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Davina graduated from Cambridge in 1989 and spent two years in mixed practice before completing an internship at the Royal Veterinary College. She then spent a further year in general practice before returning to Cambridge to complete a Wellcome Trust PhD at the Department of Pathology, and a Residency in Small Animal Surgery at the Queen’s Veterinary School Hospital. She became a Diplomate of the European College of Veterinary Surgeons in 2000 and was awarded the RCVS Diploma in Small Animal Surgery (Soft Tissue) in 2002. She is a RCVS Recognised Specialist in Small Animal Surgery and has been an Examiner for the ECVS as well as President in 2015. She has been working as a soft tissue surgeon in referral practice since 1997 and has extensive experience of management of brachycephalic breeds.

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Introduction/Overview

With the wide diversity of dog breeds available, aesthetics have had a key role in selection of phenotype traits for breeding. However, concerns should be raised when conformational features, included in breed standards, have a detrimental impact on the dog’s health and welfare (Asher et al. 2009).

Brachycephalic breeds differ from mesocephalic and dolichocephalic breeds by their shortened and wider skull, and open orbitae (Dupré et al. 2013), giving them a child-like appearance which seems to be appealing to the general public (Noller et al. 2008). A steady increase in popularity of brachycephalic breeds is being observed in the United Kingdom. According to Kennel Club registry numbers, the French Bulldog population increased from 6990 in 2013, to 21470 in 2016, being now the third most popular dog breed in the UK. Despite this, recent surveys of brachycephalic dog owners reveal a worrying lack of awareness and failure to recognise the health concerns related to these breeds (Packer et al. 2012, Liu et al. 2015). This inability to recognise the disease may often cause a delay in treatment and further deterioration, with the veterinary surgeon playing a central role in the general public education.

Brachycephalic obstructive airway syndrome (BOAS) is caused by a combination of morphological abnormalities, including a marked craniofacial shortening that ultimately results in an inappropriate resistance to airflow. This article will cover the main aspects of pathogenesis, clinical findings and treatment options for dogs diagnosed with BOAS.

Pathogenesis/Signalment

Brachycephaly results from a discrete skeletal mutation and artificial selection of individuals exhibiting this phenotype trait (Pollinger et al. 2005). In these breeds the facial skeleton is shortened and broadened, whereas the mandible is often of a relatively normal length, producing the undershot appearance: mandibular prognathism with maxillary brachignatism (Evans and de Lahunta 2013). Shortening of facial bones results in caudal dislocation of nasal structures and abnormal anatomic position of conchae (Noller et al. 2008, Dupré et al. 2013), also causing a steep course for the intranasal airways and lacrimal system (Noller et al. 2008). The frontal sinuses are usually extremely small in all affected breeds, and are often completely absent in Pugs (Noller et al. 2008, Heidenreich et al. 2016).

In general, BOAS arises because there is a mismatch between a shortened skull (Figure 1) and the soft tissues that have not accompanied this shortening, causing the airway to be crowded and consequently partially obstructed (Harvey 1989).

Although several skull measurements and ratios have been described in the literature
to better characterise what is considered a brachycephalic dog (Regodón et al. 1993, Packer et al. 2012, Evans and de Lahunta 2013), these are beyond the scope of this article. More significant, however, is the correlation between short muzzles and its negative impact on the dog’s health. Packer et al. (2015) observed that BOAS only occurs in dogs where the muzzle length is less than half of the full length of the cranium (Figure 2). In the same study, increased neck girth and obesity were also observed to have a negative impact on BOAS development (Packer et al. 2015).

Common breeds affected by this condition include, amongst others, French Bulldogs, Pugs, English Bulldogs, Boston Terriers, Cavalier King Charles Spaniels (Lorinson et al. 1997, Fasanella et al. 2010).

Mean age of presentation is usually between two and four years old (Lorinson et al. 1997, Poncet et al. 2006, Torrez and Hunt 2006, Riecks et al. 2007, Fasanella et al. 2010), although puppies from 2.5 months old are reported in the literature (Riecks et al. 2007). One should note that a significant percentage of dogs under one year old are reported in many studies (Riecks et al. 2007, Fasanella et al. 2010).

Components of BOAS

The components of BOAS can be subdivided into primary components, secondary components and concomitant conditions (Table 1).

In general, primary components increase negative pressure on the airways during inspiration, which leads to mucosal inflammation and oedema, eversion of laryngeal saccules and laryngeal collapse (secondary components) (Torrez and Hunt 2006, Riecks et al. 2007).

It is important to emphasise that, although described as separate components here, most brachycephalic dogs that present with dyspnoea will have more than one component present (Riecks et al. 2007).

Primary components

1 - Soft Palate and nasopharynx:
An elongated (Figure 3) and thickened soft palate (Figure 4) is the most common abnormality observed in brachycephalic dogs, affecting 85 to 100% of BOAS cases (Lorinson et al. 1997, Poncet et al. 2006, Fasanella et al. 2010). In brachycephalic breeds, the transition between hard and soft palate is more caudal than in mesocephalic breeds (Packer and Tivers 2015), and it is a common finding that the soft palate partially obstructs the larynx. This overly long and thickened soft palate can often be demonstrated with computed tomography (CT), extending 1 to 2 cm past the epiglottis (Dupre 2008) and a positive correlation between soft palate thickness and severity of clinical signs has been established in a CT based study (Grand and Bureau 2011). It has been reported that French Bulldogs have a thicker soft palate than Pugs, but in one study 81% of the Pugs showed no airway space dorsal to the soft palate (Figure 4,B) (Heidenreich et al. 2016).

Histopathology of elongated soft palates revealed a thickened epithelium, extensive oedema of connective tissue and mucous gland hyperplasia (Pichetto et al. 2011),

Table 1: BOAS components

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<thead>
<tr>
<th>Primary components</th>
<th>Secondary components</th>
<th>Concomitant conditions</th>
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<tbody>
<tr>
<td>Overlong and hyperplastic soft palate</td>
<td>Laryngeal collapse (including eversion of laryngeal saccules)</td>
<td>Hypoplastic trachea</td>
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<tr>
<td>Stenotic nostrils</td>
<td>Tonsillitis</td>
<td></td>
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<tr>
<td>Intranasal obstruction (including aberrant conchal growth)</td>
<td>Bronchial Collapse</td>
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“Most brachycephalic dogs that present with dyspnoea will have more than one component present”

Figure 1: Three dimensional (3D) volume rendering reconstruction of two skull computed tomography (CT) scans, illustrating the marked shortening of facial bones in brachycephalic dogs, when comparing to a mesocephalic dog. Left: German Shepherd, Right: French Bulldog. © Anderson Moores Veterinary Specialists.

Figure 2: Conformational degrees of brachycephaly. Left: mild brachycephaly in a Boxer; Middle: moderate brachycephaly in an English Bulldog; Right: Extreme brachycephaly in a Pug. © Anderson Moores Veterinary Specialists.

Figure 3: Elongated soft palate in a 6 month old English Bulldog puppy. Note the soft palate extending past the epiglottis and partially obstructing the rima glottides. © Anderson Moores Veterinary Specialists.