Radiographic Diagnosis and Surgical Planning in Brachycephalic Obstructive Airway Syndrome: Comparative Case Reports in Pekingese, French Bulldog, and Pug

Hasti Abbasi[1] - Kian Kashani[1], *
[1] Doctor of Veterinary Medicine (DVM) graduate, Faculty of Veterinary Medicine, Islamic Azad University, Baft Branch, Iran
* (Corresponding author: kian.kashani@iau.ir)

ABSTRACT

Brachycephalic obstructive airway syndrome (BOAS) is a multifactorial disorder characterized by congenital upper-airway abnormalities, with clinical severity influenced by secondary changes such as laryngeal collapse. Although Bulldogs and Pugs dominate clinical reports, toy breeds such as the Pekingese are equally predisposed. Early surgical correction of primary lesions is critical to preventing progression and optimizing long-term outcomes. Three canine BOAS cases are described. Case 1 involved a 3.5-year-old Pekingese with progressive stertor and exercise intolerance, unresponsive to conservative therapy. Radiography confirmed soft-palate elongation and Grade I laryngeal collapse. Case 2, a 2-year-old French Bulldog, exhibited lifelong noisy breathing and exercise intolerance; imaging demonstrated congenital hypoplastic trachea with concurrent soft-palate elongation and stenotic nares. Case 3, a 5-year-old Pug, presented with severe inspiratory stertor, recurrent syncope, and gastrointestinal signs. Endoscopy confirmed elongated soft palate, stenotic nares, everted saccules, and Grade II laryngeal collapse. Survey radiography provided critical initial information in all cases. A tracheal-to-thoracic inlet ratio of 0.10 confirmed hypoplastic trachea in Case 2, while endoscopy established Grade II collapse in Case 3. Arterial blood gas analysis revealed hypercapnia and hypoxemia in Cases 2 and 3, guiding anesthetic planning. All cases underwent alar wedge rhinoplasty and staphylectomy (CO_2 laser in Cases 1 and 3, cold steel in Case 2). Case 3 also required bilateral sacculectomy. Perioperative management emphasized preoxygenation, cautious sedation, rapid airway control, and corticosteroids. Case 1 achieved full clinical resolution by 30 days. Case 2 improved substantially, though persistent stridor reflected fixed tracheal hypoplasia. Case 3 demonstrated reduced syncopal episodes but residual stridor due to Grade II collapse. All owners reported improved quality of life. These cases illustrate the diagnostic and prognostic value of imaging, the importance of early intervention, and the influence of concurrent anomalies on surgical outcome in BOAS.

Keywords: Brachycephalic obstructive airway syndrome; Pekingese; French Bulldog; Pug; hypoplastic trachea

1. INTRODUCTION

Brachycephalic obstructive airway syndrome (BOAS), also termed brachycephalic airway syndrome (BAS) is a constellation of primary anatomic abnormalities (most commonly stenotic nares and elongation/thickening of the soft palate) and secondary lesions (e.g., everted laryngeal saccules and varying degrees of laryngeal collapse) that together impose clinically significant upper-airway resistance in short-skulled dog breeds [1]. The resultant chronically elevated inspiratory negative pressure fosters a vicious cycle of tissue edema, mucosal trauma, and progressive dynamic collapse, thereby amplifying airflow obstruction over time. Clinical expression ranges from exercise intolerance and stertor/stridor to cyanosis and syncope,

and may be compounded by gastrointestinal comorbidities (regurgitation, esophagitis) that further compromise peri-anesthetic safety and convalescence [2]. Although Bulldogs, French Bulldogs, and Pugs dominate contemporary BOAS caseloads, toy brachycephalics, including the Pekingese, exhibit similar pathophysiology and risk. In all breeds, early recognition and timely surgical correction of primary lesions are emphasized to limit progression to laryngeal collapse, a secondary, staged condition associated with a worsened prognosis. Grade I collapse corresponds to eversion of the laryngeal saccules; Grade II reflects medial displacement/collapse of cuneiform cartilages; and Grade III denotes collapse of the corniculate processes with near-occlusion of the rima glottidis [3]. Grades II–III are linked to higher peri-operative complication rates and may necessitate more aggressive interventions (e.g., permanent tracheostomy) when standard upper-airway surgery is insufficient. Cranio-cervical imaging plays a pivotal role in comprehensive BOAS assessment and surgical planning. Survey radiography, while limited in depicting dynamic pharyngo-laryngeal behavior, remains valuable to: (1) screen for concurrent cardiopulmonary disease; (2) estimate vertebral heart size (VHS) to contextualize cardiomegaly suspicions; and (3) document surrogate markers of upper-airway obstruction such as elongated soft palate silhouette and narrowed laryngeal/ tracheal air columns. The canonical VHS reference in dogs (mean ≈ 9.7 ± 0.5 vertebrae on lateral views across mixed breeds) aids interpretation of mild cardiac silhouette enlargement and strengthens differential prioritization when respiratory signs predominate. Cross-sectional imaging, particularly computed tomography (CT), refines airway mapping by quantifying soft-palate length/thickness, nasopharyngeal and laryngeal luminal areas, and tracheal caliber; it can also reveal synchronous lesions (e.g., turbinate hypertrophy extending into the nasopharynx) and guide the extent of palatoplasty and rhinoplasty [4]. Dynamic or forced-expiratory imaging protocols can uncover airway segment collapsibility that may be occult on static studies, informing prognosis and postoperative expectations. Where accessible, CT therefore complements endoscopic and radiographic findings and enhances pre-operative strategy.

Surgical correction targets the reduction of fixed and dynamic resistance. Standard components include alar wedge rhinoplasty to enlarge stenotic nares and staphylectomy/palatoplasty to shorten and thin an elongated, bulky soft palate; sacculotomy is added when saccules are everted (Grade I laryngeal collapse). Technique selection for soft-palate resection CO_2 laser versus cold-steel or energy-sealing devices has not consistently demonstrated clinically meaningful differences in major complication rates or long-term outcomes, allowing centers to select based on surgeon experience, equipment, and hemostatic preference [5]. Nonetheless, CO_2 laser techniques offer precise cutting with excellent hemostasis and minimal intra-operative field contamination, which many surgeons find advantageous in a confined, highly vascular workspace. Peri-anesthetic risk mitigation is integral to BOAS case management. Pre-oxygenation, gentle premedication to reduce stress without precipitating profound hypoventilation or hypotension, rapid-control induction with prompt endotracheal intubation, and vigilant monitoring (ECG, SpO_2, capnography, temperature) are standard. Post-extubation airway edema and obstruction remain the most feared early complications; therefore, judicious peri-operative corticosteroid use, head elevation, quiet recovery, and ready access to re-intubation or emergency tracheostomy are recommended [6]. Dogs with advanced laryngeal collapse or severe concurrent gastrointestinal disease face higher complication rates and may require tailored plans and owner counseling regarding prognosis and potential need for staged or salvage procedures. Outcomes after corrective surgery are generally favorable when intervention occurs before end-stage laryngeal collapse. Across contemporary cohorts, most dogs experience meaningful reductions in respiratory noise and exercise intolerance, with corresponding improvements in owner-reported quality of life [7, 8]. Persisting or recurrent signs are often attributable to unaddressed secondary lesions, progression of laryngeal collapse, or obesity, an independent, modifiable risk factor that increases airway loading and thermal burden. Accordingly, structured weight-management and activity plans are considered integral adjuncts to surgical care. Within this context, a Pekingese case is described in which radiography played a central role in establishing elongation of the soft palate, narrowing of the laryngeal airway, and early (Grade I) laryngeal collapse, thereby guiding the selection of alar wedge rhinoplasty and CO_2-laser staphylectomy. To broaden clinical relevance, two additional closely related case studies are presented: (i) BOAS with hypoplastic trachea identified on imaging, with implications for anesthetic strategy and prognosis; and (ii) BOAS accompanied by Grade II laryngeal collapse, necessitating expanded surgical planning and postoperative precautions. Taken together, these cases underscore how careful imaging triage, breed-appropriate anesthetic planning, and timely, technique-agnostic soft-palate and nares correction align with current evidence to optimize outcomes in brachycephalic dogs.

2. Case studies

2.1.Case Presentation – Case 1: Pekingese

2.1.1. Signalment and Background

A 3.5-year-old intact male Pekingese dog, weighing 5.8 kg with a body condition score (BCS) of 6/9, was referred to a veterinary specialty clinic in Kerman, Iran. The referral was prompted by progressive upper respiratory noise and exercise intolerance over four weeks, with no improvement despite empirical oral antibiotic and non-steroidal anti-inflammatory therapy.

2.1.1.1.Presenting Complaints and History

According to the owner, the dog developed gradually worsening stertor during rest and mild exertion, with occasional inspiratory stridor. Episodes of exercise intolerance were consistently noted, limiting normal activity. No cyanosis or collapse had been observed at home.

2.1.1.2.Clinical Examination

On presentation, the dog was bright and responsive. Vital parameters were as follows: body temperature 38.9 °C, heart rate 110 beats/min, respiratory rate 38 breaths/min, and capillary refill time <2 seconds. Physical examination confirmed moderate inspiratory stertor, intermittent inspiratory stridor, and grade II stenotic nares. Indirect palpation of the oral cavity suggested elongation of the soft palate. No cyanosis, murmur, or abnormal lung sounds were detected.

2.1.1.3.Differential Diagnoses

Based on the signalment, history, and clinical findings, the following differential diagnoses were considered:

- Brachycephalic obstructive airway syndrome (BOAS)
- Laryngeal paralysis
- Nasopharyngeal stenosis
- Tracheal hypoplasia

2.1.1.4.Diagnostic Investigations

Survey thoracic and cranial radiographs (lateral and dorsoventral projections) revealed elongation of the soft palate by >1.5 cm beyond the tip of the epiglottis, narrowing of the laryngeal airway, and evidence of Grade I laryngeal collapse characterized by eversion of the laryngeal saccules. Vertebral heart size (VHS) was measured at 9.5, suggesting mild cardiomegaly. No pulmonary pathology was detected. Hematology revealed a mild neutrophilia without other abnormalities, while serum biochemistry values were within normal limits. Arterial blood gas analysis was not available; however, mild hypercapnia was suspected given the clinical signs. Advanced diagnostic imaging, including computed tomography of the skull and orthogonal thoracic radiographs, as well as abdominal ultrasonography, was recommended for further evaluation and surgical planning.

2.1.1.5.Owner Consent

Written informed consent for diagnostic evaluation, surgical intervention, and publication of clinical information and anonymized images was obtained from the owner.

2.2.Case Presentation – Case 2: French Bulldog with Hypoplastic Trachea

2.2.1. Signalment and Background

A 2-year-old spayed female French Bulldog, weighing 11.2 kg with a body condition score (BCS) of 5/9, was presented to the small animal surgery service of a referral hospital. The dog had a history of chronic noisy breathing since puppyhood, with progressive worsening over the previous six months. The owner reported exercise intolerance, loud stertor at rest, and occasional gagging after activity. The dog had not previously undergone surgical intervention for brachycephalic airway syndrome (BOAS) [9].

2.2.1.1.Presenting Complaints and History

The primary complaints included persistent inspiratory noise, heat intolerance, and reluctance to engage in play or prolonged walks. The dog had experienced one episode of syncope after vigorous activity during hot weather, which resolved spontaneously. No gastrointestinal signs such as regurgitation or vomiting were reported [3].

2.2.1.2. Clinical Examination

On physical examination, the dog was alert and in good body condition. Respiratory evaluation revealed severe inspiratory stertor, mild inspiratory stridor, and increased respiratory effort, particularly during excitement. Stenotic nares of grade II severity were present bilaterally. Oral inspection under light sedation indicated an elongated and thickened soft palate with intermittent epiglottic contact. Vital parameters included body temperature 39.1 °C, heart rate 120 beats/min, and respiratory rate 44 breaths/min. Mucous membranes were pink with a capillary refill time <2 seconds [10].

2.2.1.3. Differential Diagnoses

Differential diagnoses included:

- Brachycephalic obstructive airway syndrome (BOAS)
- Tracheal hypoplasia
- Laryngeal paralysis (less likely based on breed and presentation)
- Nasopharyngeal obstruction

2.2.1.4. Diagnostic Investigations

Thoracic radiographs in right lateral and dorsoventral views demonstrated a uniformly narrowed tracheal lumen with a tracheal-to-thoracic inlet ratio of 0.10, confirming hypoplastic trachea. The vertebral heart score (VHS) was 10.2, within the upper normal limit for this breed. Elongation of the soft palate was also visible, extending 1.8 cm caudal to the epiglottis. No pulmonary parenchymal changes were identified. A complete blood count and serum biochemistry were unremarkable. Arterial blood gas analysis revealed mild hypercapnia ($PaCO_2$ = 46 mmHg) and borderline hypoxemia (PaO_2 = 78 mmHg on room air). Computed tomography of the upper airway further characterized the hypoplastic trachea, with luminal narrowing extending from the larynx to the thoracic inlet. No evidence of dynamic tracheal collapse was observed [11].

2.2.1.5. Owner Consent

The owner provided written informed consent for diagnostic evaluation, surgical intervention, and use of anonymized clinical data and imaging for publication.

2.3. Case Presentation – Case 3: Pug with Grade II Laryngeal Collapse

2.3.1. Signalment and Background

A 5-year-old neutered male Pug, weighing 9.4 kg with a body condition score (BCS) of 7/9, was presented to a university referral hospital for evaluation of progressively worsening respiratory noise, exercise intolerance, and frequent episodes of gagging. The owner reported a two-year history of noisy breathing, with a marked deterioration during the past three months. Despite previous medical management with corticosteroids and environmental modification, clinical signs persisted and worsened [12].

2.3.1.1. Presenting Complaints and History

The dog's main complaints included severe inspiratory stertor, increased respiratory effort even at rest, and reduced ability to tolerate mild exertion such as climbing stairs. The owner described two recent syncopal episodes associated with excitement. Gastrointestinal signs were also reported, including occasional regurgitation and post-prandial retching [13].

2.3.1.2. Clinical Examination

Upon presentation, the dog was in mild respiratory distress with open-mouth breathing and pronounced inspiratory stertor. Vital parameters included body temperature 39.2 °C, heart rate 128 beats/min, and respiratory rate 52 breaths/min. Capillary refill time was approximately 2 seconds. Physical examination identified severe stenotic nares (grade III) and suspected elongation of the soft palate. Inspiratory stridor was consistently audible. The mucous membranes were pink without cyanosis at rest, but effort increased with minimal handling [14].

2.3.1.3. Differential Diagnoses

The main differentials considered included:

- Brachycephalic obstructive airway syndrome (BOAS) with progression to laryngeal collapse
- Laryngeal paralysis
- Nasopharyngeal stenosis
- Hypoplastic trachea (less likely given history and imaging findings)

2.3.1.4. Diagnostic Investigations

Survey radiographs of the thorax and skull revealed elongation of the soft palate extending 2.0 cm beyond the epiglottis, narrowing of the rima glottidis, and an increased soft tissue density within the laryngeal lumen. Vertebral heart size (VHS) was 9.8, within normal limits for the breed. No signs of pulmonary disease were detected. Endoscopic examination under light anesthesia confirmed an elongated and thickened soft palate, severe stenotic nares, and Grade II laryngeal collapse characterized by medial displacement and collapse of the cuneiform cartilages. Laryngeal saccules were everted bilaterally. A complete blood count and serum biochemistry profile were within normal ranges. Arterial blood gas analysis demonstrated mild hypoxemia (PaO_2 = 74 mmHg) and moderate hypercapnia ($PaCO_2$ = 52 mmHg). Computed tomography of the upper airway further delineated laryngeal collapse and excluded concurrent tracheal hypoplasia [15].

2.3.1.5. Owner Consent

The owner provided written informed consent for diagnostic evaluation, surgical treatment, and the use of anonymized case data and images for publication. Fig.1 illustrates the diagnostic outcomes of Brachycephalic Obstructive Airway Syndrome (BOAS) across Pekingese, French Bulldog, and Pug cases, highlighting breed-specific variations in clinical presentation.

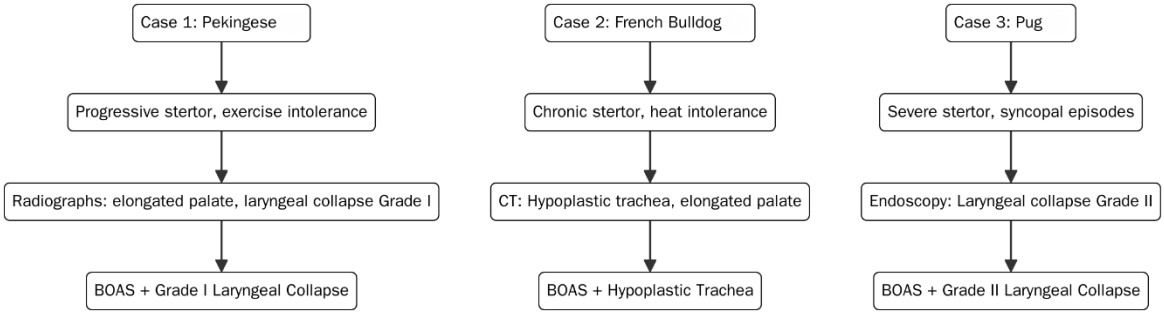

Fig. 1. BOAS diagnostic findings in Pekingese, French Bulldog, and Pug cases

3. Diagnostic Assessment

3.1. Case 1 Pekingese (BOAS with early laryngeal involvement)

3.1.1. Diagnostic reasoning and test selection

The signalment (brachycephalic toy breed), subacute progression of stertor/stridor, exercise intolerance, and lack of response to empirical antimicrobials/NSAIDs prioritized BOAS as the leading diagnosis, with laryngeal paralysis, nasopharyngeal stenosis, and tracheal hypoplasia as differentials. Initial aims were to (i) document primary BOAS lesions, (ii) stage any secondary laryngeal changes, and (iii) screen for concurrent cardiopulmonary disease that might confound anesthesia or mimic respiratory signs. Survey radiography was selected first because it is widely available, rapidly obtained without advanced anesthesia, and sufficient to screen the thorax, depict soft-palate length, and estimate cardiac silhouette size. Cross-sectional imaging (CT) was recommended for surgical planning and to quantify the degree of naso-/oropharyngeal narrowing. Still, it was not a prerequisite to proceed given the strength of clinical and radiographic evidence.

3.1.2. Imaging strategy and interpretation

Right lateral and DV radiographs of skull and thorax showed: (1) an elongated soft palate extending >1.5 cm caudal to the epiglottic tip; (2) a narrowed supraglottic airway with increased soft-tissue opacity consistent with early laryngeal obstruction; and (3) thoracic screens free of parenchymal disease. Vertebral

Heart Size (VHS) measured 9.5 vertebral bodies on the lateral view using the long- and short-axis method, indexed to the cranial edge of T4, consistent with at most mild cardiomegaly and unlikely to explain the primary respiratory noise. The aggregate radiographic pattern supported primary BOAS with early secondary change.

3.1.3. Endoscopy and dynamic assessment

Awake laryngoscopy is contraindicated; assessment under light, controlled anesthesia can clarify dynamic components. Given the clinical stability and apparent radiographic elongation of the soft palate, endoscopic staging was not mandated preoperatively; the working assumption was Grade I collapse (saccule eversion) based on luminal narrowing and history. Intraoperative/pharyngolaryngoscopic inspection was planned if needed.

3.1.4. Laboratory data and physiologic interpretation

CBC documented mild neutrophilia; serum biochemistry was unremarkable. Arterial blood gases were unavailable; given the clinical picture, mild chronic hypercapnia was considered plausible. The absence of cyanosis and the normal lung fields reduced concern for primary parenchymal disease.

3.1.5. Limitations and risk mitigation

Radiographs cannot capture pharyngo-laryngeal dynamics or quantify soft-palate thickness. Lack of ABG introduces uncertainty regarding the magnitude of gas-exchange impairment. These limitations were addressed by: (i) recommending CT for preoperative mapping, (ii) anticipating a potentially narrow airway during induction/recovery, and (iii) planning for immediate airway control (rapid intubation, perioperative steroids, and readiness for re-intubation) [16].

3.1.6. Diagnostic conclusion

Findings are most consistent with BOAS, characterized by an elongated soft palate and Grade I laryngeal collapse with contributory stenotic nares; cardiopulmonary screening did not reveal competing pathology. Imaging and clinical data justified corrective upper-airway surgery.

3.2. Case 2 French Bulldog (BOAS with hypoplastic trachea)

3.2.1. Diagnostic reasoning and test selection

Chronic noisy breathing from puppyhood, heat/exercise intolerance, and a brachycephalic phenotype supported BOAS. Early onset raised suspicion for a congenital fixed-airway component such as hypoplastic trachea (HT). The diagnostic goals were to (i) document standard BOAS lesions, (ii) confirm or refute HT, and (iii) characterize gas-exchange status to inform anesthetic risk. Radiography was prioritized because it can quantify tracheal caliber relative to the thoracic inlet. Given the potential for coexisting dynamic lesions, CT was planned to map the whole airway.

3.2.2. Imaging strategy and interpretation

Right lateral and DV thoracic views demonstrated a uniformly narrowed tracheal lumen from the larynx to the thoracic inlet. The tracheal-to-thoracic inlet ratio (minimum tracheal lumen diameter divided by the thoracic inlet diameter) was 0.10, which is markedly reduced and diagnostic for hypoplastic trachea in this clinical context. VHS was 10.2 (upper-normal for many brachycephalic breeds), and the lungs were clear. Skull views showed an elongated, thick soft palate extending 1.8 cm caudal to the epiglottis. CT confirmed diffuse reduction in tracheal caliber without focal collapse, supporting a fixed hypoplastic process rather than dynamic tracheal collapse [17].

3.2.3. Endoscopy and dynamic assessment

Because HT is fixed, dynamic imaging was less critical; however, laryngopharyngeal endoscopy remains useful to stage secondary lesions (e.g., saccule eversion) and assess edema. Given the respiratory reserve and the clear radiographic/CT diagnosis, endoscopic staging could be deferred to the anesthetic event for surgical correction.

3.2.4. Laboratory data and physiologic interpretation

CBC/chemistry were within reference limits. ABG showed mild hypercapnia ($PaCO_2$ 46 mmHg) and borderline hypoxemia (PaO_2 78 mmHg) on room air, consistent with increased upper-airway resistance. These values informed the plan for preoxygenation, controlled induction, and vigilant capnography.

3.2.5. Limitations and risk mitigation

Breed-specific VHS reference ranges vary; therefore, the cardiac silhouette was interpreted cautiously and in clinical context. The TTIR can be influenced by positioning and inspiratory effort; measurements were performed on well-positioned lateral images at peak inspiration to reduce error. The presence of HT increases peri-anesthetic risk and may blunt the magnitude of postoperative improvement compared to BOAS without HT. This was addressed in client counseling and perioperative planning (anticipating smaller endotracheal tubes and potentially prolonged recovery monitoring).

3.2.6. Diagnostic conclusion

BOAS with concurrent congenital hypoplastic trachea producing fixed extrathoracic airflow limitation, elongated soft palate, and stenotic nares. No evidence of dynamic tracheal collapse or primary pulmonary disease. These data support surgical correction of primary BOAS lesions with tailored anesthetic precautions for HT.

3.3. Case 3 Pug (BOAS with Grade II laryngeal collapse)

3.3.1. Diagnostic reasoning and test selection

Progressive inspiratory noise, exertional intolerance, and syncope in a middle-aged Pug with prior conservative management raised concern for advanced secondary airway changes. The diagnostic aims were to (i) verify primary BOAS lesions, (ii) stage laryngeal collapse, and (iii) exclude competing causes such as laryngeal paralysis or nasopharyngeal stenosis. Thoracic and skull radiography were obtained first to evaluate global airway anatomy and screen the lungs. Definitive staging required laryngoscopic/endoscopic assessment under controlled anesthesia.

3.3.2. Imaging strategy and interpretation

Radiographs revealed an elongated soft palate (~2.0 cm caudal to the epiglottis), a narrowed rima glottidis, and no pulmonary pathology. VHS measured 9.8 (not suggestive of clinically significant cardiomegaly). Increased supraglottic soft-tissue density suggested secondary laryngeal changes but could not stage them. CT excluded tracheal hypoplasia and provided spatial context for surgical planning.

3.3.3. Endoscopy and dynamic assessment

Under light, titrated anesthesia with spontaneous ventilation, endoscopy demonstrated: (i) elongated, thickened soft palate, (ii) severe stenotic nares, (iii) bilateral saccule eversion, and (iv) Grade II laryngeal collapse characterized by medial displacement/collapse of the cuneiform cartilages reducing the rima glottidis. Cord movement was present and symmetrical, making laryngeal paralysis unlikely. The endoscopic stage explained the patient's low exercise tolerance and collapses during excitement.

3.3.4. Laboratory data and physiologic interpretation

CBC/chemistry was unremarkable. ABG documented moderate hypercapnia ($PaCO_2$ 52 mmHg) and mild hypoxemia (PaO_2 74 mmHg) on room air, consistent with significant upper-airway obstruction and reduced ventilatory reserve, key for anesthesia risk stratification and postoperative planning.

3.3.5. Limitations and risk mitigation

Endoscopic staging can be influenced by the depth of anesthesia, as an excessive plane may artifactually reduce arytenoid abduction. To minimize this effect, a light plane with careful airway control was applied, and observations were correlated with radiographic or CT findings. Radiographs are unable to stage laryngeal collapse; therefore, their use was limited to screening rather than definitive diagnosis. Given Grade II collapse, there is an elevated risk of postoperative airway obstruction and edema; these risks were incorporated into the perioperative plan (perioperative corticosteroids, extended oxygen supplementation, readiness for re-intubation or temporary tracheostomy if required) and owner counseling about prognosis and the potential need for additional procedures if signs persist [18].

3.3.6. Diagnostic conclusion

BOAS with confirmed Grade II laryngeal collapse, elongated soft palate, and severe stenotic nares, without tracheal hypoplasia or primary pulmonary disease. The staging justifies comprehensive upper-airway surgery and heightened perioperative precautions, with guardedly optimistic expectations regarding return to complete exercise tolerance given the advanced laryngeal changes. Table 1 summarizes the signalment, clinical findings, and diagnostic imaging results of the three BOAS cases, facilitating comparative interpretation before individual case analyses.

Table 1. Summary of Diagnostic Findings

Parameter	Case 1: Pekingese	Case 2: French Bulldog	Case 3: Pug
Age/Sex/Weight/BCS	3.5 yr/M/5.8 kg/BCS 6/9	2 yr/F/11.2 kg/BCS 5/9	5 yr/M/9.4 kg/BCS 7/9
Primary Signs	Stertor, stridor, exercise intolerance	Chronic noisy breathing, syncope	Severe stertor, stridor, syncope, GI signs
Radiographic Findings	Elongated soft palate (>1.5 cm), Grade I collapse, VHS 9.5	Hypoplastic trachea (TTIR = 0.10), elongated soft palate, VHS 10.2	Elongated soft palate (2.0 cm), supraglottic narrowing, VHS 9.8
CT Findings	Not performed	Confirmed hypoplastic trachea	Ruled out tracheal hypoplasia, confirmed collapse
Endoscopic Findings	Not performed (assumed Grade I)	Deferred pre-op	Grade II collapse, everted saccules
Blood Gas Analysis	Not available (suspected mild hypercapnia)	$PaCO_2$ 46 mmHg, PaO_2 78 mmHg	$PaCO_2$ 52 mmHg, PaO_2 74 mmHg
Final Diagnosis	BOAS with an elongated soft palate, Grade I collapse	BOAS + hypoplastic trachea + elongated palate	BOAS with Grade II collapse, elongated palate

4. Therapeutic Intervention

4.1. Case 1 Pekingese (BOAS with Grade I laryngeal collapse)

4.1.1. Anesthetic protocol and perioperative monitoring

Given the breed-specific airway risks, preoxygenation for 5 minutes was performed before sedation. Premedication consisted of butorphanol (0.2 mg/kg IM) combined with acepromazine (0.02 mg/kg IM), the latter used at a conservative dose to minimize the risk of hypotension. Induction was achieved with titrated propofol to effect, permitting rapid, atraumatic endotracheal intubation. Maintenance was with isoflurane in 100 % oxygen. Analgesia was supported intraoperatively with an IV bolus of fentanyl (2 µg/kg) followed by a CRI (2–5 µg/kg/hr). Comprehensive monitoring included ECG, pulse oximetry, capnography ($ETCO_2$), invasive arterial blood pressure, and body temperature. Perioperative dexamethasone (0.1 mg/kg IV) was administered to mitigate laryngeal edema. Emergency equipment for re-intubation or temporary tracheostomy was prepared.

4.1.2. Surgical procedure

Stenotic nares were corrected via alar wedge resection with a cold steel scalpel, excising a full-thickness triangular wedge from the lateral alar fold bilaterally, followed by meticulous mucocutaneous apposition with 4-0 monofilament absorbable sutures. The elongated soft palate was shortened and thinned using a CO_2 laser staphylectomy technique. The CO_2 laser allowed precise resection with minimal hemorrhage and thermal spread. Grade I laryngeal collapse did not necessitate further intervention beyond sacculectomy, which was deferred given minimal obstruction at the time of surgery [19].

4.1.3. Postoperative management

The dog was extubated only after full consciousness, with supplemental oxygen and head elevation provided during recovery. Analgesia consisted of buprenorphine (0.01 mg/kg SC q8h) for three days. Dexamethasone was continued for 24 hours to reduce airway edema. Prophylactic antibiotics (amoxicillin–

clavulanate 20 mg/kg PO q12h) were prescribed for 7 days. Strict monitoring in the ICU for 24 hours included continuous SpO_2, frequent respiratory scoring, and readiness for emergency airway intervention.

4.2. Case 2 French Bulldog (BOAS with hypoplastic trachea)

4.2.1. Anesthetic protocol and perioperative monitoring

Hypoplastic trachea markedly increases intubation difficulty and recovery complications. The dog was preoxygenated for 10 minutes. Sedation included butorphanol (0.2 mg/kg IM) alone to avoid excessive cardiorespiratory depression. Induction was achieved with propofol (titrated IV) while preparing multiple sizes of cuffed endotracheal tubes smaller than predicted for weight. Isoflurane in oxygen was used for maintenance. Ventilation was assisted to maintain normocapnia. Monitoring included ECG, SpO_2, capnography, invasive blood pressure, and temperature. Arterial blood gases were sampled intermittently to guide ventilation. Emergency tracheostomy equipment was readied due to the fixed narrowing of the trachea.

4.2.2. Surgical procedure

Stenotic nares were corrected via alar wedge resection as described in Case 1. The elongated soft palate was resected and thinned using cold steel dissection with bipolar cautery for hemostasis, selected because laser facilities were not available. Given the diagnosis of diffuse hypoplastic trachea, no surgical correction was feasible for that component. The surgical plan focused on optimizing rostral airway patency to reduce overall inspiratory load.

4.2.3. Postoperative management

The dog was extubated under deep monitoring and observed in an oxygen-enriched ICU cage. Analgesia was achieved with a fentanyl CRI (2 µg/kg/hr) transitioned to buprenorphine (0.01 mg/kg SC q8h). Dexamethasone (0.1 mg/kg IV once) was administered perioperatively. Because HT increases the risk of incomplete clinical resolution, the owner was counseled regarding guarded prognosis, the need for weight management, and possible persistent respiratory noise.

4.3. Case 3 Pug (BOAS with Grade II laryngeal collapse)

4.3.1. Anesthetic protocol and perioperative monitoring

This patient presented with significant compromise and Grade II collapse, warranting maximal caution. Premedication included a low-dose opioid (butorphanol 0.2 mg/kg IM) without sedatives that risk excessive hypotension or respiratory depression. Preoxygenation was extended to 15 minutes. Induction was performed with propofol titrated IV, with rapid endotracheal intubation. Isoflurane in oxygen maintained anesthesia. A lidocaine bolus (2 mg/kg IV) was administered at intubation to blunt airway reflexes and reduce laryngospasm risk. Continuous monitoring included ECG, SpO_2, $ETCO_2$, invasive arterial pressure, esophageal temperature, and frequent arterial blood gas sampling. A temporary tracheostomy kit was prepared at the bedside, anticipating possible airway obstruction on recovery.

4.3.2. Surgical procedure

Bilateral alar wedge rhinoplasty was performed with cold steel excision and sutured mucocutaneous apposition. The soft palate was resected using CO_2 laser palatoplasty with care to thin redundant tissue without over-shortening. Bilateral laryngeal sacculectomy was performed due to marked eversion. Although Grade II collapse often carries a guarded prognosis, arytenoid lateralization or permanent tracheostomy were not pursued at this stage, as airflow was expected to improve after primary corrections. Owner consent for possible staged salvage procedures was obtained.

4.3.3. Postoperative management

The dog was extubated in the ICU with oxygen supplementation and close observation. Dexamethasone (0.1 mg/kg IV q12h for 24 hours) was administered for airway edema control. Analgesia was provided with buprenorphine (0.01 mg/kg SC q8h). Broad-spectrum antibiotics were initiated (amoxicillin–clavulanate 20 mg/kg PO q12h). The patient was maintained in sternal recumbency with head elevation. Given the advanced laryngeal changes, monitoring was extended to 48 hours with immediate access to re-intubation or tracheostomy equipment. Table 2 presents a consolidated overview of surgical approaches, anesthetic protocols, and 30-day postoperative outcomes for all three cases.

Table 2. Surgical and Postoperative Management Summary

Parameter	Case 1: Pekingese	Case 2: French Bulldog	Case 3: Pug
Surgical Procedures	Alar wedge rhinoplasty, CO_2 laser staphylectomy	Alar wedge resection, cold steel staphylectomy	Alar wedge, CO_2 laser palatoplasty, sacculectomy
Anesthesia Protocol	Butorphanol + acepromazine, propofol induction	Butorphanol only, propofol induction	Butorphanol, propofol, and lidocaine IV at induction
Steroid Use	Dexamethasone 0.1 mg/kg IV periop	Dexamethasone 0.1 mg/kg IV once	Dexamethasone 0.1 mg/kg IV q12h x 24h
Pain Management	Buprenorphine SC q8h x 3 days	Fentanyl CRI → buprenorphine	Buprenorphine SC q8h
Complications/Challenges	None	Persistent mild stridor post-op	Initial recovery difficulty, persistent stridor
30-Day Outcome	Full resolution, improved exercise/sleep	Improved tolerance, persistent stridor	Reduced syncope, improved QOL, residual stridor
Prognosis	Excellent	Fair to guarded (due to hypoplastic trachea)	Guarded (due to Grade II collapse)

5. Follow-up and Outcomes

5.1. Case 1 Pekingese (BOAS with Grade I laryngeal collapse)

5.1.1. Immediate postoperative period

The patient recovered smoothly from anesthesia without evidence of airway obstruction, aspiration, or hypoxemia. Continuous ICU monitoring for 24 hours showed stable SpO_2 (>95 % on room air) and normocapnia. Mild transient swelling at the nares resolved within 12 hours. Analgesia with buprenorphine (0.01 mg/kg SC q8h) effectively controlled discomfort, as indicated by Glasgow Composite Pain Scale (CMPS-SF) scores ≤ 4/24.

Short-term follow-up (7 days)

At re-examination, inspiratory stertor had diminished markedly. Exercise tolerance improved; the patient was able to walk 10–15 minutes without distress. No postoperative complications such as hemorrhage, aspiration pneumonia, or dehiscence were observed. The surgical sites (nares and soft palate) showed appropriate healing.

Medium-term follow-up (30 days)

The owner reported normalization of exercise capacity, absence of stridor, and improved quality of sleep. Physical examination confirmed a widely patent nasal aperture and reduced upper airway noise. No recurrence of signs was noted. The owner was satisfied with the clinical outcome. A structured weight-loss plan (target 10 % reduction over 2 months) was initiated to decrease airway loading further.

5.2. Case 2 French Bulldog (BOAS with hypoplastic trachea)

5.2.1. The immediate postoperative period

Extubation was uneventful, though prolonged monitoring was required due to the risk of airway compromise from hypoplastic trachea. SpO_2 remained >94 % with intermittent oxygen supplementation. Analgesia (fentanyl CRI transitioned to buprenorphine) maintained Glasgow CMPS scores ≤ 5/24. Mild inspiratory stridor persisted immediately after surgery but was less pronounced than preoperatively.

Short-term follow-up (7 days)

Clinical reassessment showed a significant reduction in stertor at rest. Exercise tolerance improved but remained limited compared to non-brachycephalic dogs. The surgical sites healed without complication. Persistent moderate airway noise was attributed to fixed hypoplastic trachea.

Medium-term follow-up (30 days)

The owner reported marked improvement in quality of life: the dog could tolerate routine walks without collapse, though heavy exertion continued to elicit mild stridor. No syncopal episodes occurred during follow-up. Prognosis was explained as fair to guarded due to the permanent nature of the hypoplastic trachea. Long-term weight management and avoidance of strenuous activity or heat stress were emphasized.

5.3. Case 3 Pug (BOAS with Grade II laryngeal collapse)

5.3.1. Immediate postoperative period

Recovery was protracted with episodes of increased inspiratory effort in the first 12 hours, controlled by supplemental oxygen and a single rescue dose of dexamethasone. SpO_2 fluctuated between 92–96 % during the first night but stabilized at>95 % by 24 hours. Pain management with buprenorphine maintained CMPS-SF scores ≤ 6/24. No emergency tracheostomy was required.

Short-term follow-up (7 days)

The patient exhibited partial clinical improvement: reduced stertor at rest and improved ability to walk short distances. However, inspiratory stridor persisted with moderate exertion. Surgical sites healed without complication. Endoscopic reevaluation was not performed at this stage, but was discussed as a future option.

Medium-term follow-up (30 days)

The owner reported a substantial reduction in the frequency of syncopal events (none observed during the 30 days), improved exercise tolerance indoors, and better quality of sleep. Nevertheless, inspiratory stridor remained noticeable during outdoor exertion, consistent with residual obstruction due to Grade II collapse. Prognosis was discussed as guarded, with the potential need for future staged interventions (e.g., permanent tracheostomy) if signs progress. The owner expressed satisfaction with the improvement achieved, though aware of ongoing limitations.

6. Discussion

Brachycephalic obstructive airway syndrome (BOAS) remains one of the most clinically significant upper respiratory disorders of canine patients, with its severity influenced by both primary anatomic abnormalities and secondary dynamic changes. The three cases presented illustrate the spectrum of BOAS pathology, ranging from early disease limited to soft-palate elongation with minor laryngeal involvement (Case 1), to syndromic presentation complicated by congenital hypoplastic trachea (Case 2), and finally to advanced disease characterized by Grade II laryngeal collapse (Case 3). Diagnostic and therapeutic considerations influencing both short- and long-term outcomes are demonstrated collectively [20].

6.1. Diagnostic considerations

Radiography remains a valuable, accessible tool for documenting soft-palate elongation, screening the thorax, and estimating vertebral heart score (VHS). In Case 1, radiographs alone provided sufficient evidence of soft-palate elongation and early laryngeal collapse to justify surgical intervention. Case 2 illustrated the utility of thoracic radiography in quantifying tracheal caliber, confirming hypoplastic trachea by a tracheal-to-thoracic inlet ratio of 0.10. In Case 3, radiography suggested supraglottic soft-tissue thickening but could not stage laryngeal collapse; endoscopy was required to confirm Grade II collapse [21]. Thus, while radiography is indispensable for screening, definitive staging of BOAS requires dynamic airway evaluation via endoscopy and, where available, cross-sectional imaging (CT). Blood gas analysis, performed in Cases 2 and 3, provided additional insight into ventilatory compromise and facilitated anticipation of anesthetic risk. In all cases, the integration of imaging and physiologic data enabled rational surgical planning and informed case management decisions.

6.2. Therapeutic strategies

Corrective surgery in all three patients targeted the two principal obstructive lesions: stenotic nares and elongated soft palate. Both cold-steel and CO_2 laser staphylectomy techniques were employed. The laser

offered excellent hemostasis and precision (Cases 1 and 3), while cold-steel dissection with bipolar cautery (Case 2) was adequate in the absence of laser facilities. Outcomes across these cases suggest that while the surgical technique may influence intraoperative efficiency, overall improvement depends more on timing of intervention and severity of secondary airway changes than on instrument selection [22]. Management of concurrent lesions remains critical. In Case 2, hypoplastic trachea could not be surgically corrected; this congenital abnormality imposes a permanent reduction in airway caliber, thereby limiting the degree of symptomatic improvement achievable with standard BOAS surgery. In Case 3, Grade II laryngeal collapse represented progression beyond simple saccule eversion. While partial improvement followed correction of primary lesions, residual inspiratory stridor persisted, highlighting that advanced collapse carries a guarded prognosis and may eventually necessitate salvage procedures such as permanent tracheostomy.

6.3. Outcomes and prognostic factors

Case 1 demonstrated the most favorable outcome, with near-complete resolution of clinical signs at 30 days. This underscores the importance of early surgical intervention before progression to advanced laryngeal collapse. Case 2 improved meaningfully, but persistent airway noise reflected the non-correctable contribution of hypoplastic trachea; prognosis was fair to guarded. Case 3 showed a reduction in syncope and partial improvement in tolerance, but residual stridor emphasized that once Grade II collapse develops, outcomes are inherently limited despite technically successful surgery. Across all cases, perioperative risk was managed successfully by careful anesthetic planning: preoxygenation, judicious sedation, rapid airway control, intraoperative monitoring, and prophylactic corticosteroid administration. Importantly, no patient required emergency tracheostomy, although preparedness was essential. Weight management was emphasized in Cases 1 and 2 and was particularly relevant in Case 3, where excess body condition (BCS 7/9) likely exacerbated inspiratory load. Obesity has been repeatedly shown to worsen BOAS outcomes and should be addressed as part of comprehensive management.

6.4. Clinical implications

These cases illustrate several practical lessons:

- Early recognition and intervention are key to preventing progression to irreversible laryngeal collapse.
- Radiography provides essential baseline information, but endoscopy and CT refine diagnosis and surgical planning.
- Concurrent abnormalities (hypoplastic trachea, advanced laryngeal collapse) profoundly influence outcome and must be incorporated into client counseling.
- Peri-anesthetic strategies tailored to brachycephalics preoxygenation, airway preparedness, and steroid prophylaxis are critical for safe management.
- Owner education on weight control, environmental management, and realistic expectations is central to long-term quality of life.

6.5. Limitations

These reports are limited by incomplete access to specific advanced diagnostics (e.g., arterial blood gases in Case 1, routine CT in Cases 1 and 3), which could have provided more objective quantification of disease severity. Nevertheless, each case demonstrates decision-making grounded in the best available evidence, supplemented by intraoperative findings and follow-up assessments.

7. Conclusion

These three cases underscore the spectrum of clinical presentations and outcomes in brachycephalic obstructive airway syndrome (BOAS). Early-stage disease, exemplified by the Pekingese with Grade I laryngeal collapse, responds favorably to corrective rhinoplasty and staphylectomy, with near-complete restoration of exercise tolerance. In contrast, concurrent congenital abnormalities such as hypoplastic trachea (French Bulldog) and advanced secondary changes such as Grade II laryngeal collapse (Pug) limit the degree of clinical improvement achievable despite technically successful surgery. Radiography remains indispensable for initial diagnosis and surgical planning, while endoscopy and computed tomography provide essential staging and refinement of therapeutic strategy. Careful anesthetic management, perioperative monitoring, and owner education, including weight control and long-term expectations, are integral to optimizing outcomes. In

summary, timely surgical intervention tailored to the severity of airway compromise, supported by comprehensive diagnostic assessment and meticulous perioperative care, offers the best opportunity to improve quality of life in brachycephalic dogs.

References

1. Krainer, D. and G. Dupré, Brachycephalic obstructive airway syndrome. Veterinary Clinics: Small Animal Practice, 2022. **52**(3): p. 749-780.
2. Wallace, M.L., Surgical management of brachycephalic obstructive airway syndrome: An update on options and outcomes. Veterinary Surgery, 2024. **53**(7): p. 1173-1184.
3. Bottero, E., et al., Clinical Evaluation and Systematic Classification of Endoscopic Gastrointestinal Findings in 176 French Bulldogs with Brachycephalic Airway Obstructive Syndrome. Animals, 2025. **15**(14): p. 2137.
4. Cook, L., T. Hallowell, and J. Sun, Retrospective evaluation of the outcomes of advanced airway management in intubated patients with brachycephalic obstructive airway syndrome: 52 cases (2010-2020). Journal of Small Animal Practice, 2025.
5. Åsbjer, E., Å. Hedhammar, and K. Engdahl, Awareness, experiences, and opinions by owners, breeders, show judges, and veterinarians on canine Brachycephalic Obstructive Airway Syndrome (BOAS). Canine Medicine and Genetics, 2024. **11**(1): p. 3.
6. Fitzmaurice, M., et al., Static respiratory compliance in anaesthetised and intubated brachycephalic dogs with and without brachycephalic obstructive airway syndrome. The Veterinary Journal, 2025: p. 106372.
7. Rein, A., et al., Laryngeal and pleural ultrasound and acoustic radiation force impulse elastography in dogs with brachycephalic obstructive airway syndrome. Scientific Reports, 2025. **15**(1): p. 19884.
8. Dimopoulou, M., et al., Use of respiratory signal analysis to assess severity of Brachycephalic Obstructive Airway Syndrome (BOAS) in dogs. The Veterinary Journal, 2024. **308**: p. 106261.
9. Yoshida, S., et al., Vocal fold granuloma associated with brachycephalic obstructive airway syndrome in 13 dogs. Journal of the American Veterinary Medical Association, 2025. **263**(6): p. 786-790.
10. Williams, P.J., C. De Gennaro, and J.L. Demetriou, Evaluation of the addition of adrenaline in a bilateral maxillary nerve block to reduce hemorrhage in dogs undergoing sharp staphylectomy for brachycephalic obstructive airway syndrome. A prospective, randomized study. Veterinary Surgery, 2024. **53**(1): p. 67-74.
11. Tamburro, R., et al., Histomorphological evaluation of thermal injury following palatoplasty performed with CO2 laser or LigaSure device in dogs with brachycephalic obstructive airway syndrome. The Veterinary Journal, 2024. **307**: p. 106195.
12. Cardoso, D.R., et al., Oral focal mucinosis in a dog with brachycephalic obstructive airway syndrome. Veterinary Record Case Reports, 2025. **13**(2): p. e70030.
13. Zibetti, F.L., et al., Arrhythmogenic Right Ventricular Cardiomyopathy in English Bulldog with Brachycephalic Obstructive Airway Syndrome (BOAS). Acta Scientiae Veterinariae, 2025. **53**.
14. Siddiqui, M., et al., Canine Brachycephalic Obstructive Airway Syndrome (CBOAS)-Diagnosis and Non-Surgical Management. 2024.
15. Vrijsen, E., et al. Topical application of epinephrine on everted laryngeal saccules as an alternative to surgical resection in dogs with brachycephalic obstructive airway syndrome. in BSAVA. 2024.
16. Cooper, L.M., J.A. Grimes, and A.M. Davros, The brachycephalic risk (BRisk) score's predictability for major postoperative complications varies between breeds undergoing brachycephalic obstructive airway surgery. Journal of the American Veterinary Medical Association, 2025. **1**(aop): p. 1-6.
17. Kravchenko, S., et al., A case of brachiocephalic syndrome in a French bulldog. Ukrainian Journal of Veterinary and Agricultural Sciences, 2024. **7**(3): p. 37-41.
18. Anyamaneecharoen, T., et al., Quantitative assessment of preoperative and postoperative brachycephalic airway obstruction syndrome surgery using clinical grading questionnaires and 6-minute walk test in French bulldogs. The Thai Journal of Veterinary Medicine, 2024. **53**(4): p. 495-503.
19. Vicenti, C., et al., Respiratory Effects of Continuous Positive Airway Pressure Administered during Recovery from General Anesthesia in Brachycephalic Dogs. Veterinary Sciences, 2024. **11**(2): p. 75.
20. Mostafa, A.A., K. McCutcheon, and C.R. Berry, Comparison of tracheal diameter in non-brachycephalic versus brachycephalic dogs using manubrium and thoracic inlet tracheal indexes. Frontiers in Veterinary Science, 2024. **11**: p. 1454930.
21. McDonald, A., et al., Neural network analysis of pharyngeal sounds can detect obstructive upper respiratory disease in brachycephalic dogs. Plos one, 2024. **19**(8): p. e0305633.
22. Araos, J., et al., Effect of continuous positive airway pressure helmet on respiratory function following surgical procedures in brachycephalic dogs: A randomized controlled trial. Veterinary Surgery, 2024. **53**(5): p. 872-880.