Dynamic obstructions of the equine upper respiratory tract

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Obstruktiva problem i övre luftvägarna hos häst

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SUMMARY

Dysfunction of the upper respiratory tract is a common cause of impaired performance and intolerance to exercise in racehorses and include several upper airway obstructions. The ones termed dynamic obstructions are primarily seen during physical exertion and affected horses often appear to be normal during endoscope examination at rest. The correlation between diagnoses made at rest respectively exercise is low, suggesting that an examination at rest alone is likely to be insufficient.

The upper respiratory tract is exposed to great differences in pressure throughout the respiratory cycle; variations that are further altered during strenuous exercise and affect the rigidity of upper airway structures. In presence of great fluctuations in pressure, stability is achieved through coordinated and synchronous neuromuscular mechanisms. Due to nerve damage or anatomical abnormalities, these functions may be disrupted and further cause a dynamic collapse of the upper respiratory tract when pressure changes become too severe.

The etiology behind the neuromuscular dysfunction is not yet fully understood. Since the underlying cause of the obstructions is unknown, further studies are required in order to prevent the dynamic collapses from occurring in the future.
SAMMANFATTNING

Obstruktiva problem i de övre luftvägarna är en vanlig orsak till nedsatt prestationsförmåga hos trav- och galopphästar. Förändringarna uppstår i huvudsak vid fysisk ansträngning och drabbade hästar uppvisar vanligtvis inga tecken på dysfunktion vid endoskopisk undersökning i vila. Korrelationen mellan diagnos som ställts vid endoskopi i vila respektive under arbete är således låg och att enbart göra en undersökning i vila är sannolikt inte tillräckligt för att ställa en korrekt diagnos.

Hästens övre luftvägar utsätts för stora tryckskillnader under respirationscykelnas olika faser, dessa ökar ytterligare vid hård ansträngning och utgör en stor påfrestning på de anatomiska strukturerna i svalget. Ett välutvecklat neuromuskulärt samspel ser normalt sett till så att de övre luftvägarna upprätthåller sin struktur, men nervskador eller anatomiska defekter kan rubba denna funktion och vidare leda till att en dynamisk kollaps i svalget uppstår när tryckförändringarna blir för omfattande.

Den egentliga etiologin bakom förändringarna är inte helt klarlagd, varpå ytterligare studier behövs för att uppkomsten av dessa problem ska kunna förebyggas i framtiden.
INTRODUCTION

For centuries, horse breeding has resembled a fine selection of Darwinism. Descendants of greatness are regarded as equals until one of them reaches the finish line first. In that very moment, what distinguishes a failure from success?

Dysfunction of the upper respiratory tract is a common cause of impaired performance and intolerance to exercise in racehorses (Lane et al., 2006a). Several authors have come to similar conclusions (Table 1), supporting this statement further.

Table 1. Prevalence of dynamic obstructions among horses evaluated for poor performance

<table>
<thead>
<tr>
<th>Study</th>
<th>Horses in study (n)</th>
<th>Affected horses (n)</th>
<th>Affected horses (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lane et al., 2006a</td>
<td>600</td>
<td>471</td>
<td>78,5</td>
</tr>
<tr>
<td>Wallman, 2009</td>
<td>110</td>
<td>62</td>
<td>56</td>
</tr>
<tr>
<td>Witte et al., 2011</td>
<td>100</td>
<td>81</td>
<td>81</td>
</tr>
</tbody>
</table>

In terms of respiratory dysfunction, dynamic obstructions are of particular interest concerning poor performance in racehorses. The obstructions arise when soft tissue structures collapse into the airway, in part caused by changes in airway pressure present during exercise (Franklin, 2008). Dynamic obstructions increase the resistance to airflow, thereby impairing the pulmonary gas exchange and further influencing the aerobic metabolism of the horse (Lekeux & Art, 1994). If the alveolar ventilation is reduced while the horse continues to exercise at the same metabolic work rate, the amount of energy needed to maintain speed requires to be generated anaerobically (Franklin et al., 2002). This could to a certain extent explain why horses exhibit hastened fatigue when dynamic obstructions occur, but the question of why only some horses seem to be affected remain.

By studying published research, the purpose of this literary review is to create an overview of the most common dynamic airway obstructions, to find out how they arise and what we can do to prevent their occurrence in the future.

MATERIAL AND METHODS

For research, the databases of PubMed, Web of Knowledge and Scopus were used in chronological extent. Typed search words were “dysfunction AND respiratory AND larynx AND (horse OR equine)” in various combinations with “neuropathy AND roaring” and “displacement OR entrapment”. Article collections at the Veterinary Library at the Swedish University of Agricultural Sciences were also used, along with books of equine physiology and medicine.
LITERARY REVIEW

Anatomy and physiology of the upper airways

The horse possesses a complex composition of upper airway structures, creating large dimensional changes as inhaled air approaches the lungs (Figure 1). The parts of the respiratory tract that cause airway obstructions are primarily the ones that tend to collapse during physical exertion. These are particular structures of the pharyngeal cavity and the larynx.

![Diagram of equine upper respiratory tract](image)

Figure 1. Overview of the equine upper respiratory tract (Kisonaite, 2012).

Horses are compulsory nasal breathers, an evolutionary advantage that enables them to graze and still maintain olfaction and the ability to sense predators. The base of the larynx is tightly apposed to the caudal margin of the soft palate, resulting in a non-existing communication between the oropharynx and the nasopharynx. This explains why horses in respiratory distress cannot resort to oral breathing, which other animals do as high flow resistance and turbulence arises in the airways. (Holcombe & Ducharme, 2008)

Resistance and turbulence are altered as the velocity of airflow increases. During exercise, increased oxygen demand extends the respiratory minute volume of the horse markedly. At moderate exercise, minute volume is increased due to likewise increases in tidal volume and respiratory rate. When exercise becomes strenuous, the increase in minute volume is primarily caused by an increase in respiratory rate. This results in an increase of airflow velocity, further affecting resistance, turbulence and pressure in the airways. (Lekeux & Art, 1994)
The equine larynx is composed of cartilage and muscle (Figure 2) and constitutes a connection between the pharynx and trachea. The larynx contains cricoid, thyroid, epiglottal and paired arytenoid cartilages with synovial articulation between them. Various membranes and ligaments hold the apparatus together, with a suite of small, paired intrinsic muscles that connects the cartilages and influences their mutual relations. Moreover, extrinsic laryngeal musculature connects the larynx to the pharynx, tongue, hyoid bone and sternum. (Holcombe & Ducharme, 2008)

The structures of the equine upper airways are exposed to great variations in pressure during inhalation. This can be explained by Bernoulli’s principle, stating that an increase in flow velocity will reduce the pressure and further initiate a collapse of structures towards one another. Velocity of inhaled air reaches peak values in the rostral and ventral end of the nasopharynx, where the upper airway diameter is the smallest. This results in high resistance to airflow and reduced pressure. Speed is reduced as the diameter increases in the middle part of the nasopharynx, but further constriction in the laryngeal area increases air velocity again and generates turbulence in airflow. Regions of reverse airflow arise dorsal to the rostral larynx due to constrictions and abrupt changes in the geometry of the airways, further contributing to turbulence. (Rakesh et al., 2008)

The larynx is supported by cartilage, but the pharyngeal region is principally supported by skeletal muscles and relies on their contraction for stability. Throughout the respiratory cycle, neuromuscular mechanisms are essential for dilation and stabilization of airway structures (Holcombe & Ducharme, 2008). Tensor muscle contraction is shown to inhibit collapse of the soft structures in the upper airways during inspiration (Lekeux & Art, 1994). For example, M. hyoepiglotticus, the only muscle that attaches to the epiglottis, is likely to be a dilating muscle whose activity increases with breathing effort. Contraction of the muscle stabilizes the epiglottis during inspiration, preventing prolapse of it into the rima glottidis (Holcombe & Ducharme, 2008).

Figure 2. The larynx of the horse (Kisonaite, 2012).
Multiple stimuli contribute to the contraction of dilating muscles in the upper airways during intense exercise. Decreased pressure and chemical stimuli, such as hypercapnia and hypoxia, induce contraction of upper airway muscles (Holcombe & Ducharme, 2008). These stimuli are triggered by activation of different receptors in the airways; in particular sensory receptors appear to be important for dilation and stability (Lekeux & Art, 1994).

When the laryngeal mucosa is anaesthetized, dynamic collapse of the nasopharynx arise, further supporting that dysfunction occur when sensory receptors are disrupted (Holcombe & Ducharme, 2008).

**Dynamic obstructions of the upper respiratory tract**

Five different diagnoses of upper airway collapses are commonly referred to as dynamic respiratory obstructions. This since they primarily occur during strenuous exercise, when alternations in airway pressure become too severe. The disorders often appear together and complex obstructions are not uncommon, which may explain why surgical treatment of one disorder alone often is unsuccessful. (Lane *et al.*, 2006a)

**Dorsal displacement of the soft palate**

Dorsal displacement of the soft palate (DDSP) arises, as the caudal border of the soft palate is dislocated dorsally to the epiglottis (Rakesh *et al.*, 2008). Studies by Lane *et al.* (2006a) and Wallman (2009) reported DDSP as the most common cause of dynamic obstruction of the respiratory tract in performance horses. DDSP is often preceded by a general palatal instability, characterized by dorso-ventral movements described as surging and irregular, of the rostral and caudal part of the soft palate (Lane *et al.*, 2006a). The instability causes further turbulent airflow in the nasopharynx, likely to contribute to the development of DDSP by lifting the unstable palate and thereby cause a displacement. During expiration, this results in an obstruction of the *rima glottidis*.

Following displacement, horses are found to replace the palate to its normal subepiglottic position by swallowing. The soft palate can remain displaced through multiple respiratory cycles before swallowing occurs (Lane *et al.*, 2006a). Franklin *et al.* (2002) observed affected horses to either slow suddenly or to swallow repeatedly when displacement occurred, though a majority of horses continued to run for several seconds with their soft palate displaced before any response was seen.

DDSP may be either persistent or intermittent, where the intermittent displacement primarily occurs during strenuous exercise. Lane *et al.* (2006b) and Wallman (2009) found a low correlation between results from examinations at rest respectively during exercise, further supporting that the disorder is primarily dynamic. DDSP is found to cause a flow-limiting obstruction during expiration. The resulting partial failure of gas exchange is likely to explain the impairment in performance of these horses (Franklin *et al.*, 2002). Affected horses are often described as snoring or gurgling during expiration, this low frequency sound is most likely generated by vibrations of the displaced soft palate. However, respiratory noise might as well be intermittent or absent in affected horses (Lane *et al.*, 2006a; Wallman, 2009).
The etiology of the condition is not clearly understood, but likely to be multifactorial and there are several theories of the origin. One probable cause is neuromuscular dysfunction, possibly induced by inflammation or infection of the upper airways (Couroché-Malblanc et al., 2010). This hypothesis is further supported by the development of DDSP in healthy horses when the pharyngeal branch of N. vagus is anaesthetized (Holcombe et al., 1997). Several authors have come to the conclusion that young horses are more likely to develop DDSP (Lane et al., 2006; Wallman, 2009). A possible cause of this relationship might be the high prevalence of pharyngeal lymphoid hyperplasia (PLH) in young racehorses, since upper respiratory inflammation may cause DDSP (Couroché-Malblanc et al., 2010).

**Laryngeal hemiplegia**

Laryngeal hemiplegia (LH) is commonly referred to as recurrent laryngeal neuropathy (RLN), since the cause of the dysfunction is confirmed to be neuropathy of one of the recurrent laryngeal nerves (Derksen et al., 2001). Most commonly, the left nerve is affected. Recurrent laryngeal neuropathy is considered a distal axonopathy, meaning that larger myelinated nerve fibers degenerate proximally from the end plate toward the cell body, leading to atrophy of the muscles that the axon innerves. The condition may be related to the length of the nerve fibers of the left recurrent laryngeal nerve, which are the longest motor neurons in the horse. This could possibly also explain why larger horses seem to be predisposed. It is also possible that the distal axonopathy has an inherited basis. (Rush & Mair, 2004)

Other possible causes of nerve damage initiating the axonopathy are presence of abscesses, tumours or infections in the upper airway region. There is also evidence of a correlation between nerve damage and the anatomical proximity to the left jugular vein, suggesting that damage may be caused by thrombophlebitis or perivascular injections of irritants (Davenport-Goodall & Parente, 2003).

The neuropathy leads to dysfunction and atrophy of the intrinsic laryngeal muscles, in particular M. cricoarytenoideus dorsalis (Rush & Mair, 2004). The defect can be either persistent or intermittent and affect either one or both arytenoid cartilages, though most commonly the left is affected. In examination, a failure of complete abduction of the cartilage is seen, resulting in a dynamic movement of the cartilage of the affected side towards the midline of the rima glottidis during inspiration. However, Lane et al. (2006b) found that 19 % of horses diagnosed with severe laryngeal hemiplegia at rest were able to maintain full abduction of the arytenoid cartilages during exercise. The condition severely narrows the opening to the trachea and laryngeal hemiplegia is often accompanied by ipsilateral vocal fold collapse. The defect is also known to give rise to a characteristic inspiratory noise, often referred to as roaring. (Derksen et al., 2001)
**Axial deviation of aryepiglottic folds**

Axial deviation of the aryepiglottic folds (ADAF) is a true dynamic dysfunction, only seen during strenuous exercise. The defect appears during vigorous inspiration, as the membranous part of the aryepiglottic folds deviates axially towards the *rima glottidis* (King *et al.*, 2001). When ADAF occurs in combination with other dynamic respiratory disorders, it is suggested that loss of arytenoid cartilage abduction or elevation of the epiglottis may reduce tension of the folds and thereby enable them to collapse towards the laryngeal lumen during inspiration (Franklin, 2008). When ADAF occur solely, it is suggested that the only possible explanation of the dysfunction is that tissues have become stretched (Lane *et al*., 2006a) or presence of excessive tissue (Franklin, 2008), since no muscular elements are present.

King *et al.* (2001) observed asymmetrical defects in a majority of affected horses, often with the right side appearing worse than the left. There was also evidence of a correlation between the diagnosis of ADAF and abnormal respiratory noise during inspiration, but no further association between the severity of ADAF and the incidence of noise could be proven to exist.

**Epiglottal retro flexion**

Epiglottal retro flexion (ERF) arise when the apex of the epiglottis retroverts and thereby covers the *rima glottidis*, causing an obstruction of the entrance to the trachea. The result is significant reduction of inspiratory airflow, along with high incidence of inspiratory noise. The condition has been experimentally induced by blocking the *N. hypoglossus*, suggesting that paresis of *M. hyoepiglotticus* is involved in the pathogenesis. Additionally, the condition has been reported following severe respiratory infections and surgeries that might have damaged the nerve, supporting this theory further. (Franklin, 2008)

**Epiglottal entrapment**

Epiglottal entrapment (EE) can be either persistent or intermittent. The obstruction occur when the aryepiglottal and subepiglottic mucosal folds envelopes the epiglottis, turning it inside out. While exercising, these horses make loud noise during the entire respiration cycle, due to vibrations in the subepiglottic mucosa following both inspiration and expiration. (Franklin, 2008)

The etiology of the condition is not fully understood, but epiglottic hypoplasia is considered a predisposing factor (Davenport-Goodall & Parente, 2003) and inflammation of subepiglottic or aryepiglottic tissue has also been associated with the development of EE (Franklin, 2008).
Dysfunction of the upper respiratory tract is a common cause of poor performance and intolerance to exercise in racehorses (Lane et al., 2006a; Wallman, 2009; Witte et al., 2011). The reason for this is not fully known, which truly emphasizes the need of further studies. In order to prevent the obstructions from arising in the future, the originating cause of the neuromuscular dysfunction needs to be known. Several etiologies of the collapses have been discussed, some of them proven to be more likely than others. Evolution of laryngeal anatomy, genetic predisposition and presence of airway infections are some of the suggested causes of dysfunction. However, further studies are required before any of them may be confirmed or ruled out.

It is possible that changes of the anatomical constitution of the equine upper airway have reached beyond individual differences, evolutionary creating a new standard among racehorses. This would explain why diagnosis of dynamic obstructions is far more common today than it was a decade ago, but this may just as well be due to improved diagnostic techniques. Breeding of racehorses is nevertheless a true survival of the fittest and it is not very likely that the most successful horses have suffered from respiratory defects. It may further be discussed whether we simply have reached the endpoint of equine strain, if the pressure changes in general are too severe for soft structures of the airway to handle. This would again explain why the prevalence of the obstructions has increased, but it contradicts the fact that the horse making the hardest physical effort reaches the finish line first.

There are suspicions of an inheritance of a genetic predisposition for certain collapses (Rush & Mair, 2004). If this is found to be true, equine racing federations may have to consider ruling out affected horses from breeding. Not so much because of the choking horses themselves, since no one is likely to want an offspring from a horse that always finished last. Operated horses may, on the other hand, show no sign of earlier disorder and achieve great results, while possibly passing on native defects to a future offspring. Ruling these horses out would though require access to veterinary journals and hard detective work in order to map all individuals with faults corrected. No owner could be expected to report this voluntarily, since exclusion from breeding could equal great financial losses.

It is generally known that exercising with upper airway infections may result in future obstructive problems. Many horses undergo pre-race endoscope examinations to ensure that they do not suffer from a pharyngitis that may influence their performance, but what many seems to have forgotten is that just as much damage could be done during exercise. Performing endoscope examinations or blood samplings before every training session is not possible out of several perspectives, leading to the conclusion that the very best must be to avoid irritation of the upper airway mucosa from the beginning. The value of good ventilated stable environments and high quality roughage to can therefore not be emphasized enough.

Lymphoid hyperplasia (PLH) is commonly found in the pharynx of racehorses and considered a harmless condition when the horses are still young. In older horses, it is interpreted as a sign
of upper airway inflammation and a general precaution is always taken considering racing these horses. Young horses often continue to race and exercise as before, even though diagnosed with PLH. Dorsal displacement of the soft palate (DDSP) is also found to be more common among young horses (Couroché-Malblanc et al., 2010) and there is a suggested connection between PLH and the high prevalence of DDSP in young individuals. This could imply that these follicles may cause more damage than earlier assumed, stating that this possible connection needs to be further investigated.

Another suspected cause of dysfunction is perivascular injections meant for the left jugular vein, possibly damaging the N. laryngeus recurrens and thereby inducing laryngeal hemiplegia (Davenport-Goodall & Parente, 2003). Damage could potentially be done to the nerve by perivascular injections of irritants or excesses from trombophlebitis caused by permanent catheters. Given that this is true, it may be questioned why injections are still preferably made in the left jugular vein. Injecting in the right jugular vein is seldom a problem, except for being more technically difficult for right-handed to manage. Some horses lack a functioning right jugular vein and certain surgical procedures requires the permanent catheter to be placed in the left jugular due to the head position of the horse. Otherwise, no disadvantages of right-sided jugular injections are to be found. Presence of laryngeal hemiplegia may not be of importance to an average riding horse, but the consequences could be fatal to a racehorse, why right-sided injections truly should be considered when treating racehorses.

Dynamic obstructions of the upper airways arise due to presence of great fluctuations in pressure, generated by intense breathing. This implies that examinations at rest are likely to leave out conditions that solely occur during physical exertion. Further, it is known that certain conditions, which appear at rest, are corrected during exercise (Lane et al., 2006b). The relevance of such examination alone may for that reason be questioned.

Today, fine surgical methods are developed to correct the dynamic obstructions and many surgeons perform the procedures on routine. We should ask ourselves to what limit it is reasonable to correct the faults, without dedicating a minute to consider why they are so common. When affected horses are diagnosed, they are likely to already have experienced choking several times. It is therefore of particular importance to find the cause of the dysfunctions, so that horses never have to choke from the very beginning in the future. When we have reached that point, there will be no further need of advanced surgeries.

Post surgery, there is a lack of reported success rate of the procedures. Just as successful they may be, there is also a constant risk of contributing to further nerve damage. It is thereby possible to give rise to one particular dysfunction while performing surgery to correct another. No one knows if horses will be reluctant to make a physical effort again with the memory of choking present, making the result of a surgical procedure even more insecure. After all, no one can be blamed for not running in dyspnea. And maybe, that lack of access to oxygen can be distinguished as the difference between a failure and success.
REFERENCES


A great thank you to the very best K for the illustrations (copyright Konstancia Kisonaite, 2012).