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Review of respiratory noises ('Kehlkopfpfeifen') in the horse

Marianne M. Sloet van Oldruitenborgh-Oosterbaan

Department of Large Animal Medicine and Nutrition, Faculty of Veterinary Medicine, Utrecht University, The Netherlands

Summary

Respiratory stridor and eventually decreased performance may be caused by a variety of upper respiratory diseases. In Warmbloods the implications of these problems are often different than in Thoroughbreds. This paper discusses clinical and endoscopic examinations of the upper respiratory tract and reviews the possible causes of a respiratory noise.

Keywords: horse, respiratory noises, exercise, laryngeal hemiplegia, dorsal displacement of the soft palate

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Übersicht über respiratorische Geräusche ("Kehlkopfpfeifen") beim Pferd

Abnormale respiratorische Geräusche und gegebenenfalls ein herabgesetztes Leistungsvermögen können durch eine Vielzahl von Erkrankungen der oberen Atemwege verursacht werden. Bei Warmblütern und Vollblütern sind die Begleiterscheinungen dieses Problems oft verschieden. In dieser Studie wird die klinische und endoskopische Untersuchung der oberen Atemwege diskutiert und ein Überblick über mögliche Ursachen eines respiratorischen Geräusches gegeben.

Schlüsselwörter: Pferde, respiratorische Geräusche, Belastung, Hemoplagia laryngis, Gaumensegel

Introduction

Abnormal respiratory noise (stridor) is a common complaint of horse owners. The problem seems to occur more frequently in Warmblood sport horses than in Thoroughbred or Standardbred racehorses. This might be related to the use of the horses. In racehorses the presence of a respiratory noise is not considered a problem as long as there is no exercise intolerance. In sport horses and horses used for recreational purposes, the noise per se is considered to be a problem and the reduced exercise tolerance is often not even noticed. In Germany, the problem is even more complicated: "Kehlkopfpfeifen" (read: all respiratoiry noises from the upper airways) is defined as all respiratory noises caused by chronic problems in the throat or trachea that are not responsive to therapy. The buyer of a horse may return the horse to the seller if the disease is diagnosed within 14 days after purchase. The purpose of the present review, however, is to give an overall picture of diseases that may cause respiratory noises at rest and/or during exercise, but not to solve this forensic problem in Germany.

The most common cause of respiratory noise is laryngeal hemiplegia *(Zeller* 1973). However, dorsal displacement of the soft palate, diseases of the nasal passages, epiglottic entrapment, epiglottic cysts, pharyngeal lymphoid hyperplasia, pharyngeal stricture and dynamic pharyngeal collapse also cause respiratory noises *(Robertson* 1991). Incidentally, progressive ethmoidal hematoma or artheroma may cause a respiratory noise, but other symptoms will usually be visible before the noise becomes audible.

History and physical examination of the respiratory tract

Taking an accurate history is important, although this aspect of the examination is often overlooked *(Beech* 1991). Considering the problem of a respiratory noise during exercise important questions are:

* exact complaint
* duration and progression of the noise
* does the noise wax and wane or is it always present during exercise
* does the sound increase with speed, or does it change with head position
* how long has the owner/trainer had the horse
* was the problem present at the time of purchase or did it develop subsequently

Physical examination should be done at rest and after exercise. Often it is necessary to evaluate the respiratory noise of the horse during exercise while standing beside the track. A better option, however, is to examine the horse during exercise on a treadmill, both without and with an endoscope.

At rest the breathing pattern, the effort and the rate should be noted. In most cases of respiratory noise of the upper airways, no abnormalities will be found at rest. However, if the horse extends its head and neck to breathe and seems anxious, stenosis of the upper airway should be suspected *(Beech* 1991). Air flow from both nostrils should be equal. If a nasal discharge is present, take note of the type of discharge, whether it is unilateral or bilateral, and whether discharge is only seen under certain conditions. A foul smell may be caused by tissue necrosis, certain infections and neoplasms.

Carefully inspect the head and neck, with regard to facial symmetry, nostril width, general attitude and expression, nasal drainage and cranial nerve dysfunction, and listen to the horse breathing.
After this, the head and neck are visually inspected and palpated in detail. One should check the nasal passages, false nostrils, thickness of the nasal septum, width of the nasal meatus, mucosal integrity and width of the intermandibular space. Palpation of the larynx for symmetry or muscle atrophy is frequently performed in the Thoroughbred, but is disappointing in the Warmblood.
The trachea can be palpated for anomalies such as fractured or irregular cartilage rings.
During the examination one should also look for scars or other signs of previous surgery on the larynx or neck muscles.
Endoscopic examination of the upper respiratory tract provides a ready diagnosis of most diseases that cause abnormal noise by inducing morphologic changes. This endoscopy is best performed with the horse restrained in stocks *(Rose* and *Hodgson* 1993). Laryngeal function tests that are useful during endoscopy include partial nostril occlusion to stimulate laryngeal abductor movement and flushing water through the endoscope to stimulate swallowing. In many horses with stridor, however, endoscopic examination in the resting animal reveals no overt abnormalities. In such cases, diagnosis of the cause of the noise is speculative at best and endoscopic examination while the horse is exercising on a treadmill is indicated *(Ducharme* 1992).

Diseases causing respiratory noise

Nasal passages abnormalities

The incidence of nasal cavity disease in the horse is low *(Robertson* 1991). In most cases, there is not only a respiratory noise at rest and/or during exercise, but also nasal discharge, malodorous breath and/or facial swelling *(Robertson* 1991).
Progressive ethmoidal hematoma (PEH) are slowly expanding angiomatous masses that appear to originate principally from the mucosal lining of the frontal, maxillary and sphenopalatine sinuses *(Pascoe* 1990). PEH is identified as an irregular, reddish-greenish soft mass often with some local hemorrhage around its margins. PEH is an example of a disease that normally is identified before it causes a respiratory noise, because of the intermittent unilateral epistaxis while the horse is at rest *(Lindsay* 1992). Small PEH can be treated transendoscopically using an Nd:YAG laser, while large lesions need surgery often in combination with the Nd:YAG laser *(Nickels* and *Tulleners* 1992).
Masses in the false nostrils, such as atheromas, may incidentally impair airflow during exercise *(Beech* 1991). This unilateral condition is easily recognised and usually seen in young horses (1–3 years) *(Robertson* 1991). The etiology is unknown, but it is likely due to aberrant epithelial tissue remaining after embryogenesis. Treatment is usually requested from a cosmetic point of view *(Rose* and *Hodgson* 1993) and consists of surgical resection or drainage *(Robertson* 1991).
Alar fold obstruction occurs when the alar folds are drawn into the nasal passage during inspiration producing a loud vibrating nasal noise *(Robertson* 1991). *Caron*, however, describes a biphasic fluttering noise that is most pronounced on expiration (*Caron* 1991). It is diagnosed by retracting both folds with a large temporary mattress suture and observing the horse during exercise. If there is a partial or complete resolution of the nasal obstruction, bilateral alar fold resection is indicated *(Robertson* 1991).
Congenital nasal septal cysts are usually discovered in weanling-age foals when the cysts expand to a size that produces airway obstruction and sometimes a nasal stridor *(Nickels* and *Tulleners* 1992, *Robertson* 1991). Surgical intervention usually solves the problem.
Other nasal septum diseases, like neoplastic infiltration, amyloid deposition, nasal polyps, or nasal septum cartilage degeneration are rare *(Beech* 1991, *Robertson* 1991). All these cases need surgical intervention and often have a guarded to unfavorable prognosis *(Nickels* and *Tulleners* 1992). Occasionally a horse with a septal thickening will present a diagnostic challenge if there is absence of facial deformity, no palpable abnormalities of the rostral septum, and lack of obvious narrowing on endoscopic examination *(Robertson* 1991). The loud nasal respiratory noise heard during exercise should provide a hint of the problem and diagnosis has to be confirmed radiographically *(Robertson* 1991).
Fungal granulomas of the nasal passages only occur in tropical and subtropical climates and have never been diagnosed in Utrecht.

Pharyngeal lymphoid hyperplasia (PLH)

Pharyngeal lympoid hyperplasia, also known as follicular pharyngitis or lymphoid follicular hyperplasia, is frequently observed in young horses where it is considered to be a normal physiologic finding *(Robertson* 1991).
The mucous membranes of the dorsal and lateral walls of the nasopharynx and the dorsal surface of the soft palate contain numerous lymphoid follicles. On the surface of the epiglottis small lymphoid follicles may also be observed *(Robertson* 1991). These follicles vary from small white dots beneath the mucosa to large (several millimeters) pink edematous masses. The follicles usually regress as the horse matures. Several grading systems are used to describe the severity of the lymphoid hyperplasia, but the disadvantage of a grading system is that what is "normal" in a two-year-old would be regarded as severe pharyngitis in an eight-year-old.
Severe PLH has historically been associated with abnormal respiratory noise and possibly exercise intolerance, presumably due to turbulence and reduction of the pharyngeal airway size caused by dynamic collapse during high speed exercise (*Tulleners* et al. 1992). However, the real contribution of PLH to clinical signs remains unanswered (*Shoemaker* and *Haynes* 1992a).
The etiology of PLH is not clear, but it seems multifactorial *(Robertson* 1991). It is suggested that PLH may be enhanced by exposure to one or more respiratory viruses (*Shoemaker* and *Haynes* 1992a). In most cases, treatment of PLH is not necessary and not really possible, although intensive vaccination, pharyngeal sprays containing antimicrobial and anti-inflammatory medication, rest for one or two months, corticosteriods, systemic antibiotica, and even electrocauterisation and liquid nitrogen cryotherapy have been used (*Tulleners* et al. 1992).

Pharyngeal stricture

Nasopharyngeal cicatrix is characterized by a stricturing web of tissue in the wall of the nasopharynx and has been reported in horses of all ages, sexes and breeds (*Shoemaker* and *Haynes* 1992a). Until now, it has never been diagnosed in Utrecht. Clinical signs include exercise intolerance, abnormal inspiratory dyspnea, and abnormal vocalization (*Shoemaker* and *Haynes* 1992a, *Tulleners* et al. 1992). For horses with a severely stenotic nasopharynx, removal of the cicatrix with laser or dilatation of the cicatrix with a bougie should be considered (*Tulleners* et al. 1992).

Dynamic pharyngeal collapse

Dynamic pharyngeal collapse has been described by *Ducharme* (1992) as consisting of three abnormalities: dorsal displacement of the soft palate (DDSP), unilateral or bilateral ventral displacement of the roof of the nasopharynx and dorsal displacement of the epiglottis. We believe that DDSP is a distinct entity, and that the other two phenomena often happen together and should be considered as one entity "dynamic pharyngeal collapse". In such cases no abnormalities are found on endoscopic examination at rest, but during exercise there is an inspiratory dyspnea and the nasopharynx is almost completely obstructed as result of ventral displacement of the roof of the nasopharynx, medial displacement of the walls of the nasopharynx and puffing up of the soft palate (without dorsal displacement). The cause is unknown and there seems to be no therapy.

Idiopathic left laryngeal hemiplegia

Idiopathic left laryngeal hemiplegia (ILH) is mainly diagnosed in young horses of the large breeds. The larynx on the affected side cannot fully dilate and this produces an obstruction to airflow *(Beech* 1991). In Utrecht, most horses with laryngeal hemiplegia are presented with a complaint of making an abnormal inspiratory noise during exercise; decreased exercise tolerance is seldom a problem. The noise is usually characterized as an inspiratory whistle or a roar, hence the term 'whistler' and 'roarer' have become synonymous with laryngeal hemiplegia. The paralysis is usually left sided and may be partial or complete. Right sided and bilateral paralyses are occasionally encountered.
The performance consequences of altered symmetry and movement of the arytenoid cartilages other than complete paralysis are unclear (*Hackett* 1992). At least 40% of the large breed horses have some degree of laryngeal asymmetry or asynchrony, but the effect of such abnormality on an individual's athletic ability is often a matter of speculation (*Hackett* 1992). It is suggested that asynchrony or partial paralysis precedes total paralysis (*Hackett* 1992) and that some horses with partial paralysis will undergo abductor muscle fatigue and suffer progressive compromise of the airway during exercise. The most reliable way to evaluate whether a respiratory noise is the sequella of hemiplegia, is to perform laryngeal endoscopy while the horse exercises on the treadmill.
In Thoroughbreds, atrophy especially of the cricoarytenoideus dorsalis can often be determined by palpation, in Warmbloods this is difficult or even impossible.

In Dutch Warmbloods, we use a grading scale (see below) based on that used to describe the radiological alterations of the navicular bone, and sesamoid bone (*Wagenaar* 1992), because horse owners are accustomed to interpreting the meaning of the different figures (0=ideal and 4=bad). Endoscopy is performed at rest while the horse stands in stocks restrained with a nose twitch. Horses are evaluated untouched, after nasal occlusion, during swallowing and with a slap test. The 'slap test' is advocated as a means of detecting laryngeal dysfunction. Some believe that the horse should be sedated *(Beech* 1991), but in Utrecht we consider the test helpful in the non-sedated horse with a nose twitch. While the examiner performs the laryngoscopy a helper slaps the horse behind the shoulder and withers over the dorsal thorax; if carefully done the opposite cartilage should move. If the helper slaps too hard both cartilages may move. The pathway seems to ascend through the cranial thoracic and cervical spinal cord segments, to the medulla, with the reflex movement descending through the vagus and recurrent laryngeal nerves (*Byrne* 1992). Disadvantages of the test are that it becomes less responsive to repeated stimulation, and is absent in tense or frightened horses, as well as in horses with lesions of the cervical spinal cord (*Speirs* et al. 1992). If a horse needs sedation no grading score is given.

Grading:

0 completely normal symmetrical arytenoid cartilages; normal synchronous movement; synchronous full abduction and adduction of both arytenoid cartilages; arytenoids respond to slap test
1 variable slight asymmetry and/or variable slight asynchronous movement of one of the arytenoid cartilages (usually the left), or a permanent but barely visible asymmetry; full abduction and adduction are possible; arytenoids respond to slap test
2 mild asymmetry continuously present and/or mild asynchronous movement of one of the arytenoid cartilages (usually the left); full abduction and adduction are possible; arytenoids respond to slap test
3 moderate asymmetry continuously present and/or a moderate asynchronous movement of one of the arytenoid cartilages (usually the left); full abduction and adduction are (almost) possible; the diseased arytenoid does not always respond to slap test
4 marked permanent asymmetry of the arytenoid cartilages, the diseased arytenoid cartilage does not move at all, or only slightly; full abduction and adduction are not possible and the diseased arytenoid does not respond to slap test

Horses are always evaluated through both nostrils as the angle of view affects the appearance of the larynx.
The pathogenesis of the idiopathic form of laryngeal hemiplegia remains obscure, although it is clear that the disorder results from a demyelination of the left recurrent laryngeal nerve, denervating the muscles of the larynx *(Rose* and *Hodgson* 1993). In some horses with laryngeal paralysis, however, a specific etiology can be identified, such as damage to the recurrent nerve as a sequel to perivascular inflammation, guttural pouch mycosis, strangles abscessation, neoplasms of chest and neck, or as a complication of surgical procedures of the neck, such as esophagostomy or tracheal reconstruction *(Beech* 1991).

The therapy is surgical. None of the currently used surgical techniques will restore complete and normal function to the larynx, but some do provide obvious improvement *(Beech* 1991).

Chondritis of the arytenoid cartilages

Chondritis is a condition most commonly affecting young (race) horses in which one or both arytenoid cartilages undergo dystrophic changes, including enlargement, inflammation, mineralization and the deposition of granulation tissue *(Rose* and *Hodgson* 1993). The most common signs are decreased exercise capacity and an inspiratory stridor. The cause of the condition is not known, although trauma to the cartilages, particulary by stomach tubes and infection, has been implicated *(Rose* and *Hodgson* 1993).
Endoscopy of the larynx reveals a change in shape of the arytenoid cartilage, reduction in the size of the laryngeal opening, ulcers on the mucous membranes, and possibly granulation tissue *(Rose* and *Hodgson* 1993). Lateral radiographs may reveal the presence and extent of mineralization of the affected tissues. Surgical intervention is possible with a moderate success rate *(Rose* and *Hodgson* 1993).

Epiglottic entrapment

Epiglottic entrapment is characterized by envelopment of the rostral epiglottis and its margins by subepiglottic tissue and the aryepiglottic folds (*Shoemaker* and *Haynes* 1992b). Epiglottic entrapment is a common cause of abnormal respiratory noise and exercise intolerance, but may be an incidental endoscopic finding (*Speirs* et al. 1992). The etiology of epiglottic entrapment is unknown. Although a congenital hypoplastic epiglottis is a predisposing factor, entrapment may also occur as an acquired event in adult horses with a normal-sized epiglottis (*Shoemaker* and *Haynes* 1992b). Surgical correction of epiglottic entrapment may be performed by transendoscopic contact YAG laser axial division, transnasal or transoral axial division using a curved bistoury, transendoscopic electrosurgical axial division, or surgical excision through a laryngotomy or pharyngotomy (*Speirs* et al. 1992).

(Sub)epiglottic cysts

The most common location for cysts within the pharynx is in the subepiglottic area *(Rose* and *Hodgson* 1993). The most common clinical signs in older horses (2–5 years old) are reduced performance capacity and an inspiratory and expiratory noise that is only prominent during exercise *(Rose* and *Hodgson* 1993). Dysphagia may be reported but is rare *(Rose* and *Hodgson* 1993). In foals, infrequently, subepiglottic cysts are reported. In this age group the cysts are often extremely large and produce dyspnea at rest, and possibly nasal discharge, dysphagia and an increased potential for aspiration pneumonia *(Rose* and *Hodgson* 1993).
The diagnosis is made on endoscopic examination and it is important to make the horse swallow, since some cysts may not be visible until this maneuver is accomplished *(Rose* and *Hodgson* 1993). Therapy involves removal of the offending cyst.

Dorsal displacement of the soft palate

Dorsal displacement of the soft palate (DDSP) is a disorder in which the soft palate becomes displaced from its normal position under the epiglottis to lie over the opening of the larynx. This results in turbulent and inefficient airflow with a marked reduction in performance. DDSP may be intermittent or persistent *(Rose* and *Hodgson* 1993). Once the soft palate becomes displaced, the horse slows down or stops and usually produces an abnormal gurgling respiratory noise that is thought to be caused by vibration of the soft palate and mouth breathing (*Speirs* et al. 1992). The noise is most commonly of expiratory origin but sometimes can be heard on inspiration as well.
DDSP usually occurs during fast exercise, particularly when the horse is pulling hard *(Rose* and *Hodgson* 1993). In Utrecht we find it hard to provoke the condition during treadmill exercise, but the use of a bit and long reins may improve the chances that the disorder will occur on the treadmill *(Rose* and *Hodgson* 1993).
Some horses, especially if excited, show a temporary DDSP during endoscopy, which disappears quickly after swallowing.
The cause of DDSP is not defined, although it may be a sequel to a variety of inflammatory diseases of the upper airway or to hypoplasia of the epiglottis *(Rose* and *Hodgson* 1993).
A definitive diagnosis of DDSP can be difficult to determine, which makes management of the condition difficult as well (*Speirs* et al. 1992). The use of a tongue-tie is helpful in some horses because it reduces caudal retraction of the tongue. Caudal retraction of the tongue elevates the soft palate and pushes the larynx caudally, both of which may predispose to DDSP (*Speirs* et al. 1992).
Predisposing factors, like upper airway infection or inflammation, epiglottic entrapment, laryngeal hemiplegia and arytenoid chondrosis, should be treated first, followed by adequate rest (*Speirs* et al. 1992). If these techniques do not prevent DDSP surgical intervention may be necessary.

Rostral displacement of the palatopharyngeal arch

Rostral displacement of the palatopharyngeal arch, also known as dorsal laryngopalatal dislocation, usually induces dysphagia, nasal regurgitation of food, coughing and respiratory compromise with abnormal noise at exercise *(Knottenbelt* 1994). At endoscopic examination the apices of the arytenoids will not be visible. The extent of the displacement can be exaggerated by nasal occlusion, exercise or sedation. If the condition is mild, there may be no clinical signs at rest, but when greater airflows are required the firm muscular nature of the dorsal pharyngeal arch and the tension exerted within it, are sufficient to prevent effective abduction of the arytenoids *(Knottenbelt* 1994). This causes a noise that is more prominent at inspiration than at expiration. There is no therapy.

Outside structures pressing on larynx / pharynx

The most common cause of pressure on the larynx/pharynx area is retropharyngeal abscessation as a result of severe lymphadenopathy that accompanies strangles (Streptococcus equi) *(Rose* and *Hodgson* 1993). Other possible causes are guttural pouch tympany, guttural pouch empyema, gut-

tural pouch hemorrhage or a cyst in the mucosa of the dorsal nasopharynx *(Knottenbelt* 1994). In most cases the enlargement of lymph nodes or guttural pouches is also visible externally.

Tracheal stenosis

Dorsoventral flattening of the trachea is a condition that most commonly has a congenital origin. Ponies and miniature horses are most commonly afflicted. Clinical manifestations of a collapsed trachea are a severe inspiratory dyspnea and loud inspiratory noises. Primary tracheal collapse has been reported in mature horses that demonstrated exercise intolerance and some degree of dyspnea *(Gaughan* 1995). Possible causes include strangles infection, neoplasia and trauma to the tracheal cartilages. The therapy is surgical tracheal reconstruction but this has a very guarded prognosis.

Conclusion

Endoscopic examination of the respiratory tract at rest facilitates a diagnosis in many cases of respiratory noise. Some problems, like arythenoid chondritis, sometimes require radiographic examination to evaluate the extent of the alterations. If making a diagnosis at rest is impossible, endoscopic examination during exercise is indicated. Training horses for endoscopic evaluation on the treadmill takes three to four days and 4 to 6 training sessions if the horse has been well handled before admission; in young and/or inexperienced horses a much longer training period might be necessary. It also seems advisable to evaluate horses on the traedmill before any surgery as some cases will prove to be impossible to correct or will show a completely different picture during exercise than at rest.

References

Beech, J. (1991): Examination of the respiratory tract. In: Equine respiratory disorders. Ed. *Beech, J.* Lea & Febiger, Philadelphia, pp 27–40.

Byrne, B. A. (1992): Diagnostic procedures. In: Equine surgery. Ed. *Auer J.* W.P. Saunders Company, Philadelphia, pp 554–565.

Caron, J. P. (1991): Diseases of the nasal cavity and paranasal sinuses. In: Equine medicine and surgery 4th ed. Eds. *Colahan P. T., Mayhew, I. G., Merritt, A. M.* and *Moore, J. N.* American Veterinary Publications, Goleta, California, pp 386–398.

Ducharme, N. G. (1992): Dynamic pharyngeal collapse. In: Current therapy in equine medicine 3. Ed. *Robinson, N. E.*, W.B. Saunders Company, Philadelphia, pp 283–284.

Gaughan, E. M. (1995): Surgery of the upper respiratory tract. In: The horse: diseases and clinical management. Eds. *Kobluk, C. N., Ames, T. R. and Geor, R. J.*, W.B. Saunders Company, Philadelphia, pp 243–261.

Hackett, H. P. (1992): The significance of arytenoid cartilage movement. In: Current therapy in equine medicine 3. Ed. *Robinson, N. E.*, W.B. Saunders Company, Philadelphia, pp 285–289.

Knottenbelt, D. C. and *Pascoe, R. R.* (1994): Conditions of the respiratory tract. In: Diseases and disorders of the horse. Eds. *Knottenbelt, D. C.* and *Pascoe, R. R.*, Mosby Wolfe Publishing, London, pp 91–156.

Lindsay, W. A. (1992): Ethmoidal hematoma. In: Current therapy in equine medicine 3. Ed. *Robinson, N. E.*, W.B. Saunders Company, Philadelphia, pp 274–275.

Nickels, F. A. and *Tulleners, E. P.* (1992): Nasal passages. In: Equine surgery. Ed. *Auer J.*, W.P. Saunders Company, Philadelphia, pp 433–446.

Pascoe, J. R. (1990): Ethmoid hematoma. In: Large Animal Internal Medicine. Ed. *Smith, B. P.*, C.V. Mosby Company, St. Louis, pp 557–559.

Robertson, J. T. (1991): Pharynx and larynx. In: Equine respiratory disorders. Ed. *Beech, J.* Lea & Febiger, Philadelphia, pp 331–387.

Rose R. J. and *Hodgson, D. R.* 1993): Respiratory system. In: Manual of equine practice. Ed. *Rose R. J.* and *Hodgson, D. R.*, W.B. Saunders Company, Philadelphia, pp 134–169.

Shoemaker, R. S. and *Haynes, P. F.* (1992a): Pharyngeal lymphoid hyperplasia and pharyngeal stricture. In: Current therapy in equine medicine 3. Ed. *Robinson, N. E.*, W.B. Saunders Company, Philadelphia, pp 282–283.

Shoemaker, R. S. and *Haynes, P. F.* (1992b): Epliglottic entrapment and cysts. In: Current therapy in equine medicine 3. Ed. *Robinson, N. E.*, W.B. Saunders Company, Philadelphia, pp 281–282.

Speirs, V. C., Tulleners, E. P., Ducharme, N. G. and *Hackett, R. P.* (1992): Larynx. In: Equine surgery. Ed. *Auer J.*, W.P. Saunders Company, Philadelphia, pp 460–4480.

Tulleners, E. P., Schumacher, J., Johnston, J. and *Richardson, D. W.* (1992): Pharynx. In: Equine surgery. Ed. *Auer J.*. W.P. Saunders Company, Philadelphia, pp 446–459.

Wagenaar, G. (1992): De veterinaire keuring van het paard. Wetenschappelijke Uitgeverij Bunge, Utrecht, pp 54–78.

Zeller, R. (1973): Kehlkopfpfeifen, Diagnose, differential Diagnose, forensische Beurteilung. Tierärztl. prax. 1, 163–168.

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Marianne M. Sloet van Oldruitenborgh-Oosterbaan

Department of Large Animal Medicine and Nutrition
Faculty of Veterinary Medicine, Utrecht University
Yalelaan 16
3584 CM Utrecht
The Netherlands
phone .. 31 30 2531112 fax .. 31 30 2531256