Philip Moses Memorial Lecture – Part 1: Review of brachycephaly and updates on treatment of brachycephalic obstructive crisis

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Brachycephaly is the name given to a collection of anatomic changes present in dog breeds with shortened muzzles/faces and a reduced cranio-facial ratio. There has been a marked increase in the popularity of brachycephalic dog breeds (particularly French bulldogs, pugs and English bulldogs) in the past decade, and despite increased awareness regarding the respiratory (and other) problems these breeds face, they remain an emergency room staple.

Brachycephalic breeds have a variety of different primary and secondary (Table 1) anatomic abnormalities which narrow the airways, increase airway turbulence, resulting in chronic subatmospheric airway pressures and resulting in exacerbation of the secondary problems (Table 1).

Normal respiratory anatomy

The respiratory system consists of the airways and the gas exchange portion of the lungs. The airways consist of the nares, nasal cavities, oropharynx, larynx, trachea, bronchi and bronchioles. The gas exchange portion consists of respiratory bronchioles and alveoli. The role of the upper respiratory tract is to inhale and exhale air, humidify that air, remove any particulate matter prior to movement into the lower respiratory tract. The role of the gas exchange areas of the lung is to move oxygen into the capillaries and carbon dioxide from the capillaries into the alveoli for exhalation.

In “normal dogs” the nares contribute 80% to airway resistance on inspiration. This leads to the relatively quiet respiratory noise we ordinarily associate with breathing. In brachycephalic dogs the narrowed nares and overlong soft palate (as well as other primary and secondary anatomic abnormalities) narrow the airway lumen and result in stertorous breathing. In cases of extreme narrowing, this may progress to stridor (Table 2). The importance of luminal diameter in maintaining food airflow and minimising resistance is highlighted in Poiseulle’s laws for airway flow and resistance (Figure 1). Since flow increases proportional to the 4^{th} power of the radius, and resistance decreases proportional to the 4^{th} power of the radius, it is obvious to see why even small increases (or decreases) in luminal airway diameter can be greatly clinically significant.

As well as anatomic abnormalities, there are also multiple proposed and proven airway functional abnormalities present in brachycephalic dogs. Chronic mild hypoxaemia, increased work of breathing, fibrosis of the pharyngeal dilator muscles, obstructive sleep apnoea,

pharyngeal collapse and increased risk of noncardiogenic pulmonary oedema have all been recognised in dogs with upper airway obstruction; and are either proposed or proven to occur in dogs with brachycephaly. Readers are encouraged to investigate Chapter 100 of Tobias and Johnstone 2nd Ed and associated references for further information on this matter.

Brachycephalic Obstructive Airway Syndrome (BOAS)

The clinical signs associated with BOAS can range from mild and self-limiting to severe and life threatening, and they regularly worsen with increased age and obesity as the secondary changes become more common. Clinical signs include – stertor, inspiratory stridor, exercise intolerance and in more severe cases orthopnoea and cyanosis. In severe acute obstructive crises syncope, hyperthermia and heat stroke, noncardiogenic pulmonary oedema and death can all occur.

When it comes to management of brachycephalic obstructive crisis, early client education and prophylactic preventative surgery is paramount, ideally with corrective surgery being performed on these animals at 6-12 months of age before the constellation of secondary changes can become particularly severe. It is important to realise that this will only mitigate the clinical signs and will not prevent the progression of BOAS in these animals, particularly if they become overweight or obese. One retrospective study found that there was a 30% increase in the odds of requiring a temporary tracheostomy per 1-year increase in age of the patient.

If these are unable to be performed (i.e. the patient is already older and has significant disease), weight loss, anxiety mediation, careful temperature management, and prevention of strenuous exercise can help mitigate airway crisis development. Evaluation with a surgeon and definitive staphylectomy/rhinoplasty may also be of assistance; but will not rectify the already present secondary changes.

It is also important to acknowledge that brachycephalic dogs are at higher risk of airway complications, even if they present to the ER/hospital for non-airway issues. A recent multicentre study found that while 55% of brachycephalic ICU admissions were treated for respiratory disease, more than 50% were initially admitted to the ICU for anaesthesia recovery or non-respiratory illness. 16% developed respiratory complications during hospitalisation. Indeed a 2022 retrospective evaluation of peri-anaesthetic mortality in English bulldogs found that there was a 3.9% peri-anaesthetic mortality and 6.6% all-cause mortality within the study cohort of “English bulldogs who underwent anaesthesia between 2010 and 2017”. This is particularly pertinent for brachycephalic patients who present for non-emergent reasons or elective procedures, as a perceived “low-risk” procedure or consultation can easily result in a prolonged ICU stay and adverse outcomes in this patient population.

Chief among the risks with these dogs is stress or anxiety mediated increased respiratory rate and effort, which increases the negative pressure in the airways and results in airway oedema. This airway oedema results in subtle narrowing of the airway lumen, which further increases airflow resistance. This further causes stress, resulting in increased tachypnoea, “air hunger”,

dyspnoea and further respiratory effort. The net result is a snow-ball effect, where a relatively mild change or inciting cause can result in severe upper airway obstructive crisis and subsequent adverse outcomes.

Given the high risks associated with BOAS patients, swift recognition of at risk patients and rapid intervention to reduce the risk of this progressive deterioration is essential. Judicious use of anxiolytics and sedatives (Table 3), active cooling if clinically indicated, increased inspired oxygen and utilisation of techniques to reduce airway swelling can all be effective at reducing further exacerbation of an obstructive crisis.

If an elective procedure or planned consultation is due for a known anxious brachycephalic, utilisation of a pre-consultation "chill protocol" of gabapentin (10-20mg/kg) + trazodone (5-8mg/kg) the evening before + 2 hours prior to the consultation can be helpful. Pairing this with antinausea (maropitant or ondansetron) can further aid in reducing the risk of stress induced vomiting/regurgitation and subsequent aspiration. Given the nature of ECC care – this is rarely feasible.

Rapid recognition of the at-risk patient starts with the reception staff recognising on the owner's phone call, being more likely to recommend hospital presentation in brachycephalic dogs, and allocating them a higher triage acuity category based on breed alone, regardless of presenting complaint. When the patient is triaged – minimising stress is vital – if the animal does better with the owners, allowing it to spend time with them, while anxiolytics are administered may be helpful. In a dyspnoeic patient, this is not feasible, and rapid assessment is vital to treat these patients. Administration of anxiolytics (butorphanol 0.2mg/kg +/- acepromazine 0.005-0.01mg/kg) IM or IV and allowing them to relax with oxygen supplementation. Use of alpha 2 adrenergic agonists is great in these patients, if they are cardiovascularly stable. The authors personally use dexmedetomidine 1-2mcg/kg frequently to help calm these dogs. The patient can also be given oxygen and adrenaline nebulisation while the drugs take effect.

Adrenaline nebulisation works through causing local vasoconstriction in the oropharynx and laryngeal regions, resulting in a theoretical reduction in oedema/swelling. In practice it seems to be helpful when applied in conjunction with other interventions and is a safe and easily administered therapy. It will cause some blanching of the oropharyngeal mucosa, and rarely tachycardia if higher doses are utilised.

If the patient is cyanotic/stridorous and does not immediately calm/respond to therapy, emergent intubation is likely indicated. IV propofol or alfaxalone to effect is useful to facilitate this. Be aware these animals almost always have high vagal tone, so vagal arrest is possible, and having atropine on standby is recommended. A rapid airway exam is ideal, however in an emergent patient it can be very challenging to do so thoroughly.

Once the patient is intubated, you have control of the airway, and can ventilate appropriately. Most of these animals will breathe well voluntarily, as the upper airway obstruction is relieved by bypassing the obstruction. Some will not, if they have significant pneumonia or NCPO.

The main challenge then becomes extubation in these cases. It has been shown in multiple papers that definitive airway surgery performed at the time of an obstructive crisis has a much higher risk of complications and requirement of temporary or permanent tracheostomy when compared to elective "stable" airway surgery. A novel technique which has been gaining popularity is called a "palatopexy": this consists of tacking sutures of absorbable monofilament suture (Monocryl or PDS), bringing the soft palate forward and attaching it to the roof of the mouth (Figure 2.). This allows the swelling associated with the obstructive crisis to go down, and the definitive surgery to be performed later. There is limited literature at this stage, although a retrospective evaluation of several hundred cases is currently being prepared for publication. Some of these animals do fail and require temporary tracheostomy, but the recovery is subjectively better than managing definitive surgery patients at the time of crisis. Interested readers are encouraged to read further in the 2022 JSAP paper by Sun et al.

Utilisation of anti-inflammatory glucocorticoids (dexamethasone 0.1mg/kg IV) along with mannitol-soaked gauze swabs packed in the caudal oropharynx may also help reduce laryngeal oedema. Owner assisted recovery from surgery has been shown to reduce risk of complications in one study evaluating a population of elective airway surgery BOAS patients, it has not been evaluated in the emergent obstructive crisis population.

The use of pure mu opioids has been associated with higher complications in brachycephalic surgery patients, utilisation of buprenorphine (partial mu agonist) + alpha 2 agonists may be safer in this patient population. Longer anaesthesia/procedure times are also associated with increased postoperative mortality – in one study – the risk of complications increased 18% per 30 minute increase in anaesthesia duration.

Conclusions

Brachycephalic obstructive airway syndrome is a unique challenge in the ER population, and swift, proactive intervention is necessary to maximise outcomes. It is important to remember this population of dogs is not "normal", and will never be normal, all of our interventions are aimed at slowing progression of disease and maximising quality of life. Management of owner expectations, open communication and, ideally, proactive surgical correction at a young age is vitally important to improve our patient outcomes.

$$R = \frac{8\eta l}{(\pi * r^4)}$$

$$Q = \frac{\Delta P * \pi * r^4}{8\eta l}$$

Figure 1: Poiseulle's laws for airway flow and resistance. R = Resistance, η is dynamic viscosity, l is length, r is radius. Q is flow, ΔP is pressure difference.

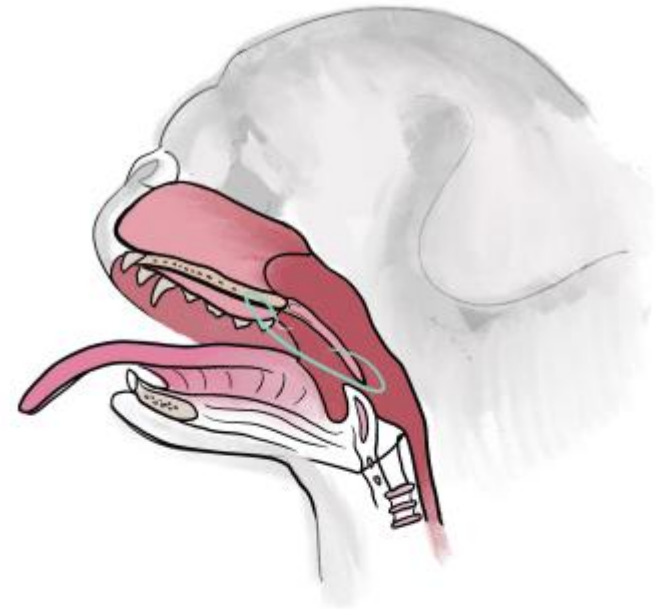

FIG 2. Suture placement for temporary palatopexy

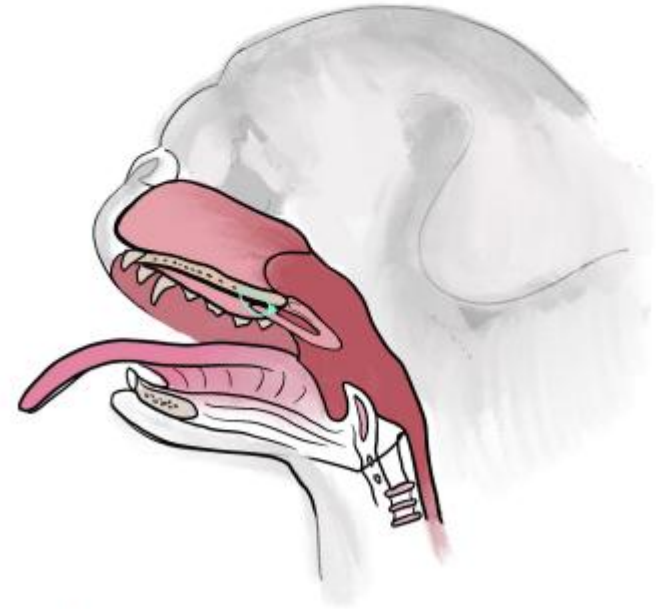

FIG 3. Suture and knot placement after tying

Figure 2: Graphic illustrating a sagittal view of a temporary palatopexy procedure. Adapted from *Evaluation of temporary palatopexy to manage brachycephalic obstructive airway syndrome in dogs in respiratory distress*, JSAP 2022

Table 1: Primary and secondary anatomic abnormalities associated with BOAS

Primary	**Secondary**
Stenotic nares	Laryngeal collapse
Macroglossia	Eversion of laryngeal saccules (grade 1 laryngeal collapse)

Redundant pharyngeal tissue	Tonsillar hyperplasia/Everted tonsils
Overlong/thick soft palate	Mucosal oedema and swelling
Hypoplastic trachea	Aerodigestive disorders
Aberrant turbinates (can also be seen as a secondary change due to increased airway turbulence)	Main stem bronchus collapse/tracheal collapse
	Sliding hiatal hernias (can be seen as a primary anatomic abnormality)
	Increase oropharyngeal fat
	Oesophageal dysfunction (including mega-oesophagus like phenotype).
	Other Secondary disorders which have been proposed to be associated with brachycephaly
Small epiglottis – inability to protect airway and increased aspiration risk.	Over-representation with chemodectoma has been theorised to be secondary to chronic hypercapnoea
	Middle ear effusion/Primary secretory otitis media (PSOM)
	Systemic hypertension
	Other gastrointestinal disorders over-represented in dogs with severe airway dysfunction - Gastroesophageal reflux (GORDS) - Oesophagitis - Pyloric mucosal hyperplasia - Diffuse gastric inflammation

Table 2: Stertor vs Stridor

Stertor	**Stridor**
- Low pitched, snoring like sound, due to airway turbulence and vibrations of soft tissues in the pharynx - Almost all Brachycephalic dogs have some degree of stertor	- High pitched wheezing/whistling sound due to rigid tissue vibrations associated with the larynx (most commonly) or trachea (less commonly - Generally considered more severe than stertor, can be exercise/excitement or oedema induced in some patients. - Often a sign of imminent requirement for intubation in BOAS crisis patients.

Table 3: Therapeutic options for BOAS crisis patients

Emergency Treatment	**Details, dose and route**
Oxygen therapy	- Fly by on presentation, continue with nasal/nasopharyngeal or oxygen cage therapy in patients requiring

	- High-flow nasal oxygen therapy, this allows FiO2 of up to 100% in a conscious patient and gives some degree of PEEP which can help keep the upper airways from collapsing. Issues with patient tolerance and possibility for overheating - CPAP helmet – similar for HFNO, however does not give FiO2 >21%, patient tolerance issues are significant. Overheating risk is present
Sedation and anxiolysis	- Acepromazine – 0.005-0.02mg/kg IM or IV. Can cause hypotension, irreversible. Can be repeated as required - Butorphanol – 0.1-0.4mg/kg IM or IV as required or 0.1-0.3mg/kg/hr CRI. Minimal respiratory depression. Has antitussive effect (undesirable in pneumonia cases). - Dexmedetomidine – 1-5mcg/kg IM or IV, strongly recommend following up with a 0.5-1 mcg/kg/hr CRI. Causes bradycardia, reduces cardiac output – not recommended in cardiovascularly unstable patients. Reversible. Can utilise Medetomidine. - Midazolam 0.1-0.3mg/kg IM or IV – rarely used in isolation (unless seizuring), can be a useful adjunct. Reversible - Gabapentin 10-20mg/kg PO Q8 – If tolerating PO medications – can give rectally dissolved in 5mL water, has good synergistic effect with trazodone + some analgesia - Trazodone 5-8mg/kg PO Q8 – If tolerating PO – can give rectally dissolved in 5mL water, has good synergy with gabapentin
Adrenaline nebulization	0.05mg/kg added to 0.9% NaCl to make a 5mL total volume. Nebulised for 10-15mins or until reservoir is empty.
Active cooling	If indicated – BOAS crises are at high risk of hyperthermia/heat stroke. Brachycephalic patients often are calmed by fans, white noise + airflow. - More aggressive cooling (flowing cool water + fans) to a goal of 39.5°C if significantly hyperthermic

Antinausea and prokinetic therapy - High risk of regurgitation/nausea and altered oropharyngeal anatomy puts these animals at high risk of aspiration events	- Maropitant 1mg/kg IM or IV Q24 - Ondansetron 0.5mg/kg IM or IV Q8 - Metoclopramide 0.5mg/kg IM or IV then a CRI @ 2mg/kg/day (0.08mg/kg/hour) - If significant gastric ileus is anticipated, or significant aerophagia – placement of a nasogastric tube to enable repeat gastric decompression is also helpful. Can give nutrition and PO medications in this way also. - If persistent ileus – erythromycin 0.5-1mg/kg slow IV Q8 - If concern for reflux/oesophagitis/gastric ulceration – Esomeprazole/pantoprazole 1mg/kg IV Q12 or omeprazole 1mg/kg PO/NGT Q12
Intubation	If a patient if poorly responsive to sedation therapy, the author will induce with propofol or alfaxalone to effect and intubate emergently. This will secure an airway and bypass the URT obstruction. Intubation with a sterile tube is ideal, as this allows an endotracheal wash/blind bronchoalveolar lavage for aspiration work up + is better hygiene for a long-term intubated patient. - Always have a selection of tubes + a very small tube (ETT 4.5) or cook airway exchange catheter for difficult intubations. - If the patient is intubated, putting mannitol or hypertonic saline soaked swabs in the caudal oropharynx may reduce local oedema and swelling to this area - Recovery from intubation can be challenging – palatopexy can be attempted, however some patients require temporary tracheostomy.
Balanced IV Crystalloid therapy - LRS/Hartmanns, Plasmalyte 148 or Normosol R preferred over 0.9% NaCl	- Many of these animals are hypovolaemic and require bolus therapy – 10 ml/kg given over 15minutes – re-evaluate perfusion parameters once administered. - If history of vomiting/GI losses – may require ongoing rehydration.
Treat aspiration pneumonia if present - High risk of regurgitation/nausea and abnormal oropharyngeal anatomy puts	- Brachycephalics have been shown to have lower oesophageal fluid pH, increased risk of aspiration events and

these animals at high risk of aspiration events	increased risk of aerodigestive disorders. Aspiration pneumonia is a common primary or secondary emergency complaint in this patient population - Broad-spectrum antibiosis if clinically indicated and supported by imaging – Amoxicillin/ampicillin @ 22-30mg/kg IV Q6-8, amoxicillin clavulanate @ 22-30mg/kg IV Q6-8, Ampicillin sulbactam @ 22-30mg/kg IV Q8, Clindamycin @ 10mg/kg IV, SC or PO Q12 - **NB** – if this is a recurrent issue, strongly recommend ETW or BAL for culture due to high incidence of resistance.
Nil by mouth (NPO) initially	These are high aspiration risk patients. They should not have ad lib water access, offering them supervised water access every few hours should be sufficient + IVFT. Offering 1-2 x 3cm "meatballs" of a low fat/highly palatable food as a food trial can be attempted after several days.
Glucocorticoid therapy	Use with discretion/caution – rule out evidence of heatstroke induced AKI/MODS or severe GI ulceration prior to administration. Can aid in airway swelling/oedema/inflammation - Dexamethasone sodium phosphate 0.1mg/kg IV

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