening respiratory compromise. Early recognition of BAOS would allow the clinician to make early recommendations for medical and surgical management, which can improve the quality of life in affected animals.

Brachycephalic breeds have early ankyloses in the basicranial epiphyseal cartilage of the skull, which leads to chondrodysplasia of the longitudinal axis of the skull. This trait has been propagated by breeders, and brachycephalic breeds have become very popular. Common brachycephalic breeds include English and French bulldogs, Pugs, Boston terriers, Pekingese, Maltese, Shih Tzu, Boxers, Cavalier King Charles spaniels, Yorkshire terriers, Miniature Pinschers, and Chihuahuas.

The deliberate selection towards a short nose, unfortunately, comes with undesirable anatomical soft tissue changes that compromise normal breathing. Primary manifestations of BAOS are due to compressed nasal passages and altered pharyngeal anatomy. Stenotic nares, the result of congenital malformations of the nasal cartilages which cause medial collapse of the alae, are found in 17%-77% of brachycephalic breeds. Nasopharyngeal turbinates, abnormal turbinates that extend caudally from the choanae into the nasopharynx, are found in 21%-100% of brachycephalic breeds. Elongated soft palate, a soft palate that extends past the epiglottis tip, is found in 62%-100% of brachycephalic breeds. Hypoplastic trachea, a trachea with a diameter < 16% of the thoracic inlet, is found in 13% of brachycephalic breeds.

The airflow through the nasal cavities accounts for 77% of the total airflow resistance. Brachycephalic dogs must overcome the increase in airway resistance and develop exaggerated intraluminal pressure gradients during breathing. Secondary manifestations of BAOS are due to airway turbulence, chronic inflammation and mucosal trauma from pathologically increased pressures. The soft tissues will become inflamed, tonsils will evert from their crypt (9%-56%), and laryngeal sacculles will evert and block the rima glottis (53%-66%). Chronic inflammation leads to cartilage degeneration and the folding of the cuneiform processes of the larynx medially, thereby decreasing the rima glottides opening and leading to laryngeal collapse. This condition is seen in 53%-90% of brachycephalic breeds. A vicious cycle of increased airway resistance that leads to edema and swelling, and worsening resistance is often responsible for acute respiratory distress and presentation to veterinary hospitals.

Most owners report heat, stress and exercise intolerance, snoring, inspiratory dyspnea, and in severe cases cyanosis and even syncopal episodes. Sleep apneas can be observed, and occasionally gastrointestinal signs such as vomiting, regurgitation, and ptyalism. The typical snoring sound (R-sound) is caused by air turbulences in the oropharyngeal region due to an elongated soft palate, G-sounds originate from the larynx, and high-pitch sounds associated with extreme inspiratory effort are related to laryngeal collapse. Endoscopic and histologic changes can be found in the esophagus (37% distal esophagitis), stomach (89%) diffuse gastric inflammation), and duodenum (53% diffuse duodenal inflammation). In addition, hiatal hernia and pyloric stenosis have also been described.
The mainstay of medical management for dogs with BAOS is weight management, since studies have shown a correlation between elevated body condition score and the severity of respiratory distress. In addition, light sedation and anti-inflammatory drugs may also be effective at reducing pharyngeal and laryngeal swelling.

Surgical management of BAOS should be considered early, to decrease the development of secondary manifestations. Instead of waiting until the animal is fully grown, the timing of the surgical correction is better based on clinical symptoms. Alaplasty, or correction of stenotic nares, and staphylectomy, or shortening of an elongated soft palate, can be performed as early as 3-4 months of age. Several techniques for alaplasty have been described, and include horizontal, vertical, and lateral plane wedge resections with the use of a scalpel blade, electrosurgical device, and CO₂ laser. A less commonly performed technique is alapexy. Dysplastic turbinates obstructing the nasal passages and nasoturbinates can be removed with CO₂ laser. Palatoplasty techniques consist of staphylectomy (resection of the caudal portion) or folded-flap palatoplasty (correction of excess length and thickness). Palate shortening can be accomplished using scissors, scalpel blade, monopolar electroscaipers, bipolar vessel sealing devices, and CO₂ laser. Laryngeal sacculles are only removed when they contribute significantly to the obstruction. They can be removed by scissors, scalpel blade, CO₂ laser, tonsil snare, or laryngeal cup forceps. Everted tonsils can be treated by tonsillectomy, however, if the primary cause is corrected, and inspiratory pressures normalized, the tonsils may return to the tonsil crypts.

The immediate postoperative period is not without risk, with the most common complications including regurgitation or vomiting, and aspiration pneumonia, excitation, laryngeal swelling, and dyspnea. Antibiotics, sedatives, anti-inflammatory drugs, proton pump inhibitors and oxygen supplementation are routinely used. In emergency situations, blood gas analysis, endotracheal intubation, or placement of a tracheostomy tubes should be available.

Several long-term studies that evaluated the results of surgical correction found overall success rates around 90%. There is no significant difference in outcome based on the age, breed, presence of hypoplastic trachea, degree of laryngeal collapse, and the number of brachycephalic components. So, even advanced cases of increased airway resistance and laryngeal collapse, might still benefit from surgical correction.

References


Laryngeal paralysis

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Laryngeal paralysis is the effect of an inability to abduct the arytenoid cartilages during inspiration, resulting in respiratory signs consistent with partial upper airway obstruction. The incidence of laryngeal paralysis is higher than commonly perceived. This is mainly a result of incorrect diagnosis because of a failure to recognize the typical clinical signs. On the other hand, many cases that are correctly diagnosed are given an improperly grave prognosis.

The etiology of the disease can be congenital (hereditary laryngeal paralysis or congenital polyneuropathy), or acquired (trauma, neoplasia, polyneuropathy, endocrinopathy). But the most common form of acquired laryngeal paralysis is typically seen in old, large breed dogs and is a clinical manifestation of a generalized peripheral polyneuropathy. This generalized polyneuropathy is a slowly progressive degenerative condition and obvious clinical signs of general polyneuropathy and dysphagia can take months to years to develop. Therefore, the term geriatric onset laryngeal paralysis polyneuropathy (GOLPP) has recently been used to describe the commonly encountered syndrome of acquired idiopathic laryngeal paralysis (AILP). It commonly occurs in breeds such as Labrador retrievers, Rottweilers, Afghan hounds, Irish setters, golden retrievers, Saint Bernards, Irish setters and standard poodles.

Dogs with unilateral laryngeal paralysis (mostly left-sided) will only display clinical signs during strenuous activities (i.e. working dogs). Failure to abduct the arytenoid cartilages during inspiration results in increased resistance to airflow and turbulence through the rima glottides leading to the typical inspiratory stridor. Dysphonia is caused by the inability to tense the vocal cords, which results in the dog’s voice changing to a weak, hoarse bark. Partial obstruction of the upper airways by the paralyzed arytenoids leads to exercise intolerance. Concurrent diseases, such as megaesophagus and aspiration pneumonia, can also be present or can develop during the course of the disease.

Diagnosing laryngeal paralysis based on clinical signs, breed and history alone, has a very high sensitivity (90%). But laryngeal inspection remains essential in order to rule out other causes of laryngeal stridor (e.g. laryngeal tumor) and confirm the suspected diagnosis of laryngeal paralysis. Direct visualization of the larynx can be achieved via transnasal or peroral laryngoscopy. As the latter has a 95% interobserver agreement, it is considered the gold standard of diagnosis. Laryngeal inspection involves the evaluation of the arytenoid cartilages for active abduction during inspiration and passive adduction during expiration. Paradoxical movement in laryngeal paralysis patients occurs when the increased negative airway pressure during inspiration results in adduction of the arytenoids and, subsequently, the positive pressure during expiration results in passive return of the arytenoids to their resting position.

Conservative management of laryngeal paralysis can be considered in older patients with minimal to moderate clinical signs. This involves anti-inflammatory drugs to decrease laryngeal swelling and a weight loss program for overweight patients. The owners should also be educated on the changes in the patient’s routine and environment. A cool area should be prepared for the patient, especially in the warmer months of the year.
Patients should not be allowed to perform strenuous exercise. Short walks using a harness can be permitted during the cooler periods of the day.

Surgical management is advised in all laryngeal paralysis patients with severe clinical signs. The aim of surgery is to increase the size of the rima glottides. Cricoarytenoid cartilage lateralization is currently considered the procedure of choice. The objective of this procedure is to prevent passive adduction of the arytenoid cartilage during inspiration by fixing it to a neutral to slightly lateralized position (low tension technique). Unilateral cricoarytenoid lateralization has a good clinical outcome, with 88%-90% of dogs showing an improved quality of life in the postoperative period. Aspiration pneumonia is the most frequently noted complication, occurring in about 8%-24% of dogs postoperatively. Complications during the postoperative period can be minimized by sound knowledge of the anatomy, meticulous tissue handling and avoidance of laryngeal lumen penetration.

A clear distinction needs to be made between the different forms of the disease. Prognosis for hereditary laryngeal paralysis is excellent as dogs are cured by surgery. Congenital laryngeal paralysis neuropathy has a poor prognosis and most dogs tend to be euthanized within 10 weeks as a result of worsening clinical signs. The prognosis for acquired laryngeal paralysis will vary depending on the cause: trauma cases can be cured, while the prognosis for neoplasia-induced laryngeal paralysis will depend on the tumor type. Evidence strongly suggests that the most common form of laryngeal paralysis in dogs is, in fact, an early stage of geriatric onset laryngeal paralysis polyneuropathy. Even though all complications should be considered when giving a prognosis in any dog developing laryngeal paralysis as a component of polyneuropathy, this specific condition progresses slowly, with dogs reaching median survival times of 3-5 years after surgical correction.

References

Oropharyngeal stick trauma

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Oropharyngeal stick trauma refers to the development of a penetrating wound in the oropharynx when dogs are playing with wooden sticks. Since human selection has strengthened the urge to retrieve objects, playing with sticks is considered a fun exercise for both dogs and dog owners. But this exercise is not without risk. When dogs walk around with one end of a wooden stick in their mouth and the other end strikes an obstacle, injury to the oral mucosa can occur. These injuries can also occur when the dog jumps up to retrieve a thrown stick and catches one end in his mouth. But the most common situation is when the thrown stick anchors itself on the landing in the soil and the dog runs up to the other end with his mouth spread (high energy lesion).

Oropharyngeal stick injuries occur most commonly in large and giant breed dogs (because of the kinetic energy required to perforate the strong pharyngeal mucosa), and dogs are most often young (playful yet less experienced). The area and direction of penetration depends on the head stance at the moment of impact. The specific location of the injury will determine the clinical signs and deficits. Most occur in the lateropharyngeal or sublingual oropharyngeal region (damage possible from intermandibular to cranial thoracic regions). But also rostral pharyngeal (damage to orbital, retrobulbar and masseter and temporalis muscles) and dorsopharyngeal (damage to soft palate, retropharyngeal and esophagus) are encountered.

Acute symptoms include pain, blood in the saliva, dysphagia, anorexia, pain on opening the mouth, pain during flexion of the neck, coughing, exophthalmia, and even respiratory complaints. When the esophagus is lacerated subcutaneous emphysema, pneumomediastinum, and/or dyspnea can occur. When the initial trauma is not recognized or treated, this injury is considered chronic when the trauma occurred > 7 days before. The symptoms will then reflect the presence of a foreign object in the neck region, and include deep tissue abscessation, an intermittent or permanent draining tract, swelling of the neck region, dysphagia, ptyalism, lethargy, pain on opening the mouth, osteomyelitis of cervical vertebrae, discospondylitis, mediastinitis and myelopathy.

Although diagnosis in the acute presentation is relatively straightforward, preoperative medical imaging is helpful to determine the extent of trauma (thoracic involvement). Diagnosis consists of cervical and thoracic radiographs, combined with direct or endoscopic oropharyngeal inspection. In chronic cases the location of the cutaneous fistula not necessarily corresponds to the location of the residual foreign material, and medical imaging is essential to guide the surgical exploration. Fistulography, ultrasound examination and computed tomography are useful techniques.

Perforation of major arteries or the presence of a wooden stick obstructing the airway can result in a life-threatening situation. Lodged sticks that are visible on inspection are preferentially only removed during the surgical exploration to prevent unwanted further trauma from side branches or the activation of major bleeding when the counter pressure from the stick is omitted.

Surgical exploration to lavage the sinus tract and to remove foreign material (wood fragments, grass, sand) is imperative in every oropharyngeal stick injury. A rigid endoscope can be used to explore the perforating tract and remove debris by flushing tissues and grasping instruments through the working channel. But with
more extensive tissue trauma a ventral cervical exploration is performed. Placing a probe in the oropharyngeal perforating tract helps to guide the surgical dissection. Thorough knowledge of the regional anatomy is essential. The wooden stick or its fragments are removed (present in 30% of dogs), a swab for culture and sensitivity is taken, necrotic soft tissues are debrided, and the neck is closed leaving an active or passive drain for 24-48 hours.

Surgery for a chronic presentation is technically more difficult since chronic inflammation, adhesions and draining tracts change the local anatomy thereby increasing surgical morbidity. When no residual wooden fragments can be retrieved all draining tracts and inflammatory tissue needs to be removed.

The prognosis for acute oropharyngeal stick injuries is excellent. When a simple perforating defect in the esophagus has occurred this can be surgically closed. But a defect in the primary esophageal sphincter or an avulsion of the esophagus from the pharynx is associated with mortality rates of 15-50%. The chance of developing a chronic draining tract is 1%. Despite extensive surgical exploration in chronic cases, symptoms persist in nearly one-third of cases due to the presence of microscopic fragments in chronically inflamed tissues.

References


Artificial urethral sphincter for canine incontinence

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Urinary incontinence, the involuntary loss of urine during the filling phase of the bladder, is a commonly seen problem in veterinary practice. The involuntary loss of urine must be distinguished from behavioral problems, dysuria or polyuria. Urinary incontinence already occurring during puppyhood is indicative of a congenital malformation such as ectopic ureters, congenital urinary sphincter mechanism incompetence, persistent urachus, bladder diverticulum, bladder or urethral hypoplasia. If the first occurrence of urinary incontinence is noted after neutering, the most likely diagnoses are urinary sphincter mechanism incompetence and/or detrusor instability, but ureterovaginal fistula and ectopic ureters should still be considered. Ectopic ureter and urinary sphincter mechanism incompetence are responsible for up to 82% of urinary incontinence in dogs.

Urinary sphincter mechanism incompetence after spaying is the most common micturition disorder. In intact bitches and in male dogs, the risk for urinary incontinence is low (0%-1%), but in spayed bitches, the prevalence varies between 5%-20%. The first episode of urinary incontinence is usually observed 2-5 years after spaying, but may occur immediately or up to 10 years after surgery.

Medical therapy is successful in most cases independent of the age at spaying and the degree of incontinence. It is however very important that any underlying disease causing polyuria and/or a concomitant cystitis are diagnosed and correctly treated. In the majority of cases, history, physical examination and simple tests including urinalysis and urine bacterial culture lead to a presumed etiology. If urinary sphincter mechanism incompetence is the most likely cause, then the advantage of further diagnostic tests should be discussed with the owner before starting a trial therapy.

The first line of therapy is alpha-adrenergic agonists, which are commonly used to stimulate the alpha-adrenergic receptors expressed in the internal urethral sphincter, leading to an increase in the urethral closure pressure. The success rate varies from 86%-97% for phenylpropanolamine (1.5 mg/kg BW 1-3x/day). Interestingly, in large breed male dogs, where neutering may also favor the acquired form of urinary sphincter mechanism incompetence, conservative management is frequently disappointing, as only 44% respond satisfactorily to treatment with phenylpropanolamine. Estrogens sensitize the alpha-receptors and thus are suitable for a combined therapy with alpha-adrenergic agonists to potentiate their effects in females that are not fully responsive to phenylpropanolamine alone. Treatment with estriol (0.5-2 mg/dog/day PO for 5-7 days, then every other day and/or reduce dose every week to establish minimal effective dose for urinary incontinence after spaying) improved continence in 65-83% of treated dogs.

Potential side effects of this therapy have to be mentioned even though they rarely occur. It is important to thoroughly evaluate the success of the initial treatment. Its failure should lead to further diagnostic testing. Specialized clinical assessments may provide an etiological diagnosis, and this could serve as a basis for discussing further treatment options.

Although medical treatment has had good short-term efficacy, there is a high recurrence rate for urinary incontinence requiring revision procedures and additional drug therapy. Possible surgical treatments
are urethropexy, colposuspension, different sling techniques, and the submucosal endoscopic/laparoscopic/cystoscopic injection of urethral bovine cross-linked collagen, polyacrylamide hydrogel or hyaluronic acid. Although these have benefit many dogs (14–56%) remain incontinent, or revert to incontinence, despite treatment.

It is unlikely that any single treatment will cure 100% of dogs long term because the cause of refractory urinary incontinence is often complex and multifactorial. Refractory urinary incontinence, where traditional medical and surgical techniques fail to resolve urine leakage, is a problem in dogs with urinary sphincter mechanism incompetence, particularly after repair of ectopic ureters or other unique anatomic urogenital defects.

The use of a percutaneously controlled artificial urethral sphincter was first reported in dogs in 2004. The system consists of a silicone ring (hydraulic occluder) that is surgically placed around the proximal urethra. The ring is connected to a subcutaneous injection port with actuating tubing. Residual incontinence is treated with percutaneous infusion of sterile saline into the device to provide extraluminal urethral compression. When owner compliance is high and recommended inflations are performed, the functional continence rate is >90%, regardless of the underlying condition. Overall, the artificial urethral sphincter improves the continence scores in all dogs, which is why there is noncompliance with recommended inflations in some cases. This technique is associated with the major complication of urethral obstruction, and this should be considered a risk before artificial urethral sphincter placement. Because of the invasive nature of any surgical technique, this procedure is only recommended in dogs that are refractory to medical therapy or where medication is not tolerated. Careful monitoring of urine bacterial cultures, the subcutaneous infusion port site, and urination habits help to optimize success.

References

Subcutaneous ureteral bypass catheter for feline ureteral obstruction

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Feline ureteral obstructions are being recognized more frequently in recent years with the most common cause being an intraluminal obstruction secondary to ureteral calculi. Other causes include ureteral strictures, neoplasia, mucus plugs, dried solidified blood stones, fibrosis and surgical trauma. Circumcaval ureters are a rare embryological malformation resulting in ventral displacement of the caudal vena cava, which crosses the ureter, potentially causing a ureteral stricture.

The diagnosis of benign ureteral obstruction was made via abdominal ultrasonography, radiography, and ureteropyelography.

Ureteral obstruction secondary to ureterolithiasis in cats is a challenging situation and, historically, options for treatment of feline ureteral obstructions were limited. Medical treatment included parenteral administration of fluids, alpha blockade, and diuretics to promote urine production and passage of the ureteral calculus and supportive treatment for renal failure. Medical treatment, while non-invasive, is not usually successful in resolving the obstruction (8-17%). Twelve-month survival rates after medical treatment were 66%.

On the contrary, surgical intervention is more effective, with twelve-month survival rates of 91%. Ureteral calculi in the proximal portion of the ureter were typically removed by ureterotomy, whereas ureteral calculi in the distal portion of the ureter were more likely to be removed by partial ureterectomy and ureteroneocystostomy. Postoperative complication rate is 31%. The most common surgical complications include a uroabdomen from leakage at the surgical site, persistent or recurrent ureteral obstruction (from nephrolith migration, failure to remove all obstructive stones or surgical edema), or postsurgical stricture formation. The perioperative mortality rate has been reported to be 18-39% for cats with stone disease, with a number of cats dying of causes related to urinary tract disorders, including ureteral calculus recurrence and worsening of chronic renal failure.

More recently the placement of feline double pigtail ureteral stents or a subcutaneous ureteral bypass device has allowed for novel treatment options for cats with ureteral obstructions that failed medical management or have conditions difficult to address with traditional surgery. These devices have the potential to relieve obstructions while theoretically avoiding some of the complications seen with traditional surgical interventions. This can provide an option for patients with complicated obstructions, such as those with multiple ureteroliths (71%), proximal strictures (13%), concurrent nephroliths (15%) or non-resectable tumors.

Double pigtail ureteral stents placement, using endoscopic, open surgical, or both techniques, under fluoroscopic guidance, are successful in 95%. The perioperative morbidity and mortality rates were lower than those reported with traditional ureteral surgery. A 15% mortality rate exists before hospital discharge. Median survival times following discharge are 419 days (range, 44-994 days). Long-term management of ureteric stents is associated with a high rate of lower urinary tract signs with 35% of cats having signs consistent with sterile cystitis.

Subcutaneous ureteral bypass catheter placement is associated with a perioperative mortality rate of 7.5% and no deaths were procedure related. The median survival time was 498 days (range, 2 to > 1,278 days). Most of the 10% short-term and 33% long-term complications were minor and associated with intermittent dysuria or the need for ureteral stent exchange.
Ureteral obstructions in cats with a circumcaval ureter have a similar outcome to those cats with a ureteral obstruction and normal ureteral anatomy. Long-term prognosis is good for benign ureteral obstructions treated with a double pigtail stent or a subcutaneous ureteral bypass catheter device. The subcutaneous ureteral bypass catheter device re-obstructed less commonly than the ureteral stent, especially when a ureteral stricture was present.

Preoperative azotemia was present in 95% of cats, and 71% remained azotemic after successful surgery.

Hospitalization time was positively associated with presenting creatinine, perioperative complications, post-procedure creatinine and potassium. No parameters were associated with survival to discharge. A higher creatinine at discharge was positively associated with a higher creatinine at follow-up. A decreased overall survival was associated with a higher presenting blood urea nitrogen, higher creatinine at hospital discharge and in over-hydrated patients during hospitalization. Cats with ureteral obstruction(s) treated with a ureteral stent or subcutaneous ureteral bypass catheter device had an overall good survival and no admitting parameter was associated with survival to discharge. No single parameter was associated with all outcomes in this study, making predicting patient survival and cost prior to ureteral decompression difficult.

References


