Brachycephalic Airway Syndrome

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Brachycephalic airway syndrome (BAS) refers to clinical signs resulting from inherited anatomic abnormalities found in canine and feline breeds with brachycephalic skull conformation. These abnormalities include stenotic nares and an elongated soft palate as primary abnormalities and varied degrees of laryngeal collapse, ranging from eversion of laryngeal saccules to complete laryngeal collapse, as a secondary abnormality. In addition, some breeds present with tracheal hypoplasia, which may further exacerbate clinical signs but is not part of the syndrome. The term brachycephalic airway syndrome does not indicate the severity of clinical signs; rather, it represents a spectrum extending from mild increased upper respiratory noise to acute upper airway obstruction and resulting respiratory distress.

Brachycephalic animals need to generate greater negative airway pressure to overcome increased upper airway resistance at the level of the nares and nasopharynx to maintain adequate ventilation. Increased negative pressure leads to hyperplasia and inflammation of mucous membranes in the nasal and oral cavities and in the pharynx. The presence of redundant pharyngeal mucosa and elongation of the soft palate may be manifestations of these changes. High-velocity airflow through narrowed passages results in stridor, which is a high-pitched wheezing or whistling noise. Movement of an elongated soft palate over the rima glottidis creates the typical stertor or snoring sound. Chronically exaggerated negative inspiratory pressure can lead to weakening of laryngeal cartilages, facilitating laryngeal collapse.

Surgical correction of anatomic abnormalities is needed for definitive treatment. BAS has been documented to coincide with gastrointestinal (GI) disease, the treatment of which may result in an improved outcome regarding resolution of respiratory signs.

Age Predisposition
- Stenotic nares represent a congenital malformation of the nasal cartilages and are present at birth. An elongated soft palate may or may not be present at birth.
- Onset of clinical signs associated with BAS can be seen at any age, but the mean age of presentation is 2.5 to 3.5 years of age.
- English bulldogs tend to show clinical signs earlier than other breeds.
- Significant clinical signs related to BAS can be seen as early as 3 months of age.

Breed Predisposition
- Brachycephalic canine breeds: English bulldogs, French bulldogs, pugs, Cavalier King Charles spaniels, Boston terriers, Pekingese, boxers, Maltese, shih tzu, Lhasa apsos, bullmastiffs, Staffordshire terriers.
- Brachycephalic feline breeds: Persians, Himalayans.
- Presence of an elongated soft palate alone has been documented in many other mesocephalic and dolichocephalic breeds.

Owner Observations
- Stertor or stridor, snoring.
- Exercise intolerance, syncope, or collapse, often exacerbated by elevated ambient temperature.
- Tachypnea.
- Cyanosis.
- Dyspnea.
- Coughing, gagging.
- Vomiting, regurgitation, dysphagia.

Other Historical Considerations/Predispositions
- Presence of stenotic nares may be detected incidentally during routine pediatric physical examination.
- Mild increased upper respiratory noise may be considered “normal” by the owner.
Obesity is suspected to magnify clinical signs but has not been shown to contribute significantly.

**Physical Examination Findings**
- Stenotic nares are present in most cases.
  - Narrowed entrance into the nasal vestibule.
  - Minimal or absent abduction of alar folds or paradoxical adduction during inspiration.
- Cavalier King Charles spaniels may not present with stenotic nares.
- Stertor or stridor, especially during inspiration. Bronchovesicular sounds are often obscured by increased upper airway noise during thoracic auscultation.
- Obstructive breathing pattern: Marked inspiratory effort and prolonged inspiratory phase with a short expiratory phase.
- Hyperthermia: Body temperature above 105°F may represent heatstroke.
- Dyspnea, tachypnea, orthopnea.
- Cyanosis.
- Collapse.

**Laboratory Findings**
- Emphasis is placed on physical examination findings. Laboratory data are rarely needed for definitive diagnosis of BAS. The respiratory status of the patient has to be taken into consideration, and if respiratory distress is present, a patent airway should be ensured before laboratory samples are obtained and other diagnostic workup is performed.
- Polycythemia secondary to chronic hypoxemia may be seen.
- Arterial blood gas analysis:
  - Hypoxemia (partial pressure of arterial oxygen \([PaO_2] <85 \text{ mm Hg}\) with hypercapnia (partial pressure of arterial carbon dioxide \([PaCO_2] >45 \text{ mm Hg}\) indicating hypoventilation because of upper airway obstruction. The alveolar–arterial (A–a) gradient should remain normal in the absence of pulmonary parenchymal disease (A–a <10).
  - Respiratory, metabolic, or combined metabolic and respiratory acidosis (pH <7.44) resulting from hypoventilation or tissue hypoxia.
  - Serum lactate concentrations may be elevated because of tissue hypoxia.
- Although no abnormalities attributable to BAS are expected on complete blood count, serum chemistry profile, and urinalysis results, these tests may help rule out concomitant disease and aid in the preanesthetic evaluation of animals presenting with BAS.
- Coagulation profile and D-dimer analysis in severely hyperthermic animals can help identify complications attributable to heatstroke.

**ANESTHETIC PROTOCOLS ALLowing ACURATE EVALUATION OF LARYNGEAL FUNCTION IN STABLE PATIENTS**

The use of premedication has been shown to alter intrinsic laryngeal function if combined with IV induction of anesthesia. If possible, protocol 1 below should be used to rule out laryngeal paralysis. If sedation is necessary, protocol 2 appears to have no effect on arytenoid motion. Mask induction, however, may not be well tolerated by a healthy or struggling patient.

Combination of acepromazine plus thiopental, acepromazine plus propofol, and ketamine plus diazepam are not recommended because arytenoid motion is not always present in normal dogs anesthetized with these protocols. Using one of the following protocols has been shown to consistently allow accurate evaluation of laryngeal function.

**Protocol 1**
- Premedication: None.
- Induction: Thiopental 10–20 mg/kg IV given slowly to effect.
- Doxapram 1.1 mg/kg IV after initial evaluation of the larynx.

**Protocol 2**
This is used if sedation is necessary at any point before induction.
- Premedication: Glycopyrrolate 0.005–0.1 mg/kg IM, acepromazine 0.05–0.2 mg/kg IM, butorphanol 0.22–0.44 mg/kg IM, and metoclopramide 0.2–0.4 mg/kg IM or IV administered 20 minutes before anesthesia.
- Followed by preoxygenation for 5 minutes.
- Followed by mask induction with isoflurane in oxygen (use of sevoflurane has not been evaluated but may be preferable because of the shorter induction period needed).
- Doxapram 1.1 mg/kg IV after initial evaluation of the larynx.

**Other Diagnostic Findings**
- Thoracic radiography.
  - Elongated soft palate: The tip of the soft palate extends caudal to the tip of the epiglottis.
- Hypoplastic trachea may be present (ratio of tracheal lumen diameter at level of thoracic inlet to thoracic inlet diameter <0.16 for brachycephalic breeds).
- Secondary pulmonary parenchymal pathology or cardiac abnormalities may be present.
- Oxygen saturation via pulse oximetry may show hypoxemia (arterial oxygen saturation \([SaO_2] <92%)\. 
- Oral and laryngeal examination under light plane of anesthesia (see the box above for appropriate anesthetic protocols) should be done immediately.
before surgical procedures needed for definitive treatment. $

—Thought should be given to positioning of the animal to avoid distortion of oropharyngeal anatomic features. The oral examination should be performed with the patient in ventral recumbency. Excessive extension of the neck and traction on the tongue should be avoided. Gentle depression at the base of the tongue with a laryngoscope blade facilitates visualization of the rima glottidis and the relative length of the soft palate.

—Elongated soft palate: The tip of the soft palate extends caudally beyond the tip of epiglottis or beyond the caudal aspect of the tonsillar crypts. Alternatively, the epiglottis is displaced dorsally by the soft palate. The soft palate may be thickened and erythematous.

—Laryngeal collapse may be present. Stage I collapse includes eversion of the laryngeal sacculles with eversion of the laryngeal ventricles into the glottis to a varied degree. Everted sacculles emerge as round white masses from the laryngeal ventricles, obscuring the vocal cords. Stage II collapse is partial laryngeal collapse with medial displacement of the cuneiform processes of the arytenoid cartilages. In stage III collapse, the cuniculate processes of arytenoid cartilages collapse, and the dorsal arch of the rima glottidis is lost.

—Laryngeal function should be evaluated to rule out concurrent laryngeal disease not associated with BAS. Movement of the vocal cords and arytenoid cartilages should be normal in the absence of other laryngeal pathology.

—Redundant pharyngeal mucosal and tonsillar tissues may be present.

• Upper GI endoscopy: A high prevalence of GI lesions has been noted in dogs presenting for BAS, even in the absence of clinical signs directly attributable to GI disease. $$

—GI biopsies are indicated even if no gross lesions are visible on endoscopic evaluation. Gastric and duodenal biopsies should be obtained to evaluate for inflammatory changes in the GI tract.

—GI abnormalities include esophageal deviation, hiatal hernia, cardiac sphincter atony, gastroesophageal reflux, distal esophagitis, gastric hypomotility, pyloric mucosal hyperplasia, pyloric stenosis, duodenogastric reflux, diffuse gastritis, and diffuse duodenal inflammation.

**Summary of Diagnostic Criteria**

• Breed signalment paired with the presence of a certain history and clinical signs (varied degree of dyspnea, increased upper airway noise, obstructive breathing pattern) raises suspicion for the presence of BAS.

• Physical examination and anesthetized oral examination findings confirm the diagnosis.

• Ancillary diagnostic tests are not needed for a diagnosis of BAS but are helpful in detecting complicating disease.

• Patients can show signs of BAS at any age but commonly present at a young age.

• Severity of clinical signs dictates the diagnostic and treatment approach. Severe dyspnea necessitates immediate treatment without further diagnostic evaluation.

**Diagnostic Differentials**

• Concomitant or exacerbating laryngeal disease can be ruled out by visual inspection of the oral cavity and larynx and via thoracic radiography.

—Laryngeal paralysis: Failure of one or both vocal cords and arytenoid cartilages to abduct during the inspiratory phase.

—Laryngeal mass obstruction: Foreign body, neoplasia, granuloma.

—Trauma.

• Tracheal disease resulting in upper airway obstruction.

—Tracheal collapse: Ruled out by thoracic radiography or fluoroscopy or endoscopy of the upper airways.

—Tracheal mass obstruction: Foreign body, neoplasia, granuloma (ruled out by thoracic radiography or endoscopy of upper airways).

—Tracheal trauma.
• Concurrent lower airway disease (aspiration pneumonia, pulmonary edema, neoplasia): Ruled out by thoracic radiography.

TREATMENT RECOMMENDATIONS

Initial Treatment
The initial treatment approach varies greatly with the patient’s severity of clinical signs. Prompt treatment for severe dyspnea, respiratory distress, or acute complete upper airway obstruction is paramount.

• Oxygen supplementation should be done to accomplish 40% inspired oxygen concentration (an oxygen cage, nasal cannula, or mask can be used). §–$$

• Cooling should be done if the patient’s body temperature is above 105˚F; this treatment may be initiated by the owner before transport to the hospital because early cooling has been shown to increase survival in dogs presented with heatstroke. $

• Sedation of anxious but stable patients (drugs that tend to induce emesis should be avoided): $
  — Acepromazine 0.02–0.05 mg/kg IV or IM or acepromazine 0.022 mg/kg and butorphanol 0.22–0.44 mg/kg IM or IV.
  — Sedation can lead to relaxation of laryngeal muscles with resulting laryngeal collapse or worsening of the obstruction. Immediate induction and intubation may be indicated.
  — Induction of patients in respiratory distress: Glycopyrrolate 0.005 mg/kg IV and butorphanol 0.22 mg/kg IV followed by propofol 4–6 mg/kg IV to effect administered in increments of one quarter to one third of the calculated total dose.

• IV catheter placement and IV crystalloid fluid therapy as needed based on hydration status. $

• Metoclopramide to prevent emesis before and after surgery. The dosage is 0.2–0.4 mg/kg IM or IV initially followed by 0.2–0.4 mg/kg IV or IM q6h or 1 mg/kg/day IV constant-rate infusion. $

• Dexamethasone sodium phosphate 0.2–1 mg/kg IV administered once before surgery. This can be repeated after surgery as needed for complications associated with inflammation and edema. $

• If clinical signs are mild or moderate and immediate action does not need to be taken, the animal should be fasted for 24 hours before surgery to minimize the risk of aspiration pneumonia.

• The box on page 10 lists protocols that have shown to allow for accurate evaluation of laryngeal function to rule out the presence of laryngeal paralysis. In unstable patients, the luxury of tailoring a protocol for this purpose often cannot be afforded. The need for rapid induction to gain immediate airway access by intubation obviously takes precedence over accurate laryngeal evaluation. Intermittent positive-pressure ventilation may be needed, especially if pulmonary parenchymal dysfunction is present in addition to upper airway obstruction.

• Doxapram 1.1–2.2 mg/kg IV can be administered to stimulate respiration to allow accurate evaluation of laryngeal function.

Surgical Treatment $$$–$$$$

Correction of Stenotic Nares (Alaplasty)
• Naris stenosis is usually encountered bilaterally but can occur unilaterally in a few cases. Surgical correction aims at increasing the opening into the nasal vestibule and maintaining or creating symmetry. Alar wing wedge excision is the preferred method of correction. Subtotal removal of the alar wing has been described in cats with BAS.

• The patient is placed in sternal recumbency, and the nasal planum is prepared for aseptic surgery. Alar wing wedge excision consists of removal of a pyramid-shaped section of tissue that has the appearance of a wedge on the surface of the alar wing. The apex of the wedge represents a pivot point around which the free medial section of the wing is rotated laterally to increase the naris diameter. A #11 Bard-Parker blade is used to make a stab incision into the wing. The cutting edge of the blade is oriented medially, with the tip entering at the apex of the wedge and directed caudally (deep). The blade is removed and reintroduced, with the tip entering at the apex of the wedge and directed caudally (deep) to come to the same caudal point as before. For the second incision, the cutting edge is directed at an angle deviating slightly from the ventral direction, depending on the desired width of the base of the wedge. The angle at the apex of the wedge determines the amount of rotation allowed to the remaining medial aspect of the alar wing. The section of tissue—a pyramid with the caudal point forming the apex, two cut surfaces and intact nasal mucosa forming the faces, and the nasal planum forming the base—is removed.

• Hemorrhage is common and usually subsides with direct pressure and placement of sutures. The edges of the wedge are opposed by placing two to three simple interrupted sutures using 3-0 or 4-0 absorbable suture. A small half-circle cutting needle facilitates ease of placement.

Soft Palate Resection (Partial Staphylectomy)
• The patient remains in ventral recumbency. The maxilla is suspended from strips of tape anchored to a metal frame on each side. The mandible may be anchored to the table with tape to facilitate surgical exposure.

• The oral cavity is swabbed with an antiseptic. The patient remains in ventral recumbency. The soft palate is grasped, both the ventral and dorsal
aspects are swabbed, and resection levels are marked. Transient extubation is needed to evaluate for correct margins for soft palate resection. The tongue is relaxed, and the soft palate is marked where it touches the tip of the epiglottis at its midline. Some surgeons prefer the caudal dorsal margin of the tonsillar crypts as the caudal landmark for resection. Marking the soft palate can be done with a sterile surgical marker or a scalpel blade.

- The patient is reintubated, and the endotracheal tube cuff is inflated immediately after determination of surgical margins. The tube should be tied to the mandible to allow for better exposure of the soft palate. Placing a smaller-than-usual endotracheal tube can help to maximize exposure.

- Stay sutures are placed in the free border of the soft palate at its midpoint and at its lateral margins with 4-0 or 5-0 suture. An Allis tissue forceps can be used at the midpoint instead of sutures. Low tension is placed on the central suture or forceps and one lateral stay suture. An incision is made starting laterally and coursing toward the mark previously placed. The incised edge is closed with 4-0 or 5-0 absorbable suture. Needle bites penetrate the oral mucosa, exit through the cut edge of the soft palate ventral to the muscular layer, enter the cut edge dorsal to the muscular layer, and exit through the nasal mucosa. A simple, continuous pattern is used and should start very close (1 mm) to the lateral margin of the incision and bites 2 mm apart. The suture is continued medially for the length of the incision but not tied. The remainder of the palate is incised from its midpoint to the other side to complete the excision. The suture is continued for the length of the incision as already described.

- Use of clamps or electrocautery to achieve hemostasis is not usually needed. Electrothermal damage from electrocautery may exacerbate postoperative edema. Long-handled, curved instruments can help to maximize surgical exposure. Metzenbaum scissors or a scalpel can be used for sharp excision. Some surgeons prefer using a carbon dioxide laser for soft palate resection. Clinical results are similar for sharp mechanical and laser resection. If a laser is used, moist gauze sponges should be packed around the endotracheal tube.

- Blood remaining in the laryngeal area is removed by suction, and the endotracheal tube is removed to evaluate the length of the soft palate. Laryngeal saccul resection can be performed at this point if indicated. The endotracheal tube is replaced and remains in place as long as possible during recovery from anesthesia if laryngeal sacculae are not excised.

Laryngeal Saccule Resection (Laryngeal Ventriculectomy) If Needed

- Laryngeal sacculae represent edematous tissue lin-

- With the endotracheal tube removed, the saccul is grasped with an Allis tissue forceps and sharply excised at its base with curved Metzenbaum scissors. Injury to vocal fold tissue must be avoided. The contralateral saccul is removed in similar fashion.

- The endotracheal tube should be replaced immediately for recovery from anesthesia. The cuff can be placed at the level of the larynx and inflated to apply pressure on laryngeal tissues. Residual fluid and blood are removed with suction if needed.

Additional Surgical Information

- Redundant or everted tonsillar tissue is not routinely excised because it does not significantly contribute to obstruction of the upper airway.

- If stage III laryngeal collapse is present, a permanent tracheostomy may be required. Partial laryngectomy (partial arytenoidectomy) has a high postoperative mortality rate and is not recommended. If a permanent tracheostomy is performed, redundant skin folds should be removed to prevent them from covering the stoma.

- After the last surgical procedure is done, the endotracheal tube should be replaced and remain in place as long as possible during recovery from anesthesia. The patient should be recovered in a cool environment under close supervision to monitor for postoperative airway obstruction.

Alternative/Optional Treatments/Therapy

- Immediate induction of general anesthesia and intubation must be performed if sedation and oxygen supplementation do not result in improvement or if clinical signs worsen.

- A temporary tracheostomy tube may have to be placed if upper airway obstruction occurs after surgery. If laryngeal collapse was observed during the initial oral examination, performing a permanent tracheostomy is indicated at that time.

- Repeated doses of steroids (dexamethasone sodium phosphate, 0.2–1.0 mg/kg IV q24h) can be administered if complications are suspected because of upper airway inflammation or edema. Furosemide 2 mg/kg IV can be administered if soft tissue edema is severe.

- Glycopyrrolate 0.005 mg/kg IV, IM, or SC can be administered in an attempt to reduce upper airway secretion and accumulation of mucus. Intermittent suction of the oral cavity can be used as adjunct treatment. Insipssation of secretions can result from anticholinergic therapy.

- Weight should be managed in obese patients. Obe-
Appropriate treatment of GI disease in conjunction with surgical airway treatment has been shown to have an improved outcome in treating patients with BAS. A high incidence of gastritis or duodenitis in dogs with BAS has been shown. Generic treatment consists of proton pump inhibitors and prokinetic agents. This regimen should be modified as indicated by gross and histopathologic diagnosis:

- Omeprazole 0.7 mg/kg PO q24h for 2 to 3 months, depending on the degree of GI inflammation.
- Cisapride 0.2 mg/kg PO q24h for 2 to 3 months, depending on the degree of GI inflammation.
- Magnesium hydroxide 1 ml/kg PO bid after meals for 14 days if esophagitis is present.
- Sucralfate 0.5–1g PO tid administered 1 hour apart from meals and other medications for 2 to 3 months, depending on the degree of GI inflammation.
- Corticosteroid therapy should be initiated for severe GI inflammation. Therapy consists of prednisone 1 mg/kg/day (given in single dose or divided bid) initially and then tapered over time.

- In uncomplicated cases, cefazolin 22 mg/kg IV can be administered at the time of anesthetic induction. Administration is repeated every 90 minutes intraoperatively. In the presence of lower respiratory disease, broad-spectrum antibiotic therapy may be necessary. Second- and third-generation cephalosporins, fluoroquinolones, amikacin, and gentamicin can be used for empirical antibiotic therapy. Therapy should be changed based on identification of the causative agent and susceptibility testing.

- Premedication should include administration of metoclopramide 0.2–0.4 mg/kg IV or IM to reduce the risk of emesis and potential aspiration of gastric contents.

- Close postoperative monitoring for complications should be continued for 24 to 72 hours after surgery.

- Oxygen supplementation during the immediate postoperative period is recommended.

- Water may be offered after complete recovery from anesthesia. Food should be withheld for 24 hours after surgery.

- An Elizabethan collar should be applied to avoid self-trauma resulting in dehiscence of the sutures present in the nares.

- Appropriate treatment of concurrent GI disease should be initiated.

**Patient Monitoring**

- During postoperative recovery, the patient should be closely monitored for any signs of airway obstruction regardless of respiratory status before surgery. Monitoring should be continued for 24 to 72 hours after surgery.
  - Constant monitoring of respiratory frequency and pattern.
  - Intermittent pulse oximetry.
  - Blood gas analysis if indicated by respiratory pattern and frequency.

- Aspiration pneumonia can be seen in up to 9% of patients, with an even higher incidence reported in English bulldogs. Intermittent pulse oximetry, repeated arterial blood gas analysis, and thoracic radiology are valuable in detecting this complication.

- Patients experiencing heatstroke at the time of presentation should be monitored for development of disseminated intravascular coagulation for 48 hours after admission.

- Improvement of respiratory signs can be seen for up to 6 weeks after surgery; further improvement after that period is unlikely. If surgical results are unsatisfactory, reevaluation and surgical revision may be indicated.

- Sutures in the nasal planum should be removed 10 days postoperatively.
to 14 days after surgery. Sedation may be necessary to accomplish removal.

**Home Management**
- Heat stress should be avoided.
- Exercise should be restricted if exercise intolerance remains.
- A weight loss plan should be implemented if the animal is obese.
- Tracheostomy care should be implemented if a permanent tracheostomy tube has been placed.

**Milestones/Recovery Time Frames**
- Most patients are markedly improved immediately after surgery. Coughing and gagging are commonly seen in the immediate postoperative period.
- Although a majority of patients have improved clinical signs after surgical correction of abnormalities associated with BAS, mild clinical signs are likely to remain.
- Persistent stertor, stridor, or snoring is seen in more than 50% of cases.
- Exercise intolerance and episodes of dyspnea recur in up to 20% of cases.
- Improvement of clinical signs can occur for up to 6 weeks after surgery. If significant clinical signs remain, surgical revision may be needed. If laryngeal collapse was noted on initial examination, permanent tracheostomy tube placement may be needed. Arytenoid lateralization and partial laryngectomy have been attempted.

**Treatment Contraindications**
- Drugs that tend to cause emesis (e.g., morphine, hydromorphone, buprenorphine, oxymorphone, xylazine) should be avoided if at all possible to minimize the risk of aspiration pneumonia.
- Overzealous resection of the soft palate can lead to regurgitation of food and water into the nasopharynx and aspiration pneumonia.
- Tonsillectomy is not indicated, even if redundant tonsillar tissue is present.
- Severe concurrent disease such as pneumonia or cardiac disease can pose unacceptable anesthetic risks for complete upper airway surgery. If obstructive disease indicates immediate intervention, temporary tracheostomy placement with sedation or a very short-acting anesthetic should be performed until the patient is sufficiently stable for prolonged surgical procedures. Similarly, aspiration pneumonia or other pulmonary parenchymal disease may need to be addressed before corrective surgical intervention can be done.
- Doxapram should not be used for laryngeal evaluation in the presence of hypertension, suspected increased intracranial pressure, or seizures.

**PROGNOSIS**

**Favorable Criteria**
- Early correction of stenotic nares and an elongated soft palate likely prevents secondary mucosal and degenerative changes of the laryngeal cartilage. A correlation of age at the time of surgery with outcome has been shown in some (but not all) studies. Animals younger than 2 years of age tend to have fewer secondary changes of the upper airway, which presumably leads to a better outcome. Some surgeons have advocated correction of stenotic nares as early as at 3 to 4 months of age.
- The outcome of alaplasty performed concurrently with soft palate resection compares favorably to soft palate resection alone.
- Immediate improvement of respiratory signs after surgery likely indicates a good or excellent outcome.
- Concurrent treatment of GI inflammation or anatomic abnormalities potentially leads to a higher success rate for resolution or improvement of respiratory signs.

**Unfavorable Criteria**
- The presence of stage II or stage III laryngeal collapse has been shown to be associated with a less favorable outcome and higher mortality rates in some studies. The presence of laryngeal saccule eversion (stage I laryngeal collapse) may carry a poorer prognosis as well.
- The presence or development of aspiration pneumonia is associated with higher perioperative mortality.
- English bulldogs seem to have a higher incidence of poor outcome than all other breeds.
- Excessive amounts of mucus or regurgitation of mucoid material after surgery may necessitate placement of a temporary tracheostomy tube if laryngeal airway patency is severely compromised.
- Medical treatment with steroid therapy at antiinflammatory doses, weight loss, and exercise restriction is unlikely to result in favorable outcome. Surgical treatment is needed for definitive treatment of BAS.

**RECOMMENDED READING**


