Dynamic collapse of the upper respiratory tract: A review

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Summary

Dynamic collapse of the upper respiratory tract is a common cause of poor performance in athletic horses. Most commonly, airway obstruction occurs during strenuous exercise when the upper respiratory tract is exposed to high pressure swings. In horses undertaking submaximal exercise, the pressures may also be increased due to flexion of the neck. The nasopharynx and larynx are particularly prone to dynamic collapse and a number of different forms of upper airway obstruction are now recognised. However, due to the dynamic nature of the collapse a definitive diagnosis is often not possible from resting observations alone.

Introduction

Dynamic collapse of the upper respiratory tract (URT) describes respiratory obstructions caused by the collapse of one or more soft tissue structures into the airway during exercise. In the majority of cases, horses appear normal during a resting endoscopic examination and the obstruction only becomes evident during exercise. The reason for this lies with the dramatic increases in airflow and airway pressure changes that occur during exercise (Table 1). The horse is an obligatory nasal breather (Negus 1949) and unlike other species, including man, is unable to switch from nasal to oral breathing in order to adapt to the movement of large volumes of air. Many forms of URT obstruction only occur during strenuous exercise when airflow and the collapsing forces are at their peak. This explains the high prevalence of such conditions in racehorses. However, neck flexion has also been shown to increase upper airway pressures (Petsche et al. 1995) and may be an exacerbating factor in equine athletes such as dressage horses, show horses and showjumpers working at lower exercise intensities (Franklin et al. 2006).

Certain structures within the airway are at particular risk of collapse. These include the nares, the nasopharynx and larynx, which rely on muscular activity to maintain airway patency. Any weakness (either pathological or physiological) may result in an inability to resist the increased pressure gradients associated with exercise and hence will result in dynamic airway collapse. A variety of forms of collapse are now recognised and these will be discussed in this review. The clinical significance and prevalence of the different disorders will also be addressed.

For many years, URT obstructions have been diagnosed on the basis of the detection of abnormal respiratory sounds and endoscopic findings during quiet breathing at rest. However, in the light of recent treadmill studies, the reliance on such methods has been questioned. This review will discuss the usefulness of the different diagnostic modalities available to the clinician.

Assessment of respiratory sounds

The association between obstructive disorders of the URT and the production of abnormal respiratory sounds has been acknowledged for many years (Williams 1874; Cook 1965; Gerring 1985; McCann 2000). It has been proposed that the nature of the sound together with other information such as the time in the respiratory cycle that it occurs, and whether the noise is intermittent or continuous, may be helpful in diagnosing the cause of the condition. However, evaluation of respiratory sounds by ear may be difficult because of observer location, resulting in poor transfer of sound to the ear or superimposition of extraneous noise, limitations of hearing

<table>
<thead>
<tr>
<th>Respiratory parameter</th>
<th>Rest</th>
<th>Strenuous exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing frequency (F&lt;sub&gt;B&lt;/sub&gt;) (breaths/min)</td>
<td>10–15</td>
<td>120–150</td>
</tr>
<tr>
<td>Tidal volume (V&lt;sub&gt;T&lt;/sub&gt;) (l)</td>
<td>3–6</td>
<td>14–20</td>
</tr>
<tr>
<td>Minute ventilation (V&lt;sub&gt;l&lt;/sub&gt;) (l/min)</td>
<td>40–60</td>
<td>1500–2000</td>
</tr>
<tr>
<td>Peak inspiratory flow rate (PIF) (l/s)</td>
<td>3</td>
<td>80</td>
</tr>
<tr>
<td>Peak expiratory flow rate (PEF) (l/s)</td>
<td>3</td>
<td>100</td>
</tr>
<tr>
<td>Peak inspiratory pharyngeal pressure (cm H&lt;sub&gt;2&lt;/sub&gt;O)</td>
<td>-20 to -26</td>
<td></td>
</tr>
<tr>
<td>Peak expiratory pharyngeal pressure (cm H&lt;sub&gt;2&lt;/sub&gt;O)</td>
<td>10–20</td>
<td></td>
</tr>
</tbody>
</table>

Data from 1Marlin and Nankervis (2002) and 2Ducharme et al. (1994).
acuity and difficulties in differentiating between the sounds associated with specific conditions.

In recent years, there has been some interest in the recording and spectral analysis of respiratory sounds during exercise as a potential method for diagnosis of URT obstructions (Attenburrow 1978; Derksen et al. 2001; Franklin et al. 2003). Horses with laryngeal collapse have been shown to make high frequency sounds during inspiration (Derksen et al. 2001; Franklin et al. 2003) and horses with dorsal displacement of the soft palate (DDSP) are characterised by a low frequency peak, during expiration, which corresponds to the frequency of vibration of the caudal border of the soft palate (Franklin et al. 2004).

Whilst respiratory sound analysis has shown some promise, authors have yet to distinguish between conditions that result in audibly similar inspiratory sounds. In addition it has been shown that a large proportion of horses may exhibit multiple forms of dynamic collapse (Tan et al. 2005; Lane et al. 2006a). To date there is no evidence that horses with complex dynamic airway collapse can be diagnosed definitively using respiratory sound analysis. Therefore, further studies are required before this technique can be used clinically.

**Resting endoscopic examination**

A resting endoscopic examination is useful in order to identify static obstructive conditions including sub-epiglottal cysts, congenital palatal defects, cases of persistent epiglottal entrapment (EE) and true laryngeal hemiplegia. In certain circumstances a resting endoscopic investigation may also give useful information regarding nasopharyngeal and laryngeal function. Where possible assessment of the URT should be made with the horse unsedated as sedation may influence the evaluation of laryngeal cartilage movements (Archer et al. 1991; Ducharme et al. 1991) and pharyngeal function. In the majority of cases endoscopy may be performed without the need for additional restraint. However, a ‘nose twitch’ may be useful in some cases and does not significantly influence the interpretation of resting laryngeal activity (Archer et al. 1991).

A thorough endoscopic investigation should include an examination of both nasal passages as well as the nasopharynx and larynx. Perspective artefacts arise due to the fact that the larynx is viewed via one nasal cavity rather than from a central position (Fig 1). Therefore, when assessing laryngeal function, it is recommended that the same nostril be used routinely for introduction of the endoscope (Robinson 2004).

Nostril occlusion and the stimulation of swallowing may be used to aid the evaluation of laryngeal and pharyngeal function during the course of the examination. Nasal occlusion has been shown to simulate upper airway pressures achieved during high intensity exercise (Holcombe et al. 1996) and is routinely used as a simple and effective method to assess arytenoid function at rest (Archer et al. 1991). Stimulation of swallowing by introducing a small volume of water or by using a probe passed down the biopsy channel may also assist the assessment of laryngeal function. In addition, swallowing may reveal entrapment in horses with intermittent EE. Dorsal displacement of the soft palate may also occur spontaneously in response to nasal occlusion or after swallowing.

**Assessment of pharyngeal function**

The use of nasal occlusion and the stimulation of swallowing to assess the function of the soft palate and nasopharyngeal walls are questionable and should be interpreted with caution. Some horses show nasopharyngeal collapse during a resting examination but have normal nasopharyngeal function during exercise. Others that are normal at rest may exhibit dysfunction during high-speed treadmill endoscopy (HSTE).

Lane et al. (2006b) found that prolonged spontaneous displacement may be indicative of soft palate dysfunction during exercise: 26/34 (76%) of horses that showed spontaneous displacement at rest also had DDSP during exercise. However, the majority of horses with DDSP during exercise showed no abnormalities at rest. Similarly, other studies have reported a weak link between resting findings and a diagnosis of DDSP during HSTE (Kanegieter and Dore 1995; Parente and Martin 1995; Parente et al. 2002).

Where displacement of the soft palate can be induced, it is useful to examine the caudal border for evidence of previous surgery, in the form of a staphylectomy, and for evidence of ulceration. Where present, this may be indicative of trauma...
arising from DDSP (Norwood 1983; Robertson 1991), although again this appears to be relatively uncommon (Parente et al. 2002; Lane et al. 2006b). Examination of the caudal border of the soft palate and the under side of the epiglottis may also be performed by retroverting the epiglottis with a probe following the administration of local anaesthesia (Blea and Arthur 2003).

Historically DDSP has been associated with abnormal epiglottic conformation or epiglottic hypoplasia (Linford et al. 1983; Robertson 1991). However, recent HSTE studies report that the majority of horses with DDSP have a normal epiglottic appearance (Kannegieter and Dore 1995; Martin et al. 2000; Parente et al. 2002; Tan et al. 2005; Lane et al. 2006b). Furthermore, there is both clinical (Parente et al. 1998) and experimental (Holcombe et al. 1997a) evidence that the epiglottis is not required to maintain the soft palate in a normal subepiglottic position.

Assessment of laryngeal function

In recent years, there has been much debate regarding the relevance of resting observations in predicting arytenoid cartilage collapse (ACC) during exercise. A number of scoring systems have been used in order to describe resting observations including a 4 point scale (used widely in the USA) (Hackett et al. 1991), a 5 point scale (used in the UK and Australia) (Lane 1993) and a 6 point scale (Dixon et al. 2002). More recently it has been proposed that a single 7 point scale should be adopted worldwide (Robinson 2004).

Regardless of the grading system used, it is clear that those horses with true laryngeal hemiplegia, i.e. with no active movement of the left arytenoid cartilage at rest will also fail to achieve abduction during exercise. However, the significance of laryngeal asymmetry or asynchronous movements of the arytenoid cartilages is less clear. Duncan et al. (1977) suggested that laryngeal asymmetry or asynchrony at rest represents a subclinical form of recurrent laryngeal neuropathy (RLN) that may result in a performance-limiting malfunction under exercise conditions. However, subsequent studies by Morris and Seeherman (1990) and Rakestraw et al. (1991) found that many horses with laryngeal asynchrony and asymmetry at rest have normal laryngeal function during exercise. In contrast, a later study by Morris and Seeherman (1991) found that some horses that were considered normal or had asynchronous movements at rest progressed to complete dynamic collapse of the arytenoids during exercise. Hammer et al. (1998) also found that the majority of horses with asymmetry at rest developed severe dynamic laryngeal collapse during exercise and argued that if treadmill exercise had been more strenuous in the earlier studies, abnormal laryngeal function may have been detected in some of these cases.

When reviewed in combination the results of these and other recent studies (Kannegieter and Dore 1995; Martin et al. 2000; Tan et al. 2005; Lane et al. 2006b) suggest that the majority of horses that fail to achieve maximal abduction in response to nasal occlusion or induction of swallowing will develop dynamic collapse during exercise. In contrast, the majority of those that are able to achieve full abduction will similarly achieve full abduction during exercise.

It is acknowledged that a small proportion of horses with ‘normal’ laryngeal function at rest may develop dynamic laryngeal collapse under exercise conditions (Morris and Seeherman 1991; Kannegieter and Dore 1995; Lane et al. 2006b). Furthermore, Davidson et al. (2007) confirmed that RLN may be progressive in some cases and that horses showing normal laryngeal function or mild asymmetry may develop true hemiplegia over time (from months to years), although the reported incidence of progression is low (5–15%) (Anderson et al. 1997; Dixon et al. 2002). There are also many horses (up to 20%) that are unable to achieve full abduction during a resting examination, yet are capable of full symmetrical abduction during strenuous exercise (Hammer et al. 1998; Martin et al. 2000; Lane et al. 2006b).

Additional procedures including palpation of the larynx in order to assess left sided atrophy of the cricoarytenoideus dorsalis muscle and the use of the thoracolaryngeal reflex (‘slap test’) have been used to aid the diagnosis of RLN for many years (Cook 1965; Greet et al. 1980; Lane 1993). The use of electrolaryngeography was subsequently developed as an objective method to determine the latency of the thoracolaryngeal reflex (Cook and Thalhammer 1991). This technique has been reported to be useful in distinguishing between horses with subclinical RLN (Curtis et al. 2005). Others have shown such techniques to be unreliable (Newton-Clarke et al. 1994; Hawe et al. 2001) and currently none of these tests is believed to be as effective as resting endoscopic examination for diagnosing RLN (Robinson 2004). However, Lane et al. (2006b) suggested that when used in combination with resting endoscopy, other findings relating to laryngeal palpation and respiratory noise may aid the clinician in diagnosing RLN.

Further investigations are warranted for those horses in which the endoscopic findings are inconsistent with the clinical history. In particular, horses that make abnormal inspiratory noises during exercise yet have normal or equivocal laryngeal function at rest should undergo an endoscopic examination during exercise on a high-speed treadmill in order to make a definitive diagnosis.

Limitations of resting endoscopy

Recent studies have shown resting endoscopy to be unhelpful to make a diagnosis of dynamic airway collapse in many cases (Kannegieter and Dore 1995; Tan et al. 2005; Lane et al. 2006b). The majority of horses with dynamic nasopharyngeal collapse during exercise show no abnormalities during a resting examination. Similarly, horses with axial deviation of the aryepiglottal folds (ADAF), vocal cord collapse (VCC) and other less common forms of dynamic laryngeal collapse usually appear ‘normal’ during a resting examination. In an attempt to improve the diagnostic value of resting endoscopy some clinicians perform endoscopy immediately after exercise. However, this is unlikely to be beneficial as most forms of dynamic airway collapse resolve within seconds of the completion of the exercise test.
The use of the respiratory stimulant lobeline has been described as an aid to facilitate upper airway evaluation in the resting horse, by inducing respiratory hyperpnoea (Marlin et al. 2000; Brink 2005). Use of this drug achieves a minute ventilation of approx 920 l/min and peak flow rates of 41 l/s (during inspiration) and 61 l/s (during expiration) (Marlin et al. 2000), equivalent to those achieved at exercise at a canter (Lekeux and Art 1994). However, this does not equate to flow rates achieved during maximal exercise (Table 1) and hence may be of no use in making a definitive diagnosis in cases that only show dynamic airway collapse during more strenuous exercise (Strand et al. 2004). The only method that may currently be used to make a definitive diagnosis of dynamic airway collapse is to perform an endoscopic examination during exercise on a high-speed treadmill (Fig 2). Unfortunately the limited number of centres providing treadmill facilities, the cost and time implications, and misconceptions regarding the safety of the technique mean that this is not always performed. It is hoped that in the future, technological developments may allow for over-ground endoscopy whereby a video-endoscope is placed into the URT during ridden exercise.

**Disorders of the nasal passages and nasopharynx**

**Alar fold collapse**

This is an uncommon cause of abnormal respiratory noise and exercise intolerance in horses. During exercise affected horses make a characteristic loud ‘buzzing’ noise during both inspiration and expiration but have no abnormalities during resting or exercising endoscopy. Diagnosis is confirmed by decreased respiratory noise as a result of securing the alar folds dorsally with a temporary mattress suture (Fig 3). The aetiology of the condition is unknown although it has been proposed that excessive alar fold tissue or inappropriate function of the transversus nasi muscles may be implicated. Standardbreds and American Saddlebreds are reported to be predisposed to this condition (Hawkins et al. 1995), although all cases seen at the University of Bristol have been Thoroughbreds.

**Palatal malfunction**

This is now recognised to be the most common URT disorder affecting racehorses with a prevalence of 20–83% in horses referred for poor performance investigations (Morris and Seeherman 1991; Hackett et al. 1994; Kannegieter and Dore 1995; Lumsden et al. 1995; Martin et al. 2000; Lane et al. 2006a) and an estimated prevalence of 1–6.5% in the general population of racehorses (Cook 1965; Franklin 2002). The condition may also be observed in eventers and other horses performing less strenuous exercise (Franklin et al. 2006).

During exercise, the soft palate is normally positioned ventral to the epiglottis. Dorsal displacement of the soft palate occurs when the caudal border of the soft palate becomes displaced to a position above the epiglottis resulting in obstruction of the rima glottidis, predominantly during expiration (Fig 4). Instability of the caudal soft palate that does not lead to DDSP during treadmill exercise has been described by several authors (Kannegieter and Dore 1995; Tan et al. 2005; Lane et al. 2006a). This is manifest as progressive dorso-ventral billowing movements of the caudal portion of the soft palate, with flattening of the ventral surface of the epiglottis against the dorsal surface of the soft palate (Fig 5). From observations of video-recordings it is apparent that the billowing is identical to that observed in horses with DDSP in the period immediately before displacement (Kannegieter and Dore 1995; Lane et al. 2006a). This leads to the conclusion that PI and DDSP are manifestations of the same condition (Lane et al. 2006a).

Treadmill exercise cannot completely reproduce the conditions experienced during over-ground exercise and hence it may not always be possible to make a definitive diagnosis of DDSP in horses with a history suggestive of the condition. Despite this HSTE remains the only way to make a definitive diagnosis of palatal malfunction and is also required to rule out additional forms of dynamic airway collapse such as ADAF, which may occur in many cases (Martin et al. 2000; Tan et al. 2005; Lane et al. 2006a). Although many horses with DDSP are reported to make a characteristic ‘gurgling’ sound during exercise (Cook 1965; Heffron and Baker 1979; Haynes 1983; Robertson 1991), reports suggest that up to 30% cases may make no audible abnormal sound (Ahern 1999; Martin et al. 2000; Parente et al. 2002). Horses with palatal instability also make no abnormal inspiratory sounds, although low grade inspiratory sounds are produced (Franklin 2002) that may in some cases be confused with some forms of dynamic laryngeal collapse.

Where DDSP occurs, there is a sudden reduction in minute ventilation and consequently in oxygen supply (Table 2), resulting in impaired performance, with many horses described as ‘fading rapidly’ or ‘stopping suddenly as if shot’ (Franklin et al. 2002). The clinical significance of palatal instability in the absence of DDSP is unclear but is likely to vary between horses depending on the degree of nasopharyngeal obstruction and the type of work the horse is required to perform.

The underlying aetiology of palatal malfunction remains unclear although the current general consensus is that the condition arises as a result of some weakness of the intrinsic and/or extrinsic musculature. Experimental evidence that DDSP results from weakness of the intrinsic palatal musculature has been provided by Holcombe et al. (1998) whilst, more recently, malfunction of the thyro-hyoideus muscles - accessory muscles of respiration - has been implicated (Ducharme et al. 2003). The cause of any muscular weakness remains speculative and whilst it has been suggested that this may arise as a result of previous URT inflammation, a causal link remains to be proven.

Rostral soft palate instability occurs rarely and is likely to be a different condition to that described above. Rostral soft palate instability has been alluded to only briefly in the literature (Ducharme 2006) but has been observed in 6 cases (predominantly in ponies) at the University of Bristol (Allen et al. 2007). This condition results in the production of loud
inspiratory sounds at low exercise intensities and even at rest in some cases. Endoscopic examination at rest may or may not elicit excessive movement of the rostral soft palate. During exercise, video-endoscopy confirms that there is minimal movement of the caudal soft palate. However, with the endoscope positioned more rostrally within the nasal passages, marked dorso-ventral excursions of the rostral soft palate are observed (Fig 6). In many cases this may completely obstruct the choanae, causing severe obstruction to airflow. Similar instability of the rostral soft palate has been reproduced experimentally by tenectomy of the tensor veli palatini (Holcombe et al. 1997b).

### Pharyngeal Wall Collapse

This condition occurs much less commonly than palatal malfunction. Treadmill studies commonly report a prevalence of 1.3–4% in racehorses referred for investigation of poor performance and/or abnormal respiratory noise (Morris and Seeherman 1991; Kannegieter and Dore 1995; Tan et al.

### Table 2: Respiratory Parameters in Thoroughbred Horses with Dorsal Displacement of the Soft Palate (DDSP) and Laryngeal Hemiplegia (LH) during Strenuous Exercise

<table>
<thead>
<tr>
<th>Respiratory Parameter</th>
<th>Mean (± s.d.) value in TB horses before and during DDSP</th>
<th>Mean (± s.e.) value in TB horses with and without induced LH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing frequency (F_B) (breaths/min)</td>
<td>Before DDSP = 124 (9)</td>
<td>Not recorded</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 121 (9)</td>
<td></td>
</tr>
<tr>
<td>Tidal volume (V_T) (l)</td>
<td>Before DDSP = 16.7 (2.5)</td>
<td>Not recorded</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 14.9 (2.8)*</td>
<td></td>
</tr>
<tr>
<td>Minute ventilation (V_E) (l/min)</td>
<td>Before DDSP = 2036 (244)</td>
<td>Without LH = 2419 (163)</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 1769 (268)*</td>
<td>With LH = 1457 (186)*</td>
</tr>
<tr>
<td>Oxygen consumption (VO_2) (ml/kg bwt/min)</td>
<td>Before DDSP = 189.2 (10.0)</td>
<td>Without LH = 165.3 (3.4)</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 169.9 (17.2)*</td>
<td>With LH = 140.0 (3.2)*</td>
</tr>
<tr>
<td>Peak inspiratory flow rate (PIF) (l/s)</td>
<td>Before DDSP = 79.0 (8.1)</td>
<td>Without LH = 92.7 (10.5)</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 78.1 (9.7)</td>
<td>With LH = 61.2 (11.6)*</td>
</tr>
<tr>
<td>Peak expiratory flow rate (PEF) (l/s)</td>
<td>Before DDSP = 87.9 (10.9)</td>
<td>Not recorded</td>
</tr>
<tr>
<td></td>
<td>During DDSP = 78.6 (12.5)</td>
<td></td>
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</tbody>
</table>

Data from Franklin et al. 2002 and Seeherman et al. 1995. Key: *significant difference between value before DDSP and after DDSP; † significant difference between value without LH and with LH.

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**Fig 3:** A mattress suture is used to secure the alar folds.

**Fig 4:** Dorsal displacement of the soft palate.

**Fig 5:** Palatal instability affecting the caudal soft palate. The epiglottis is ‘flattened’ against the dorsal surface of the soft palate.

**Fig 6:** Billowing of the rostral soft palate.
2005; Lane et al. 2006a), although one study reported a prevalence of 27% (Martin et al. 2000). This study included a greater proportion of Standardbreds. It is possible that pharyngeal wall collapse may be exacerbated in this breed due to the enforced neck flexion when harnessed (Boyle et al. 2006). Davidson et al. (2002) and Franklin et al. (2006) have reported pharyngeal collapse to occur more frequently in sport horses than in racehorses. Again neck flexion was found to be an important contributing factor.

Dynamic pharyngeal wall collapse may involve either the lateral pharyngeal walls (Fig 7a) or the dorsal pharyngeal wall which makes up the roof of the nasopharynx (Fig 7b) (Strand and Staempfli 1993; Boyle et al. 2006). In some horses, a combination of dorsal and lateral pharyngeal collapse together with upward movements of the soft palate may result in pronounced circumferential pharyngeal collapse (Fig 7c) (Boyle et al. 2006). Affected horses make rough or musical inspiratory sounds during exercise, which may be confused with different forms of laryngeal collapse. At rest, the majority of horses have no notable findings. In some cases, nasopharyngeal collapse may be observed in response to nostril occlusion. However, other horses that show signs of nasopharyngeal collapse at rest have normal nasopharyngeal function during exercise. During exercise, it is necessary to position the endoscope rostrally in order to observe lateral pharyngeal wall collapse, since this may be missed if the endoscope is positioned in its normal site closer to the larynx.

In some horses, pharyngeal wall collapse is associated with hyperkalaemic periodic paralysis (Carr et al. 1996). However, in the majority of cases, the aetiology is unknown. Experimentally, pharyngeal wall collapse has been induced as a result of local anaesthesia of the laryngeal mucosa, suggesting that disruption of the mucosal mechanoreceptors or branches of the superior laryngeal nerve may be involved in nasopharyngeal collapse (Holcombe et al. 2001). In addition, bilateral blockade of the glossopharyngeal nerve results in stylopharyngeus muscle dysfunction and subsequent dorsal pharyngeal wall collapse during exercise, indicating that dysfunction of this muscle may be implicated in clinical cases (Tessier et al. 2004).

The prognosis for continual athletic performance in racehorses with pharyngeal collapse is guarded and often horses are retired from racing (Boyle et al. 2006). In other sport and pleasure horses, exercise tolerance may not be compromised and the degree of pharyngeal collapse may be reduced by riding the horse with the neck in a less flexed position.
Disorders of the larynx

The use of high-speed treadmill endoscopy during recent years has confirmed that there are a number of forms of dynamic collapse that may affect the laryngeal airway. These different forms of collapse invariably result in the production of musical, ‘whistling’ or ‘roaring’ sounds during inspiration and may be very difficult to differentiate by ear.

Left sided arytenoid cartilage collapse

This is most commonly attributed to RLN (Cahill and Goulden 1987; Dixon et al. 2001). During exercise, the corniculate process of the arytenoid cartilage moves towards the midline of the rima glottidis during inspiration and in some cases may collapse into the right half of the rima glottidis (Fig 8). Ipsilateral vocal fold collapse invariably accompanies arytenoid cartilage collapse, as a result of passive movement when the arytenoid is not fully dilated. In some cases bilateral vocal fold collapse may be observed (Lane et al. 2006a) (Fig 9).

In some horses, partial arytenoid collapse may occur whereby the arytenoid cartilage is not fully abducted but there is no further tendency to dynamic collapse towards the mid line during inspiration (Fig 10). In horses with partial arytenoid collapse, the degree of abduction of the left arytenoid is often the same or better than that achieved by laryngoplasty. However, where vocal fold collapse is present it may obstruct the ventral aspect of the rima glottidis.

The clinical significance of ACC will depend on the degree of obstruction of the rima glottidis and the type of work that the horse is expected to perform. In racehorses, even a small degree of airway obstruction may result in reduced performance. Where there is complete left sided collapse present, marked reductions in minute ventilation are recorded during exercise (Table 2) (Lumsden et al. 1993; Ehrich et al. 1995; Seeherman et al. 1995). However, this may not compromise exercise capacity in those horses performing less strenuous work (Seeherman et al. 1995). Instead, the main complaint may be abnormal respiratory noise.

The prevalence of RLN varies between breeds. It is most common in larger horses including Thoroughbreds, heavyweight hunter types and draught breeds such as Clydesdales (Cahill and Goulden 1987; Dixon et al. 2001). True prevalence is difficult to assess because there is no universally accepted definition of the disease and the parameters used to confirm the diagnosis differ between authors. In Thoroughbreds, the prevalence of true laryngeal hemiplegia is estimated to be 0.3–3% (Pascoe 1981; Raphel 1982; Anderson et al. 1997; Brown et al. 2005), whilst subclinical RLN is reported to affect 40–91% of horses (Gunn 1972; Baker 1983; Cook 1988).

Right sided arytenoid cartilage collapse

Right sided arytenoid cartilage collapse (Fig 11) is observed much less frequently than left sided collapse. A prevalence of 0.34% was reported in a study of 3497 yearlings (Lane 2003). Right sided collapse is most commonly associated with fourth branchial arch defects (4BAD) (Kannegieter et al. 1986; Lane 2001). A diagnosis of 4BAD may be made by palpation of the larynx which reveals that the wing of the thyroid cartilage on the right side is absent. Resting endoscopic examination frequently reveals some degree of right sided laryngeal dysfunction (39/60 cases vs. 5 left sided and 16 bilateral; Lane 2001). Some horses may also show rostral displacement of the palatopharyngeal arch (Fig 12), although this is not always present. During exercise, some horses have dynamic collapse of the right arytenoid cartilage and vocal fold. Others may have additional or alternative forms of dynamic collapse including DDSP and ADAF (Lane et al. 2006a). The clinical significance of this condition will depend on the type of dynamic airway collapse that occurs during exercise. Frequently affected horses are ineffective athletes and aerophagia in some cases may also make affected horses prone to colic (Lane 2001).

Other potential causes of right sided arytenoid collapse include guttural pouch mycosis, abscession or tumours of the head and neck or iatrogenic damage to the right recurrent laryngeal nerve as a result of trauma or perivascular injection of the right jugular vein (Goulden and Anderson 1981; Dixon et al. 2001). Finally, right sided arytenoid cartilage collapse has been described in a small number of cases where no obvious underlying cause could be determined (Lane et al. 2006a).

Laryngeal paralysis

Laryngeal paralysis, i.e. where both sides of the larynx are dysfunctional, is an uncommon finding in horses referred for investigation of abnormal respiratory noise because afflicted horses are likely to present with asphyxiation. Laryngeal paralysis most commonly arises as a result of primary hepatic disease (McGorum et al. 1999; Dixon et al. 2001). The condition has also been associated with organophosphate poisoning (Duncan and Brook 1985), lead poisoning (Sojka et al. 1996), ingestion of plants such as Lathyrus spp. (Indian vetch) and Cicer arietinum (chick pea) (Cahill and Goulden 1987), hyperkalaemic periodic paralysis (Carr et al. 1996) and overextension of the neck during general anaesthesia (Dixon et al. 1993). In these cases, a diagnosis is possible during a resting endoscopic examination (Fig 13). Dynamic bilateral arytenoid and vocal fold collapse has been reported rarely. Strand et al. (2004) reported 5 cases, associated with head flexion, in Norwegian Trotters. These horses showed normal arytenoid function at rest.

Vocal cord collapse

Whilst VCC is most commonly observed in association with ACC, dynamic collapse of one or both vocal cords has also been described in the absence of ACC (Fig 14) (Kannegieter and Dore 1995; Dart et al. 2001; Tan et al. 2005; Lane et al. 2006a). Affected horses are reported to make abnormal inspiratory sounds during exercise but invariably appear normal during a resting endoscopic examination and hence a definitive
diagnosis requires video-endoscopy during treadmill exercise. The degree of airway obstruction is less than with ACC and exercise tolerance may not be compromised, particularly if horses are exercising at submaximal intensities. The prevalence in the general population is unknown, but in horses referred for treadmill endoscopy the prevalence is reported to be 2–12% (Kannegieter and Dore 1995; Martin et al. 2000; Dart et al. 2001; Tan et al. 2005; Lane et al. 2006a).

Holcombe et al. (2006) have shown that vocal fold instability may be induced experimentally by bilateral myotomy of the cricothyroid muscle, leading these authors to suggested that VCC may arise as a result of cricothyroid muscle dysfunction, possibly as a result of damage to the external branch of the cranial laryngeal nerve. It has been suggested that iatrogenic damage to this nerve may result in vocal fold collapse after laryngoplasty where ventriculectomy/cordectomy is not performed (Holcombe et al. 2006). Dart (2006) has also reported VCC in a horse following ‘tie-forward’ surgery to correct DDSP where, again, it was suggested that iatrogenic damage to the nerve may have inadvertently occurred during surgery. Vocal fold dysfunction during exercise has also been reported in human athletes where the condition is often confused with exercise-induced asthma (Rundell and Speiring 2003). However, the aetiology of this condition in both species remains unclear.

**Axial deviation of the aryepiglottal folds**

This condition results in medial displacement of the vertical margins of one or both aryepiglottal folds during inspiration (King et al. 1991) (Fig 15) and produces an inspiratory ‘whistle’ similar to that heard in horses with VCC. The condition only arises during exercise and cannot be diagnosed during a resting endoscopic examination. A prevalence of 5–55% has been reported in horses referred for treadmill endoscopy (Kannegieter and Dore 1995; Martin et al. 2000; Dart et al. 2001; Tan et al. 2005; Lane et al. 2006a). Frequently ADAF occurs in combination with other forms of dynamic airway collapse (Tan et al. 2005; Lane et al. 2006a). Some authors have suggested a possible association with palatal malfunction (Parente et al. 1994; Parente 1997; Lane et al. 2006a). However, the condition may also occur as an isolated entity (Kannegieter and Dore 1995; Martin et al. 2000; King et al. 2001; Tan et al. 2005; Lane et al. 2006a). Axial deviation of the aryepiglottal folds has primarily been reported in racehorses. However, it has also been reported in sport horses undergoing treadmill investigation of abnormal respiratory noise and/or poor performance (Franklin et al. 2006). The clinical significance will depend on the degree of obstruction of the rima glottidis and whether other forms of dynamic airway collapse are present concurrently.

The aetiology of this condition is unclear: the aryepiglottal folds comprise a doubled layer of mucous membrane that is continuous rostrally with the glossopiglottic mucosa, lying between the base of the tongue and the ventral aspect of the epiglottis. Where ADAF occurs in combination with other forms of dynamic airway collapse, loss of arytenoid abduction and/or elevation of the epiglottis may also reduce tension on the aryepiglottic folds, allowing them to collapse into the laryngeal lumen during inspiration. However, this would not explain those cases that occur in isolation. In the absence of any muscular elements the logical explanation for ADAF is that there is excessive tissue present or that the tissues have become stretched. Recent histological examination of the aryepiglottal folds from horses with ADAF has shown focal inflammation and oedema of the folds, similar to that observed in human patients with laryngomalacia (McCluskie et al. 2006). However, it is not possible at this stage to determine whether these findings are causal or result from mechanical trauma to the folds as a result of the dynamic collapse.

**Interruptent epiglottal entrapment**

Epiglottic entrapment describes the envelopment of the epiglottis by the subepiglottic mucosa and aryepiglottal folds such that the margins of the epiglottis can no longer be seen (Fig 16). The entrapping tissue may be smooth or ulcerated. In the majority of cases, EE may be diagnosed during a resting endoscopic examination. Surveys of racehorses examined at sales or after racing indicate the prevalence of the condition to be 0.1–2% (Raphel 1982; Sweeney et al. 1991; Lane 2003; Brown et al. 2005). Occasional cases of intermittent EE may only become apparent during strenuous exercise (Morris and Seeherman 1991; Kannegieter and Dore 1995). During exercise, horses make a loud noise during both inspiration and expiration as a result of vibrations of the subepiglottal tissue that envelops the epiglottis. Some horses with intermittent EE also experience intermittent or persistent DDSP during exercise (Haynes 1983; Kannegieter and Dore 1995). The underlying cause of EE is unclear in many cases, although in some horses it has been associated with inflammation of the subepiglottic or aryepiglottic tissue, the presence of subepiglottal cysts or epiglottic hypoplasia (Haynes 1983; Linford et al. 1983).

**Epiglottal retroversion**

This is a rare form of dynamic airway collapse whereby the apex of the epiglottis retroverts and covers the rima glottidis during inspiration (Fig 17). This results in loud inspiratory sounds and significant obstruction to airflow. In contrast to earlier beliefs, epiglottal retroversion does not result in DDSP, confirming that the role of the epiglottis in stablising the caudal portion of the soft palate during exercise is limited (Parente et al. 1998). This condition has been reproduced experimentally by anaesthetising the hypoglossal nerve, indicating that paresis of the hyoepiglotticus muscle is involved in the pathogenesis of the condition (Holcombe et al. 1997a). Clinically, the condition has been seen in horses following severe respiratory infections and after surgery that may have damaged the hypoglossal nerve (Holcombe et al. 1997a; Parente 1998). In 2 of the 3 cases observed at the University of Bristol, the condition was found to be associated with enforced neck flexion (Franklin et al. 2006).
Fig 11: Right sided arytenoid cartilage collapse.

Fig 12: Rostral displacement of the pharyngeal arch in a horse with 4th branchial arch defect.

Fig 13: Bilateral laryngeal paralysis observed during a resting endoscopic examination in a horse with lead toxicosis.

Fig 14: Vocal fold collapse in the absence of any arytenoid cartilage collapse.

Fig 15: Axial deviation of the aryepiglottal folds.

Fig 16: Epiglottic entrapment. Ulceration of the entrapping mucosa can be observed in this case.

Fig 17: Epiglottal retroversion.

Fig 18: Collapse of the apex of the corniculate process of the arytenoid cartilage.
**Collapse of the apex of the corniculate process of the arytenoid cartilage**

This uncommon form of dynamic airway collapse (Fig 18) was recently described by Dart et al. (2005). Collapse of the apex of the corniculate process was observed whilst abduction of the ventral aspect of the corniculate process was maintained. Fifteen out of 309 (4.9%) of horses referred for examination of poor performance and/or upper respiratory noise during exercise were found to be affected. These were reported to be normal at rest. At the University of Bristol, the condition has been observed less frequently (5 out of 1000 horses, including 2 that developed the condition after URT surgery) (Franklin, unpublished data). Again, horses appeared normal at rest and a definitive diagnosis was only made using HSTE. Barakzai et al. (2007), however, have reported this condition in 7/133 (5.2%) Clydesdale horses examined at rest.

The pathogenesis of this condition is currently unknown. It has been speculated that the left arytenoideus transversus muscle is unable to support the dorsal apposition between the arytenoid cartilages and that, as the inspiratory pressures increase during exercise, the left corniculate process collapses under the right and into the airway (Dart et al. 2005). In the study by Barakzai et al. (2007) necropsy examination was performed in one horse and revealed an excessively wide (10 mm) transverse arytenoid ligament that allowed easy separation of the apices of the corniculate processes.

The clinical significance of this condition is unclear. However, when observed during exercise, it has been associated with other forms of dynamic airway collapse, resulting in marked airway obstruction. Dart et al. (2005) reported that in all cases, collapse of the apex of the corniculate process was followed by progressive collapse of the aryepiglottal fold and vocal fold. Four out of the 15 horses also had other forms of dynamic airway collapse. Similarly, at the University of Bristol, all afflicted horses had additional forms of dynamic URT collapse including VCC, ADAF and DDSP.

**Disorders of the trachea**

**Intratracheal prolapse of the crico-tracheal membrane**

This rare condition has received little mention in the literature (Goulden 1977) but is again reported to be associated with abnormal inspiratory noise and decreased exercise tolerance. During inspiration, the collapsing pressures within the trachea cause the membrane to prolapse upwards, resulting in some obstruction to airflow. The aetiology is unknown although it has been suggested that affected horses may have a congenitally wide space between the trachea and the first tracheal ring.

**Tracheal collapse**

Dynamic tracheal collapse has been cited as a cause of abnormal respiratory noise and exercise intolerance in a single horse in the literature (Tetens et al. 2000). Diagnosis of this condition was possible by the use of tracheal video-endoscopy during treadmill exercise.

**Conclusions**

This review has outlined the different forms of dynamic airway collapse which may affect exercising horses. The clinical significance of dynamic URT collapse will depend on the degree of airway obstruction and the exercise activity that the horse is expected to perform. There is likely to be a greater negative impact on ventilation and subsequently on performance where multiple abnormalities exist.

Clinical history and resting endoscopic findings are of limited value in predicting the events occurring during exercise in many instances. In particular, caution should be exercised when making a diagnosis of laryngeal dysfunction based on the presence of inspiratory noise alone because a variety of conditions may produce respiratory sounds which are audibly similar. At present the only means to make a definitive diagnosis of dynamic airway collapse is to perform an endoscopic examination of the airway during high-speed treadmill exercise. In the future, the development of portable systems would greatly aid the diagnosis of dynamic URT obstructions.

**References**


