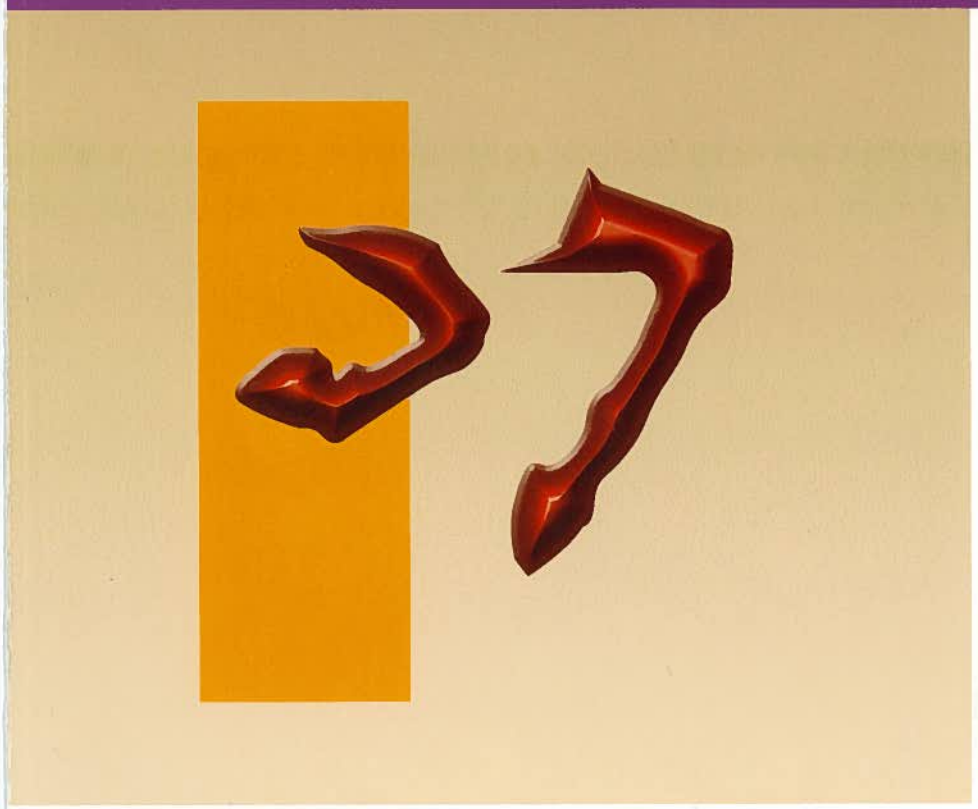
GANADERÍA

# IV Congreso Internacional de Medicina y Cirugía Equina

SICAB´03

28 al 30 de noviembre de 2003. Sevilla



JUNTA DE ANDALUCIA

Consejería de Agricultura y Pesca

# IV Congreso Internacional
# de
# Medicina y Cirugía Equina

**IV CONGRESO INTERNACIONAL DE MEDICINA Y CIRUGÍA EQUINA**



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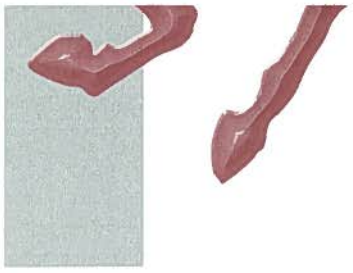

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***Palacio de Exposiciones y Congresos FIBES***

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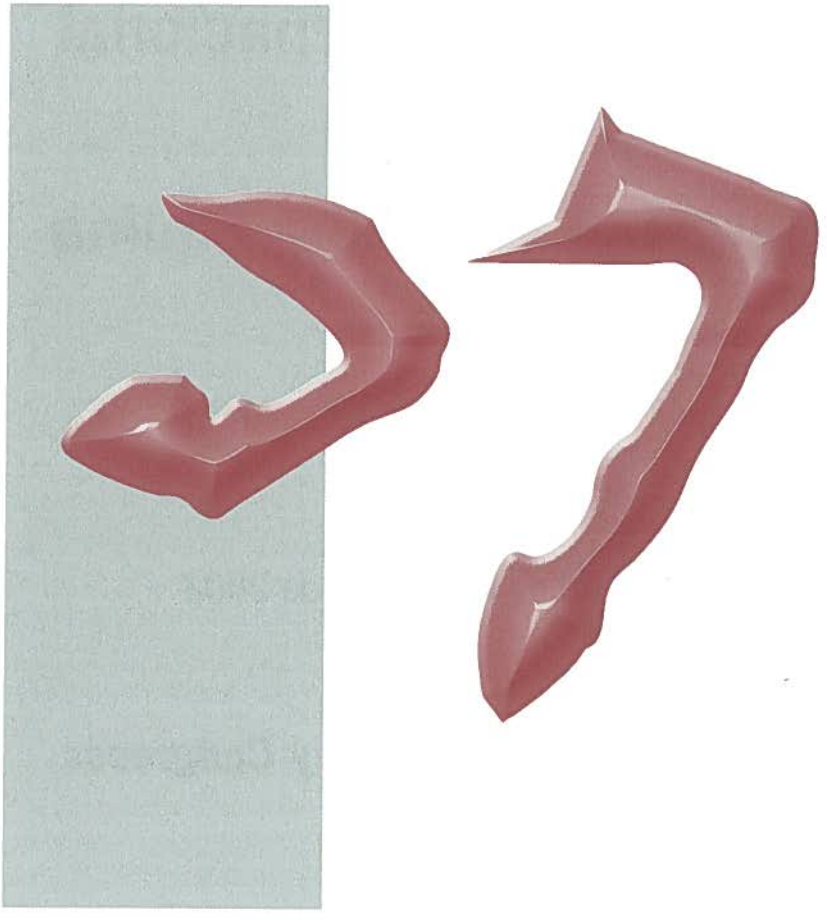

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- Laboratorios Intervet
- Equinvest

# ÍNDICE

# EQUINE RESPIRATORY VIRUS NEWS

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## Background

- Most of the upper respiratory tract infections in horses are caused by one of two groups of viruses
    - o The Influenza group
    - o The Herpes group.
- Now we are hearing more about Rhinovirus.
- We do know that there are many outbreaks that don't appear to be caused by herpes or influenza
- Influenza and Herpes virus can co-exist in the horse

## Equine Herpes

- Equine herpes virus 1 (EHV-1) and 4 (EHV-4) infect the respiratory tract
- EHV-1 may also infect white blood cells, subsequently causing a viremia and dissemination of the virus to the reproductive tract or central nervous system (CNS).
- EHV-4 infections are usually limited to the upper respiratory tract and are a common cause of respiratory disease in young horses.

## Equine influenza virus

- An important characteristic of influenza viruses is their ability to undergo antigenic drift
- Fortunately, drift is slower for equine influenza than for human influenza virus.
- Antigenic drift allows the virus to avoid neutralization by antibody present in the equine population and thus infect seropositive animals.
- Strains of A/equine/2 viruses have been responsible for all known outbreaks of the disease since 1980.
- Remains in circulation in most parts of the world
- Recent studies of viral antigenic drift have identified two diverging lineages of equine-2 influenza

- The American lineage, which is now widespread
- The Eurasian lineage, which has been rarely isolated (once)in the western hemisphere.

## Equine influenza virus vs Equine Herpes: Clinical differences?

- The influenza virus tends to cause more coughing while the herpes virus causes more of a snotty nose.
- But there is a lot of overlap in symptoms and the two can be hard to differentiate clinically.
- Shown that virus can be coughed over 30 ft, thus easy to spread

## Why bother to diagnosis which virus?

- Many clinicians consider definitive diagnosis as unnecessary since therapy is often empirical and laboratory confirmation is often obtained after clinical resolution.

## Let's rethink this approach!!

- Maybe we can justify this approach based on legitimate issues concerning treatment, recovery period, control and surveillance.

  Is influenza occurring in vaccinated horses?
- Get those field strains into the labs so that current field viruses get into the newer vaccines.
- Need to keep the vaccines moving forward
- If it is influenza you should worry more about the potential to spread and may wish to use barrier clothing.
- Professionally, it adds credibility to know what a horse has rather than always just saying "I think it is Flu"
- With the new IN vaccine, you may want to know if is influenza, as you may then want to vaccinate in the face of an outbreak, as there is some early proof that this MLV vaccine may block receptors and stop the spread of the virus??

## Working up a case

- While the standard methods of virus isolation and serology have not changed significantly, additional laboratory tests are now available.
- Increase the sensitivity (Influenza is difficult to grow)
- Decrease the time for a test result.

## Diagnostic Testing : Influenza

- Directigen Flu A: detection of viral antigens
- Virus isolation
- Serologic testing

### Directigen Flu A

- These kits were designed for humans but have been tested on horses with excellent results. Becton-Dickinson Micro Systems and Centaur
- Designed to detect influenza antigens in nasal secretions of humans.
- It happens that the antigens are present in equine influenza strains also.
- These tests are easy, completed in 15 minutes and accurately rule in or out influenza as the cause of the symptoms.
- The test is very accurate on nasal secretions when used during the first 2 days of remarkable clinical signs.
- The test is somewhat less accurate on horses that have been ill longer than a few days of exhibiting mild signs.

Nasopharyngeal swabs are better for this harder to detect group.

In field conditions

- o Sensitivity (33-70%)
- o Specificity (78-98%)

- Reported to be comparable to or better than virus isolation.

## Virus Isolation

- Nasal swab samples are taken by passing a swab as far as possible into the horse's nasopharynx via the ventral meatus to absorb respiratory secretions.
- Should be taken as promptly as possible.
  - o Results of experimental challenge studies suggest that peak viral titers are obtained during the initial 24 to 48 hours of fever, on the second or third day after infection, and duration of viral shedding is usually not more than 4 or 5 days.
- Swabs should be transferred immediately to a container with virus transport medium and transported on ice to maintain viability of the virus.
  - o Virus is unlikely to survive if dry swabs are taken and there is increased chance of contamination if bacterial transport medium is used.
  - o Sterilized custom-made swabs and transport medium can be obtained from your reference laboratory.

## Serological Testing

- A four-fold increase in convalescent serum neutralizing antibody titer collected over a two-week period is diagnostic.
- Serological detection of influenza virus infection is rarely useful in immediate clinical management.
- Retrospective diagnosis may establish a clinical diagnosis in the absence of virus isolation or antigen detection.
- The hemagglutination inhibition (HI) test measures the capacity of influenza-specific antibody present in serum samples to inhibit the agglutination of red blood cells by virus.

- If horses are vaccinated in the face of infection, it is not possible, using the HI and SRH assays, to determine whether any increase in antibody levels is due to vaccination or infection.
- An ELISA to detect antibody to the non-structural protein NS1, which is in development, has the potential to differentiate between vaccine- and infection-induced antibodies.

## Diagnostic Testing : Herpes

- Virus isolation
- Polymerase chain reaction (PCR)
- Immunofluorescent detection of viral antigens
- Serologic testing

## Virus isolation:

- Virus can be isolated from nasopharyngeal swabs.
- Since herpes viruses are sensitive to chemicals and variations in temperature and humidity, veterinarians should contact their diagnostic laboratory for transport media and for specific collection and transportation procedures.
- One method is to use a sterile gauze swab attached to a flexible metal wire placed in the nasal passages and pharynx to obtain material for virus isolation.
- Swabs should be placed immediately in media and refrigerated or frozen until transported to the laboratory.
- Virus may also be isolated from citrated or heparinized whole blood.
- Viremia frequently occurs during clinical signs of respiratory tract, CNS, or reproductive tract disease.
- Nasopharyngeal secretions may be evaluated by immunoperoxidase staining for EHV-1/4 antigen.

## Polymerase chain reaction (PCR)

- Amplification of viral DNA using PCR is a rapid, sensitive and increasing utilized assay for detection of EHV-1 or EHV-4 respiratory tract infection
- Portions of the same clinical specimens used for virus isolation can be processed for herpesvirus detection by type-specific PCR.

## Immunofluorescent detection of viral antigens

- Antigen detection methods also exist for the rapid diagnosis of EHV-1 and EHV-4 URTD directly from clinical material.
- Cells from nasopharyngeal secretions can be stained using immunofluorescent antibodies that reveal the presence of the herpesviral antigens.
- When direct antigen detection methods are used for a rapid laboratory diagnosis of EHV-1 or EHV-4, it is important to confirm the direct test results by virus isolation.

### Serologic testing: Antibody titers

- A four-fold increase in convalescent serum neutralizing antibody titer to EHV-1/4 collected over a two-week period is diagnostic.
- Because most horses have been exposed to, or vaccinated against, EHV-1/4, positive titers are common, making interpretation of a single sample difficult.
- Natural infection usually causes a rapid and dramatic increase in titer such that serum must be collected early in the course of the disease to demonstrate an increasing titer.

## Equine Influenza Prevention

- A modified live intranasal vaccine
- Developed by attenuating a field strain of the virus with same technology used for the human influenza vaccine.
- Protects naïve animal 11 months and older against both North American and European strains of the virus
- Protection is achieved after a single application of the vaccine
- Efficacy has been proven with several independent challenge trials.

## Equine Rhinovirus

- Infection occurs world-wide
- A major cause of clinical respiratory infection?
- The Equivir IgG ELISA
- Detects IgG antibodies that have been generated in response to infection by Equine Rhinovirus type 2 in equine serum or plasma.

## Control

- Control of an outbreak relies on early diagnosis and isolation of affected animals.
- Viral shedding may continue for up to 10 days. It is critical to realize that infected animals may appear clinically normal and still shed virus.
- This is the most common source of spread to new premises.
- All sick and contact animals should be quarantined for a period of 2 to 3 weeks.
- Cessation of exercise and training for all contact animals is important since there is an increased incidence and severity of disease in animals that continue training.
- It is important to also recognize the potential for spread by fomites.
- The virus is highly susceptible to detergents and commonly used disinfectants.
- Environmental and personal hygiene can help limit the spread of an outbreak.

SICAB Conference on Equine Respiratory Disease, Seville, 28 - 30 November 2003

# RADIOLOGY OF THE EQUINE HEAD

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## Introduction

The excellent contrast which exists between air, bone and, to a lesser extent, soft tissues renders the equine head a highly suitable subject for diagnostic radiography. However. many practitioners are deterred by the perception of insurmountable difficulties in the making and interpretation of radiographs in this area. First, it is falsely imagined that the output of X-rays required to penetrate the bony structures of the facial region is beyond the capacity of equipment normally at their disposal. This is not the case because acceptable results can be obtained with relatively low output units, particularly if cassettes with rare earth intensifying screens are available. Second, it is felt that the anatomy of the area is intimidating and that interpretation of films of this area is beyond the ability of mere mortals. Such an attitude generally stems from a lack of defined objectives and provided that a logical approach is adopted worthwhile results can be obtained consistently. Radiography of the head and neck should be employed to compliment other diagnostic modalities, most notably endoscopy. It can often provide an additional diagnostic dimension particularly when access is limited or details of changes in the depth of tissues are sought.

## Indications

Unilateral **nasal discharge** is the commonest single reason for radiography in this region and in many instances recognisable radiological signs will be present on erect lateral films. Other presenting signs for which radiography is likely to be helpful include **facial swelling or deformity; trauma; unilateral epistaxis not caused by guttural pouch mycosis; and obstruction of the nasal airways.** In contrast bilateral nasal discharges generally arise from the lower respiratory tract and the source can usually be confirmed by endoscopy so that radiography becomes unnecessary. Radiography of the heads of horses showing behavioural or neurological disturbances is also consistently unrewarding except as an adjunct to the assessment of animals after overt trauma. The **temporo-mandibular joints** are not easily examined radiographically and **ultrasonography** provides more definitive information.

one of the following circumstances will apply:

1. **Unilateral foul-smelling nasal discharge**
2. **Anomalous dental eruption**
3. **Maxillary swelling rostral to the facial crest**
4. **Swelling and/or discharging sinus tracts at the ventral aspect of the mandible**
5. Eating patterns and behaviour suggestive of dental pain
6. **Fracture of a cheek tooth has been seen on oral inspection**
6. **Before dental extraction to check the line of repulsion**
7. **After dental extraction** to confirm that there are no residual dental fragments or damage to adjacent tooth roots.
8. In the evaluation of **mandibular fractures** to establish whether there has been injury to dental structures.
9. Oral neoplasia.

Helpful radiographic information regarding the **soft tissues** of the pharynx, larynx and adjacent structures can be obtained in a wide range of clinical situations:

1. Palpable **swellings in the laryngeal, parotid and submandibular regions of the throat**
2. Compression or distortion of the airways or upper alimentary tract by intra-mural or extra-mural lesions, i.e. **when luminal distortion can be seen at endoscopy but the cause cannot be determined**
3. Suspected or endoscopically visible intra-luminal lesions, e.g. swellings, masses, foreign bodies, strictures or persistent displacement of structures from their normal positions, i.e. **permanent dorsal displacement of the soft palate**
4. Some disorders of the auditory tube diverticula (ATDs)
5. **Severe obstructions of the upper respiratory tract** when endoscopy may not be feasible
6. **Dysphagia**

## Radiographic Techniques

In the logical management of clinical cases, common-sense dictates that as much information as is safely possible is obtained with the horse in the standing position. Thus, it is simpler to perform radiography after the initial endoscopic investigation per nasum as this will generally help to localise the source of clinical signs and aid the rational use of radiography. The following projections and techniques are used in the radiography of the equine head:

1. **Erect lateral**
2. **Lateral oblique** to isolate the individual dental arcades
3. **Ventro-dorsal** of the nasal chambers and paranasal sinuses
4. Lesion-oriented oblique views to sky-line areas under suspicion
5. Intra-oral occlusal

6. Studies of deglutition by plain radiographs and fluoroscopy using ingesta impregnated with contrast medium
7. Carotid angiography to outline the branches of the carotid circulation as they relate to the ATDs.

The **erect lateral view** alone is almost always sufficient for the investigation of the soft tissues of the throat but generally forms one of a series of projections in the investigation of suspected nasal, sinus or dental disorders. Sedation is often needed to ensure that the patient does not move during exposure and remains correctly positioned. The X-ray tube should be held at head level and if a stand is not available the top platform of a step ladder may be used.

Safe practice to protect all personnel from exposure to ionising radiation must be observed. In the context of equine head radiography a light beam diaphragm is required and it should be illegal for film cassettes to be held by hand even with lead gloves. For radiography of the head in standing horses suspension in a bag attached to a drip stand or similar support is effective. Additional protection is provided by backing the cassette with a lead sheet. One of the primary objectives of this view is **to identify free fluid in the paranasal sinuses** and if small quantities of fluid are present, the horizontal interface may become obscured by the overlying tooth roots. To some extent this limitation can be overcome by avoiding a position with the head extended. Thus, the poll should be semi-flexed. The centring point for the erect lateral projection is midway between the medial canthus and the rostral limit of the facial crest. The are exposed should extend from the level of the commissures of the lips to the midpoint of the zygomatic arch and include the nasal chambers, maxilla and all of the paranasal sinuses.

The **erect lateral view** is invaluable in the decision-making process in the management of horses with sinu-nasal disorders as, in combination with the results of endoscopy, it generally yields sufficient information to determine (1) whether there is disease of any form in the area, (2) whether this can be managed conservatively, (3) whether further investigations in the standing position are needed, i.e. direct sinus endoscopy or (4) whether a general anaesthetic is required to complete the diagnosis before proceeding to surgery.

Only in exceptional cases are projections other than the straight lateral of any value in the investigation of the soft tissues of the throat because oblique views make for highly confusing anatomical orientation and in the ventro-dorsal plane the cranium and vertebral column overly the site. For investigation of the pharynx, larynx and ATDs the beam is centred at the midpoint between the base of the ear and the angle of the mandible. For the more distal soft tissues of the throat, a series of overlapping projections centred over the trachea is required and these are more easily achieved with the head lowered.

The superimposition of the left and right arcades renders lateral views of the head unreliable for the diagnosis of dental diseases and, therefore, the **30° lateral oblique projection** is required to separate the two jaws.. It is possible to achieve this view in conscious horses particularly with the advent of modern effective chemical restraint, and with the head of the sedated horse supported on a table of appropriate height.

For the lateral oblique view the patient is positioned with the jaw under examination nearest to the film and with the beam angled at 30° from the vertical in a root-to-crown direction. For horses with narrow jaws, the angle should be increased slightly. The primary beam should be centred at a point 6-7 cms dorsal to the rostral limit of the facial crest on the uppermost side of the face for the underlying maxillary arcade and at the lower border of the upward lying mandible for the lower arcade. For intra-operative lateral oblique projections, for example when confirming the progress of an extraction procedure, it is impracticable to turn the horse over for the surgical site to be adjacent to the film. Thus, a compromise is necessary and the radiograph must be made with the affected side lying uppermost. The 30° angle is then reversed in a crown-to-root direction. Although this method is not radiographically ideal because of the increased subject / film distance, the results will be adequate, for example, to confirm the line of repulsion or to identify residual dental fragments.

**Ventro-dorsal projections** are best made under general anaesthetic with the patient in dorsal recumbency. The structures of the head are more radiodense in this plane and, thus, a grid is essential to eliminate scatter and the exposure factors must be increased. The purposes of this view are to provide a means to compare the diseased nasal chamber with the healthy side and to show how far the condition has extended in both latero-medial and rostro-caudal planes. Clearly it is advantageous for the exposure to be made as near parallel as possible to the midline septum and a foam trough is useful to obtain straight films. The endotracheal tube should be removed while the exposure is made. The beam should be centred at the midline, at a level half way along the horizontal mandibular ramus.

**Lesion-oriented obliqued views** can help to demonstrate superficial lesions of the supporting bones of the face or lower jaw such as fractures or masses and this projection can often be made in the conscious horse.

The **intra-oral occlusal view** (film in mouth) is usually used to isolate the upper or lower incisors from the teeth in the occluding jaw or for the examination of the structures of the rostral nasal cavities without superimposition of the mandible.

**The radiographic investigation of deglutition** may be indicated in cases of dysphagia and generally involves the study of contrast-impregnated ingesta as it passes through the pharynx and oesophagus. A mixture of bran mash and barium sulphate is generally accepted voluntarily by horses although on occasions liquid barium sulphate suspension may be introduced by syringe into the mouth. This latter technique may also be used to outline the oropharynx and to delineate abnormal structures - so-called contrast pharyngogram. Plain lateral radiographs taken shortly after the administration of contrast medium are far from ideal as it is a matter of chance whether the exposure is made at a moment of diagnostic value. Gantry-mounted units where the tube head and image intensifier move in unison are usually only to be found in teaching hospitals, but this is the only satisfactory means to make dynamic studies of deglutition. It is generally simple to incorporate a video-recorder into the system to allow slow-motion analysis of swallowing sequences later.

**Carotid angiography** may be used to demonstrate abnormalities of the carotid circulation which are present in all cases of guttural pouch mycosis. The technique depends upon the infusion of an iodide-based contrast medium into the common carotid artery to outline the branches of the carotid artery as they pass in the walls of the ATD. This technique is rarely needed in clinical cases because the site of haemorrhage can usually be identified by endoscopy. However, it does have a role in those few instances when no other means is available to decide whether the internal or maxillary branch of the carotid is diseased.

## Radiographic interpretation

### Nasal chambers and paranasal sinuses

Anatomical orientation is essential before attempting to identify abnormalities. In the lateral view of the nasal and sinus areas the landmarks to find first include the ethmoid turbinate "onion"; the concho-frontal and frontal sinus compartments; the infraorbital canal; the roots of the cheek teeth; and the dorsal and ventral conchae in the rostral nasal region. Whenever the dental arcade is incompletely shown on this view, the third cheek tooth can be identified because it has the longest reserve crown in contrast to the fourth tooth which has the shortest roots. The general features of the bony septa in the paranasal sinuses may be confusing because they are not only variable between horses, but also between the two sides of the same horse. However, the dorsal outline of the maxillary sinuses can usually be seen as dome shaped curving lines against the background of air in the conchofrontal sinus and in some horses the bulla of the RMS is particularly obvious. The septum which divides the RMS from the CMS can be seen as a vertical line arising between the fourth and fifth cheek teeth. There are two normal features which can be mistaken for 'lesions' by novices. First, the rostral wall of the RMS arises as an irregular dense bony plate immediately dorsal to the roots of the third cheek tooth and might be misinterpreted as a dental periapical reaction. Second, the globe of the eye provides a discrete soft tissue density which in straight lateral projections is obscured by the overlying ethmoidal "onion" where the rims of the orbital margins provide the outer skin of the onion. However, in films which are slightly obliqued the bony margins of the two orbits will be seen separately and the eye may appear rostral to the ethmoids imitating a soft tissue mass such as a progressive ethmoidal haematoma. As the length of the reserve crowns shortens with age the roots become more pointed and the radiolucent dental sacs disappear. In young horses the infraorbital canals may be partly obscured by the tooth roots, but with age the maxillary sinuses become more air-filled and the horizontal elongated bony tubes of the canals become more obvious.

The superimposition of the dental arcades and mandibles limits the availability of the sinuses for radiological interpretation on ventro-dorsal projections. However, this view is valuable to compare the changes within the nasal chambers. The radiographic midline septum consists of the vomer bone, the nasal septum which is largely cartilaginous and the bony thickening where the two nasal bones are apposed. The position of the septum and jaws will establish whether the patient was correctly aligned for the exposure but the septum may be displaced by expansive space-occupying lesions towards the contralateral chamber. The contents of the compartments of the maxillary sinuses lateral to the dental arcades can be compared and extension of sinus disease into the adjacent conchae will be seen.

The check-list below summarises the radiological signs which may be identified relating to the nasal chambers, paranasal sinuses and dental arcades, together with the most likely causes of these changes .

| Radiological signs | Possible causes |
|---|---|

**a) Shown on standing lateral radiographs**

| | |
|---|---|
| 1. Free fluid levels: | Usually mucopus/pus, occasionally blood. Sometimes localised within compartments. Trephination with or without direct endoscopy will differentiate pus from blood. Blood may arise from trauma or ethmoidal haematoma. The discharge in cases of dental apical infection will always be malodorous |
| 2. Localised opacification of sinus compartments: | Early primary sinus empyema; dental periapical infection; mycotic sinusitis/rhinitis. In primary empyema the RMS may be the only compartment involved - note the caudal bulla of this structure for distension |
| 3. Generalised opacification: | Chronic sinus empyema - absence of free fluid may indicate inspissation of pus; sinus cyst formation, especially if lesion has expansive appearance with well defined outline; neoplasia |
| 4. Circumscribed soft tissue masses: | Ethmoidal haematoma - these are usually evident at the margins of the ethmoid "onion" and note that the lesions can be multiple; early sinus cyst; neoplasia |
| 5. Increased bony density around tooth roots - osteitis: | Dental periapical infection - localised loss of definition and coarsening of the bony architecture typify these lesions but a 30° oblique view is needed to make a definitive diagnosis |
| 6. Mineralisation of the soft tissues within the nasal chambers and sinuses: | This is a non-specific finding where the cause may range from incidental dental cementosis to extensive rhinitis with conchal metaplasia; some sinus cysts have well-defined mineralised walls |
| 7. Facial distortion: | Trauma; expansive lesions; sinus cysts; neoplasia. Facial distortion rostral to the facial crest is likely to be caused by dental disease, but caudal to it the reverse is true. |
| 8. Bone destruction: | Malignant neoplasia; expansive lesions including sinus cysts and benign neoplasia. |

**b) Additional changes shown on 30° oblique films**

| | |
|---|---|
| 1. Absence of cheek teeth / extra cheek teeth: | Previous dental extraction; congenital partial anodontia – also supernumerary cheek teeth are sometimes seen horses most commonly caudal to CT6. False diagnoses may result from a positioning artefact of the radiograph. |
| 2. Deformity of crowns and/ or reserve crowns: | Maleruption; dental fractures; overgrowth of occluding tooth after loss of tooth in opposing arcade. |
| 3. Periapical lucency and/or root distortion. Divergence of lamina dura: | Periapical infection. |
| 4. Increased bony density around roots - osteitis: | Dental disease with infection. Do not confuse with other densities in the background. |
| 5. Periapical cementosis: | Increased cement production in response to infection - note that clearly defined cement 'pearls', in the absence of other changes, are occasional incidental findings. |
| 6. Shrinkage and/or flattening of tooth roots: | Space-occupying lesion of maxillary sinus compressing onto roots, i.e. sinus cyst or neoplasia. |

**c) Shown particularly on ventro-dorsal radiographs**

| | |
|---|---|
| 1. Increased density confined to maxillary sinus | Sinus empyema; sinus cyst |
| 2. Circumscribed mass in nasal chambers, sinuses: | Progressive ethmoidal haematoma; sinus cyst; sinus empyema confined to RMS causing medial distension; neoplasia. |
| 3. Nasal septum displacement: | Space-occupying lesions: sinus cyst; extreme cases of empyema with distension of sinus compartments by inspissated pus; neoplasia. |
| 4. Destruction of conchal pattern rostral to sinuses: | Destructive rhinitis; mycosis. |
| 5. Irregular mineralisation of conchae | Conchal metaplastic ossification - "coral" formation - invariably the result of extension of infection from dental disease, usually involving one of the three most rostral cheek teeth. |

**d) Shown on lateral oblique radiographs of the mandible**

| | |
|---|---|
| 1. Sclerosis of the ventral cortex: | Dental periapical infection; dental impaction; trauma; neoplasia. |
| 2. Discrete areas of rarefaction at the roots of several teeth | Normal dental sacs from which the mineralised tissues of the permanent cheek teeth will differentiate. |
| 3. Localised rarefaction at the root of a tooth with divergence of the lamina dura and a zone of sclerotic bone: | Dental periapical infection. In some cases sinus tracts can be seen passing through the mandibular cortex. |
| 4. Fissures through the ventral cortex of the mandible: | Sinus tract from periapical abscess; fracture of the mandible. The extent of bony reaction will depend on the duration and stability of the fracture. Also devitalisation of the teeth through which the fracture line passes is likely and this will add to the overall reaction. |
| 5. Circumscribed areas of rarefaction within the mandibular medulla: | Dental sacs; developmental cysts. |
| 6. Localised areas of intense radiodensity: | Odontogenic neoplasia such as odontoma, ameloblastoma, cementoma. |
| 7. Diffuse increased radiodensity: | Fibrous dysplasia. |

## Soft tissues of the throat

The air-filled ATDs are usually triangular and form useful reference points between the base of the skull and the pharynx, but the pouches themselves and the structures which pass through them are rarely exactly superimposed. The outlines of the smaller lateral compartments can be distinguished separately. Dorsally, the basilar portions of the occipital and sphenoid bones stand out clearly. The stylohyoid bones can be seen crossing the ATDs and nasopharynx, but their rostral extremities are partly obscured by overlying teeth and mandibles. The thyrohyoid is not visible as a distinct structure but the rostral tips of the thyroid cartilages are mineralised at their junction to form a small radiodense 'tear-drop' at the base of the epiglottis.

The nasopharyngeal airspace is situated immediately ventral to the ATDs, separated from them only by a thin band of soft tissue. Rostrally, the shadow of the nasopharynx, where it merges with the choanae, is lost amidst the maxillary dental structures. The ventral limit of the nasopharynx is marked by the dorsal border of the soft palate, the epiglottis and the larynx. The airspace bordered by the palatopharyngeal arches contours around the apices of the corniculate processes of the arytenoid cartilages and extends a little caudal to them at the oesophageal additus in a blunted funnel shape. This landmark is important in the evaluation of laryngo-pharyngeal relationships.

Caudal to the nasopharynx, the cricopharyngeal sphincter is normally closed except at the instant of deglutition, so it is not distinguishable from the surrounding soft tissues. Similarly, in normal horses, air is almost never to be seen in the proximal oesophagus. Although the width of the soft tissues which occupy the area between the ventral border of the second cervical vertebra and the trachea and nasopharynx should be only slightly greater than the dorso-ventral width of the nasopharynx, this dimension varies considerably with the head position of the horse.

The laryngeal ventricles are seen as paired oval radiolucent shadows immediately caudo-ventral to the arytenoid cartilages. After ventriculectomy these shadows should no longer be visible although the extent to which they are radiographically ablated very much depends on the technique used. Between the dorsal border of the epiglottis and the arytenoid cartilages, the undulant shadows of the aryepiglottic folds cross the nasopharyngeal airspace. The area beneath them represents the course of the lateral food channels, the piriform recesses. The contour of the dorsal border of the soft palate is also undulant and occasionally a triangular air shadow can be seen in the oropharynx between it and the base of the tongue, but more often the two structures are in contact and therefore indistinguishable.

The cartilages of the larynx are generally difficult to distinguish although, sometimes, the dorsocaudal prominences of the thyroid wings are mineralised and can be seen as rectangular radiodensities where they are attached to the cricoid. The crico-tracheal junction is usually marked by a distinct narrowing of the airway but the tracheal rings are rarely sufficiently mineralised to be seen clearly. On under-exposed films the lobes of the thyroid glands may be seen overlying the proximal trachea and should not be mistaken for abnormal masses.

The presence of an endotracheal tube obscures and distorts the anatomical relationships within the airways and if radiographs of this region are to made on an anaesthetised patient, the tube should be withdrawn while the exposure is made.

## Radiological signs relating to the soft tissues of the throat

Whenever there is an increase in the volume of soft tissue abutting onto the pharynx, larynx or within the ATDs compression or distortion of the airspaces will be evident. The cause of such swellings may be abscessation, lymph node enlargement, cellulitis or neoplasia. The earliest radiographic signs of retropharyngeal soft tissue enlargement are increase in the distance between the second cervical vertebra and the nasopharyngeal airspace with thickening of the strip of tissue between the ATDs and the nasopharynx. The volume of the ATDs is reduced as these structures become flattened ventro-dorsally and the nasopharyngeal airspace is also narrowed with loss of its funnel-shaped dorsal contour and obliteration of the rostral margins of the arytenoid cartilages. Bubbles or steaks of gas in the fascial plains indicate emphysema which may be due to the presence of gas-forming organisms or indicate penetration of the wall of the upper airway, pharynx or oesophagus.

Although horses with empyema of the ATDs usually have a copious nasal discharge, drainage is occasionally obstructed, for example by chondroids, so that swelling and/or dyspnoea are the major presenting signs. In such cases, obliteration of the ATD air shadows is almost total and compression of the nasopharynx is likely to be visible.

Soft tissue masses developing in sites too deep to be palpated may produce obstruction of the pharyngo-laryngeal airway by compression or by luminal occupation. Sub-epiglottic cysts are seen on radiographs as masses which are sited rostral to a dorsally deviated epiglottis and which may distort the outline of both epiglottis and palatal arch. The commonest cause of intraluminal swelling is chondritis of the arytenoid cartilages. Alterations in the outlines of the affected structures are readily seen endoscopically but radiography provides an additional dimension. The changes which may be present range from minor irregularities of the outline of the corniculate processes to gross swelling with foci of mineralisation.

Other causes of intraluminal masses include cysts, granulomas and neoplasia, which may arise in a variety of sites and sizes. Congenital cysts associated with the palatal arch may occur within the sheet of the soft palate or be attached to it by a pedicle. Contrast pharyngograms may help to delineate the more diffuse lesions.

Whenever the soft palate is **permanently displaced dorsally**, the epiglottis is not available for endoscopic inspection, and this constitutes a firm indication for radiography because both structures and their relationships with each other can be assessed on a lateral projection. The objective of this technique is to identify the predisposing cause of the persistent DDSP before resorting to further investigation under general anaesthesia. Radiography is not indicated for exertion-induced DDSP as studies have suggested that there are no radiographic differences between normal and afflicted horses.

Epiglottal entrapment whereby the epiglottis becomes enveloped by the glosso-epiglottic and aryepiglottic mucosae is a possible cause of persistent DDSP which can be recognised reliably on radiographs. Most other cases can be diagnosed by endoscopy per nasum but an appreciation of the length of the epiglottis is not straightforward from this perspective. However, this may have a crucial influence on the prognosis for surgery if the epiglottis is hypoplastic.

Rostral displacement of the palatopharyngeal arch is a feature which can be recognised on radiographs in some cases of fourth branchial arch defect (4-BAD) which are discussed at length elsewhere in these proceedings. Suffice it to say here that the radiographic evidence for this condition consists of absence of the cricopharyngeal sphincter with a continuous air column extending from the nasopharynx to the proximal oesophagus. A "dew drop" formed by the caudal pillars of the palatal arch is seen positioned rostral to the apices of the corniculate processes of the arytenoid cartilages.

Most disorders of the ATDs can be diagnosed by endoscopy but radiographs of these structures can be useful to demonstrate fluid levels, filling with inspissated pus or chondroids. Tumours or other swellings may cause indentations into the airspaces. Fractures at the junction of the basisphenoid and occipital bones may occur in horses which have sustained violent head trauma by rearing over backwards. Apart from blood showing as free fluid in the floor of the ATDs, a step in the base of the skull will be visible on radiographs. This can make a valuable contribution to prognosis as horses which have sustained this injury may well be unable to rise and show a range of neurological deficits from which they unlikely ever to recover. Proliferative bony changes may be seen on the proximal stylohyoid bone in cases of chronic otitis media with temporohyoid osteoarthrosis.

Fluoroscopy with image intensification is an invaluable aid to define the cause of **dysphagia** when the pharyngeal phase of deglutition is compromised. The passage of ingesta from the oropharynx can be observed for the efficiency of the pharyngeal stripping waves; the sites of retention of contrast in the pharynx; reflux into the nasopharynx; aspiration into the larynx and trachea; dilation of the cricopharynx and the initiation of primary oesophageal peristalsis. Dysphagia through functional or structural disorders of the oesophagus can be examined using contrast medium with plain films or fluoroscopy. The site of physical obstruction by impacted ingesta may be confirmed without contrast medium, but other causes of obstruction such as localised dilatations, strictures and megoesophagus may be identified. Poor or absent oesophageal motility is a feature of equine dysautonomia (grass sickness) and it is advantageous to be able to confirm this diagnosis early in the course of the disease to save the patient from prolonged distress and to avoid futile intensive care.

Distortions in the outline of the cervical trachea by extraluminal swellings such as abscesses or by congenital or acquired narrowing of the airway are readily identified on lateral radiographs of the throat.

## Conclusion

Although endoscopy is undoubtedly the most valuable and direct method of examining the equine upper airways and alimentary tract, radiography can make an important contribution to diagnosis. In a number of conditions it may provide essential information with regard to the mural and extramural soft tissue structures which cannot be fully appreciated by endoscopic inspection from the lumen of the tracts._

There are specific circumstances when ultrasonography has advantages over radiography in the investigation of disorders of the equine head and throat i.e. to investigate the retro-bulbar soft tissues and to help to distinguish between solid and fluid-filled masses in the throat. The role of alternative imaging techniques such as scintigraphy is being evaluated for the diagnosis of dental periapical inflammation.

There has been a recent increase in interest in the value of scintigraphy in the diagnosis of head and neck disorders in the horse. Although some authors have been enthusiastic about its value in the diagnosis of dental disease the lack of anatomical accuracy is a major limiting factor. in this author's experience it can be very valuable in the diagnosis of dental disease before radiographic signs have appeared.

# PREVENTION AND TREATMENT OF *STREPTOCOCCUS EQUI VAR EQUI*

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## Introduction

*Streptococcus equi* var *equi*, a Lancefield Group C (*Streptococcus*) is the cause of equine strangles, a highly contagious purulent lymphadenopathy of the head and neck. Strangles is one of the most feared equine diseases due to its severity and prevalence. Established outbreaks may last for months or even years, particularly in large horse populations, with frequent new arrivals providing a continuous supply of susceptible animals

## Prevention

### Control of Strangles in Affected horses

Control of strangles after outbreaks has relied on a four-week quarantine period to give time to detect intermittent shedding of *S. equi* that occurs with some convalesced cases. Three negative weekly nasopharyngeal swabs are taken as a minimal evidence of freedom from infection. However, animals from over 50% of outbreaks studied in Great Britain (1) carried *S. equi* for more than 6 weeks mainly in the guttural pouches. These carriers were apparently healthy and were negative for *S. equi* when serial nasopharyngeal swabs were collected and cultured over weeks or even months. However, these carriers were regularly identified by culture of lavage samples collected endoscopically from the guttural pouch(2). The average period that *S. equi* was recovered from 23 long term carriers was 9.2 months. The longest period of recovery of *S. equi* from first isolation was 42 months in one case, and another horse continued shedding for 39 months up to post mortem examination. In 21 of 28 (75%) asymptomatic carriers, the site of carriage was a one or both guttural pouch and infection was usually associated with chronic empyema and in some cases chondroid formation (2).

Failure of conventional culture techniques of nasopharyngeal swabs is the probable explanation for carriers escaping detection and subsequently introducing strangles to herds of susceptible animals(2). Highly sensitive *S. equi* detection is now feasible with the use of polymerase chain reaction (PCR) to detect living or dead organisms draining into the pharynx from the guttural pouches as an aid to presumptively identifying carriers. A PCR assay based on the M-like protein gene in conjunction with bacterial culture of nasopharyngeal swabs presumably detected over 90% of guttural pouch carriers whose culture alone would have detected less than 60%. Unfortunately this

improved rate of detection still depends on the results on a series of 3 nasopharyngeal swabs(2). By use of repeated swabbing after outbreaks or as part of a quarantine program for incoming horses, long term carriers of *S. equi* can be identified. Through their isolation, they can be prevented from spreading disease to susceptible contacts.

This system of identification of carriers by culture and/or PCR, while not 100% reliable, is more reliable than the usual recommendation for the control of strangles. These have been to isolate or quarantine new arrivals for two to three weeks and look for evidence of strangles-like upper respiratory tract infection. Owners may not be prepared to take this more aggressive route to control strangles due to the financial costs(3).

**Control of Strangles in the Environment**

Clinically affected animals or identified carriers should be isolated immediately in a quarantine area and their water buckets or feed containers cleaned and disinfected daily. Bedding can be burned or alternatively composted under a plastic sheet (to prevent spread by flies). Any areas contaminated by infected horses should be scrubbed with water and detergent, and then disinfected by steam cleaning and/or application of the effective disinfectants. Fly control is required to prevent spread during an outbreak(3).

Under optimal conditions, the bacteria can survive probably six to eight weeks in the environment. Jorm (4) has shown that *S. equi* survived for 63 days on wood at 2° C and for 48 days on glass or wood at 20 ° C. The organism is readily killed by heat (60 ° C) or disinfectants. Contaminated pasture areas should be rested for four weeks, since the organism will be killed by the natural antibacterial effects of drying and of ultraviolet light. Quarantine area staff should change their coveralls and boots before leaving the quarantine area, and should wash their arms and hands carefully with chlorhexidine soap (3).

**Control of Strangles in Non-affected Horses** (5, 6)

Most horses develop a solid immunity to re-infection during recovery from strangles. This immunity does not persist in about 30% of animals, which become susceptible again over the following months. Acquired resistance appears to be mediated by a combination of SeM-specific opsonophagocytic antibodies in serum and by locally produced SeM-specific antibodies in the nasopharynx. Local immunity may block infection at the mucosal level because resistant animals re-challenged with large numbers of *S. equi* do not make anamnestic responses, suggesting that clearance occurs before entry into the mucosal induction sites. The involvement of SeM at an early stage of infection is also indicated by experiments demonstrating that ponies inoculated intranasally with large numbers of a SeM-negative mutant do not become infected. Opsonophagocytic antibody in serum is associated mainly with the IgGb subisotype, although serum SeM-specific IgA and IgGa also have opsonic activity for *S. equi*(5).

Opsonic antibodies are very effective in clearance of *S. equi* from blood, and so metastases to remote body sites are rare. However, opsonic antibody by itself is not effective in preventing abscess development in lymph nodes. Antibody levels begin to rise a week or two after exposure to *S. equi* and peak 3–4 weeks later. Mucosal IgGb appears before IgA but decline in 8–10 weeks. SeM-specific IgA in mucosal secretions peaks about 2 weeks after IgGb but persists much longer.

Active immunization of horses against strangles was first attempted nearly a century ago, but results were generally disappointing. Bacterin-type vaccines were introduced in Australia in the early 1940s and subsequently in the United States in the 1960s. These vaccines were produced by moderate heat inactivation of logarithmic phase cultures, which should have resulted in preservation of all protective antigens available on cultured organisms. The frequency of adverse reactions, including inflammation and abscess formation at the injection site and muscle soreness, were the impetus for subsequent development of extract vaccines lacking irritant cell wall constituents such as peptidoglycan. Protein-rich vaccines, produced either by hot acid treatment (Strepvac ™) or mutanolysin, a muramidase that releases proteins from the cell wall (Strepguard ™), were marketed in the United States in the 1970s and 1980s. Extract vaccines were better tolerated than bacterins. Studies on their efficacy suggested a reduction in the clinical attack rate of about 50% in vaccinates. Commercial vaccines have been shown to stimulate strong serum opsonic activity but not mucosal SeM-specific IgGb or IgA responses. Because both SeM-specific serum and mucosal immunoglobulin responses are correlated with resistance to reinfection, the lack of a mucosal response to parenterally infected strangles vaccines may explain their low level of efficacy (5).

On the basis of this hypothesis a live, attenuated, nonencapsulated mutant of *S. equi* (707-29) was developed that stimulated mucosal and systemic antibody responses similar to those produced during convalescence. Ponies vaccinated with the nonencapsulated mutant (709-27) were resistant to intranasal challenge with S. equi and made strong systemic and mucosal antibody responses to SeM. This mutant became the progenitor of Pinnacle IN ™ intranasal strangles vaccine, marketed in the United States since 1998. The vaccine is administered intranasally in two doses at an interval of 1–2 weeks. Because the vaccine is a living mutant of *S. equi*, inadvertent contamination of injection sites remote from the nose has resulted in formation of deep abscesses. Unique complication arose from live bacteria in the vaccine getting on the hands on the person administering the vaccinations and contaminated the needles and syringes used to administer the intramuscular doses. Transient nasal discharge, submandibular or pharyngeal lymphadenopathy, limb edema, and bastard strangles abscesses have also been reported after administration of vaccine. The majority of adverse systemic responses were submandibular or pharyngeal lymphadenopathies with or without abscessation. Purpura-like disease was severe in 3 of 13 cases reported. Most of these cases were on farms where strangles was endemic; thus it is unclear whether the vaccine or a wild strain of *S. equi* was the inciting antigen. Nevertheless, it is clear that intranasal vaccination with avirulent *S. equi* carries a slight risk of purpura in an animal with a high level of *S. equi* antibody at the time of vaccination. Three cases of bastard strangles occurred in horses vaccinated during strangles outbreaks, but it is unclear whether all 3 cases were caused by the vaccine. The intranasal vaccine has been shown to be safe in mares at all stages of pregnancy at 10 times the normal dose. Although there are very few reports of adverse effects in foals, their inability to make mucosal IgA responses until a month after birth combined with potential interference from maternal immunoglobulin suggests there is little merit in vaccinating foals under 4 months of age (5).

Rapid progress is being made in development of alternative methods of intranasal immunization of horses with SeM and other antigens of *S. equi*. Of the genus *Streptococcus*, *Streptococcus zooepidemicus* is the species of bacteria, which has evolved to coexist with the horse. It is the most frequently cultured organism from a variety of equine infections. That *S. equi* is a more virulent clone of an ancestral *Streptococcus zooepidemicus* has become apparent from the over 97% commonality of DNA that *Streptococcus zooepidemicus* and *S. equi* share. The 2 to 3% of the genetic material that is not shared with *Streptococcus zooepidemicus* and is unique to *S. equi* is currently

being investigated (6). Timoney proposes that it is this unique portion of the bacterium's genome, which will code for immunogenic proteins, which are specifically protective against strangles in which when added to existing vaccines suspensions may significantly improve efficacy. His studies suggest that an antibody response on two different levels is needed in order for a vaccinated horse to be protected. Antibodies produced at the surface of the mucous membranes where organisms first invade are necessary because the presence of antibodies there will bind and neutralize the infectious organisms preventing their binding to the horse's tissue. Second, antibodies must additionally be present in the tissues, which lie between the pharyngeal lining and the deeper lymph nodes where they perform the same function, namely to bind and neutralize bacteria so that metastasis of infection to deeper lymph nodes is prevented (6).

A nasal vaccine that can successfully prompt both of these immune responses would conceivably eradicate strangles from a vaccinated closed herd. The surface M-protein (SeM) that has formed the basis of the parentally administered subunit vaccines is highly immunogenic and will probably remain a primary component of future vaccine preparations. Timoney has identified two additional proteins on the bacterium's surface, SePE-H and SePE-I, which may appear to be key players in eliciting an immune response from the horse. Timoney first recognized all three of these immunogenic surface proteins, SeM, SePE-H, and SePE-I. The genes, which code for the three unique wall proteins of *S. equi* are part of the 2 to 3% difference in genetic material that exists between *S. equi* and *Streptococcus zooepidemicus*. Timoney stipulates that there are probably a total of 20 to 30 proteins included in this sequence, however and that additional proteins are likely present which will turn out to be important in the horse's immunologic response to strangles. Such bacterial proteins, which elicit a significant immune response, could be targeted for investigation into their possible suitability for inclusion in future vaccines. Timoney postulates, for example, that SePE-I may be one of the bacterial cell wall proteins, which stimulates an immune response in the infected horse. Antibodies made against SePE-I may be what confer the resistance to future infections seen in naturally infected organisms. If so than SePE-I could be an important addition to protein M (SeM) in future vaccines (6).

Timoney and his group were able to inactivate capsule synthesis in an avirulent, non-encapsulated strain (Pinnacle) by integrating a recombinant pTW100 plasmid into the *has*A sequence and then screened for additional mutants for which the plasmid sequence had excised imprecisely, giving rise to antibiotic sensitive and capsule negative mutants. Use of this *has* deletion mutant in place of the parent Pinnacle strain currently used in the field may permit rapid genotypic recognition of the vaccine strain by the difference in size of amplicon produced using the *has* 261 and *hap* 158R3132 primers. The large deletion of the *has* operon removes any possibility of simple reversion to a potentially more virulent encapsulated form(7).

## Treatment

**Treatment of horses with strangles depends on the stage of the disease in the individual.**

*Horses Exposed to Strangles*: Antimicrobial therapy at that time of exposure may prevent the "seeding" of the pharyngeal lymph nodes by *S. equi*. Ideally, a horse that is exposed to other horses with strangles could be treated with penicillin until the affected horses are isolated and no longer serve as sources of infection for the susceptible horse. Antibiotic therapy can prevent an exposed horse or foal from contracting strangles during the period of therapy. If the animal is still exposed when the antibiotics are discontinued, infections may then develop.

*Horses with Early Clinical Signs*: Often, the first clinical signs of strangles are fever, anorexia, depression, and purulent nasal discharge. If these signs are suggestive of *S. equi* efforts should be directed at isolation of the affected horse. Further development of clinical signs of strangles can be arrested at this time with the appropriate antimicrobial treatment. However there is a high probability of relapse following cessation of therapy if the horse remains exposed to infected horses. Protective immune responses are poor in antibiotic treated horses and thus these horses are susceptible to *S. equi* infection upon future exposure. Immediate treatment of horses that show early clinical signs should be an effective way of controlling strangles outbreaks in racing stables or riding barns though the disadvantages of treatment just discussed should be weighed.

*Horses with Lymph Node Abscessation*: The second category of therapeutic consideration is treating strangles-affected horses that have already developed lymph node abscessation. Therapy should be directed toward enhancing maturation and drainage of the abscesses. Recommended procedures include: isolation of the sick horse, local application of hot packs and poultices to the abscess, and possible lancing of the ventral surface of the abscess. After an abscess is draining, it should be flushed regularly with 3% to 5 % povidone-iodine. Most authors agree that using antibiotics in not beneficial after an abscess has formed. Parenteral antibiotics given after abscess formation tend to prolong rather than arrest the disease.

Some horses with advanced signs of strangles require antimicrobial treatment. If a horse shows signs of prolonged fever, anorexia, depression, lethargy or dyspnea resulting from severe swelling of retropharyngeal lymph nodes, systemic treatment with penicillin is recommended. Rarely, affected horses may require intensive supportive therapy, including intravenous fluids, feeding by nasogastric tube, and tracheostomy. Any animal requiring a tracheostomy should be given systemic antimicrobial drugs to prevent secondary bacterial infections of the lower respiratory tract.

*Horses with Complications:* Horses that develop complications from strangles must receive therapy directed at treatment of the specific problems. Antimicrobial therapy with penicillin is appropriate for metastatic abscessation to either the thoracic or peritoneal cavity. Horses with purpura hemorrhagica require systemic antibiotics (penicillin) and corticosteroids. Guttural pouch empyema must be treated locally either by flushing of the pouches through the pharyngeal openings or by surgical incision into the pouches through Viborg's triangle.

## Conclusions

Despite significant advances in veterinary medicine, strangles persists as a debilitating and much feared equine disease. Understanding of the disease has been considerably advanced by the application of molecular diagnostics to epidemiological studies, which have led to the important recognition of an asymptomatic carrier population. Labor-intensive nature of carrier detection and treatment indicates that development of an effective strangles vaccine represents the best prospect for disease control. To date only the M-like protein SeM has been extensively studied as a protective antigen, but has proven unsuitable. It remains to be seen whether other potentially protective antigens such as *hap* will be more efficacious either alone or as part of a multi-virulent vaccine in combination with SeM.

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# THE ENDOSCOPIC DIAGNOSIS OF ABNORMALITIES OF THE SOFT PALATE, PHARYNX AND LARYNX

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## Introduction

Endoscopy is possibly the most important diagnostic aid used in the assessment of clinical health and disease of the upper respiratory tract. Elsewhere in these proceedings the endoscopy techniques used to inspect the nasal chambers, the paranasal sinuses and the auditory tube diverticula are presented. This presentation will summarise the more common abnormal endoscopic features of the equine nasopharynx, soft palate and larynx.

## What can an endoscopic examination of the region be expected to reveal?

- Evidence of structural abnormality
- Evidence of dysfunction during quiet breathing
- Evidence of previous surgery

## 1. Structural abnormalities include:

### Nasopharynx:

- **Congenital defects of the palate** These are almost invariably confined to the soft palate and may consist of simple midline clefts, unilateral hypoplasia, complete aplasia and even pseudo-uvular deformities. Frequently there is concurrent entrapment of the epiglottis. Horses with simple midline clefts are likely to show signs of dysphagia from the earliest age but some with unilateral palatal hypoplasia may escape diagnosis until middle age
- **Pharyngeal lymphoid hyperplasia** - although this a normal feature of all young horses note should be made if this entity is particularly marked
- **Acquired palatal defects** "Over-enthusiastic" palate resection surgery may result in chronic palatal instability with persistent DDSP. The end-point of the resection can usually be seen and a laryngotomy cicatrix should be palpable. Attention should be drawn to such findings and their significance in the report to the potential purchaser

- **Palatal ulceration** An ulcer at the free border of the soft palate implies contact with thickened tissue at the ventral aspect of the epiglottis. Such ulceration is not uncommon concurrent with lymphoid hyperplasia in young horses but should be viewed with suspicion in the older animal as it may be evidence of intermittent epiglottal entrapment or sub-epiglottic cyst
- **Permanent dorsal displacement of the soft palate**, i.e. the epiglottis does not come into view at any stage of the examination even after repeated stimulation to swallow (c.f. intermittent DDSP - see below). This is a serious finding as it implies an abnormality of the palate, epiglottis or of the structures at the base of the tongue it would constitute grounds for advising against purchase. Radiography to investigate the structures on the ventral surface of the soft palate or endoscopy/ per os under anaesthesia is required for a specific diagnosis
- **Pharyngeal cysts** usually comprise discrete, thin-walled lesions attached to the pharyngeal wall, most commonly at the margins of soft palate
- **Intra-palatal cysts** are seen as swellings within the soft palate and make cause prolonged periods of DDSP. They carry a poor prospect of successful surgical removal
- **Distortion of the pharynx through external compression** by lymphadenopathies, distension of the guttural pouches or neoplasia. Swellings are likely to be palpable from the outside of the throat
- **Neoplasia** A tumour could be expected to consist of an ulcerated soft tissue mass - lymphosarcoma is the neoplasm most commonly found at this site

## Larynx

- **Epiglottal hypoplasia**. Hypoplasia may take one of two forms: gross foreshortening which is rare in Thoroughbreds and partbreds, and apparent lack of rigidity which is highly subjective and is a frequent feature of the larynx of foals and yearlings, i.e. it is a phenomenon associated with immaturity
- **Epiglottal entrapment (see below)**
- **Sub-epiglottic cysts (see below)**
- **Fourth branchial arch defects (4-BAD) (see below)**
- **Arytenoid chondropathy** Suppuration within the cartilage matrix of the arytenoid cartilages produces endoscopic changes which vary from reduced motility through ankylosis of the crico-arytenoid articulation to gross axial displacement with large granulomatous projections from the medial face of the infected cartilage and contact lesions on the contralateral arytenoid. In the early stages there is the possibility of confusion with RLN. In some cases the proliferation is pedunculated and a diathermy loop passed via the biopsy channel of the endoscope can be used to resect the mass. Endoscopy can be used to monitor the progress of arytenoid chondropathy following both medical and surgical treatments.
- **Neoplasia** The most frequent proliferative lesions of the larynx consist of the chondropathy mentioned above and these lesions are often mistaken for neoplasia. True neoplasia of the equine larynx is very rare

**2. Functional abnormalities** which may be detected at rest or immediately after exercise include:

## Soft palate

- **Intermittent displacement of the free border of the soft palate** above the epiglottis (DDSP) may be seen at rest, particularly after swallowing or tracheoscopy, but it should not be regarded as indicative of whether or not dynamic DDSP will occur during periods of exertion. The use of a nose twitch quite commonly provokes prolonged periods of DDSP in normal horses. The author's policy in such cases is to report to the client: "During the endoscopic examination periods of dorsal displacement of the soft palate were noted. There is no evidence that this finding constitutes evidence of a horse prone to soft palate displacement (DDSP) at exercise"
- **Permanent dorsal displacement** of the soft palate is a far more significant finding – see above.

## Pharynx

- A diagnosis of **pharyngeal paralysis** as a cause of nasal regurgitation of ingesta is straightforward - the pharyngeal walls lack tone, the palatal arch is persistently dorsally displaced and food debris and saliva are present in the lumen. Inspection of the ATDs is indicated as mycosis is a likely explanation for this glosso-pharyngeal neuropathy. In most cases the disorder is in the strictest sense pharyngeal hemiplegia because the paralysis is unilateral. This is confirmed by observing the ostia of the ATDs and noting whether one or both fails to dilate after swallowing

## Larynx

- A system of grading laryngeal function is described in the discussion of recurrent laryngeal neuropathy (RLN) together with comments on the significance of the findings.

**3. Evidence of previous airway surgery** can be reliably identified by endoscopy in most instances even when palpable evidence of an external cicatrix is equivocal:

- **Soft palate resections** (staphylectomy) usually leave the margin of the palatal arch somewhat more rounded than the normal V-shaped conformation; a small tell-tale irregularity at each end of the excision can usually be seen
- **Ventriculectomy with or without cordectomy** ("Hobday" operation) leaves one or both ventricles replaced by scar tissue and the sharp edge of the vocal cord will be absent if cordectomy was included in the technique
- **Prosthetic laryngoplasty** ("Tie-back") is intended to leave the left arytenoid cartilage in a state of fixed abduction. The degree of abduction should be noted. Even when the surgery has failed the left arytenoid cartilage will be immobile and it is common practice to perform ventriculectomy surgery concurrent with a "tie-back". The lumen of the trachea should be checked for the presence of flecks of ingesta which may indicate iatrogenic dysphagia

The previous surgery which is more difficult to detect by endoscopy comprises:

- **Epiglottal entrapment relief** should leave no distortion of the epiglottal cartilage in the long term, but occasionally a small mucosal granuloma is left at each lateral margin when resection via laryngoplasty is performed. Relief by hooked bistoury per os or per nasum or by laser surgery is generally undetectable. In the days immediately after any of these forms of surgery, the epiglottal cartilage tends to appear inflamed and soft
- **Sub-epiglottic cyst removal** is generally performed via a ventral laryngotomy and the only likely residual internal evidence of this surgery may be a slight thickening of the sub-epiglottic soft tissues. Lateral granulomas marking the lateral limit of the mucosal incisions at the ventral edge of the epiglottis adjacent to the palatal pillars may also be seen
- **Nerve-muscle pedicle graft surgery to relieve RLN** is not yet widely performed and resting endoscopy is a useless method to assess whether the operation has been effective. The omo-hyoideus, an accessory muscle of respiration is used to form the grafts. This muscle is not normally in a contracted state at rest and treadmill endoscopy is required if the function of the grafts is to be checked. In the resting state the larynx is likely to show motility indicative of Grade 5 RLN

## What may an endoscopic examination at rest or immediately after exercise fail to reveal?

The major limitation of endoscopy performed at rest is that functional disorders only present during, or precipitated by, extreme exertion will not be identified. Fortunately the majority of the conditions causing dynamic collapse of the pharyngeal or laryngeal airways will lead to an audible inspiratory noise during an exercise test. This reinforces the view that **untoward respiratory sounds heard during an exercise test must never be ignored even when endoscopy at rest has failed to reveal the cause**. Examples of these conditions include:

- Dorsal displacement of the soft palate
- Dynamic collapse of the pharyngeal walls
- Dynamic collapse of the left arytenoid cartilage and vocal fold
- Intermittent epiglottal entrapment
- Epiglottal retroversion
- Dynamic medial collapse of the ary-epiglottic folds

# Miscellaneous conditions of the larynx : Epiglottal Entrapment, Sub-epiglottal Cysts and Fourth Branchial Arch Defects

## Epiglottal entrapment (EE)

### Aetiopathogenesis

In this condition the cartilage of the epiglottis becomes enveloped by a fold of glosso-epiglottic mucosa which arises between the epiglottis itself and the base of the tongue and extends laterally as the aryepiglottic folds. The aetiology of EE is usually not known as the condition can be reproduced on most equine laryngeal post-mortem specimens and it is not clear why some horses develop the clinical condition and others do not. The aetiology of EE is more straightforward when the epiglottis is congenitally hypoplastic or where it is associated with a sub-epiglottic cyst. These possibilities should be checked by endoscopy and/or radiography before surgical correction is attempted.

### Clinical signs

The signs associated with EE are highly variable and include exercise intolerance with inspiratory and/or expiratory noises at exercise, intermittent gurgling from secondary DDSP and coughing after eating. The noises which are associated with EE are usually vibrant and are more readily provoked than those of dynamic DDSP. In addition, it should be noted that EE may be a chance endoscopic finding in horses which are asymptomatic.

Reports of the prevalence of EE in endoscopic surveys of athletic horses at rest suggest an incidence in the range of 0.75% to 3.3%. However in the author's video-endoscopic survey of an unselected population of 3497 yearlings only 2 cases were identified - in the same survey there were 5 sub-epiglottal cysts and 7 yearlings afflicted with fourth branchial arch defects. The implication is that EE is an acquired disorder rather than a congenital defect.

The relationship between EE and dynamic DDSP is confusing. Treadmill studies have confirmed what has been suspected for some time inasmuch as EE is likely to provoke secondary DDSP during exercise. However this does not explain the clinical observation that horses which have been afflicted with EE and have been subjected to surgical correction may become susceptible to DDSP later, and vice versa.

### Diagnosis of epiglottal entrapment

Definitive diagnosis of EE is achieved by endoscopy: the normal epiglottis has a sharply defined wrinkled cartilage outline and the superficial blood vessels on the dorsal surface are clearly visible. In EE the epiglottis loses its wrinkled cartilage border and the superficial blood vessels are obscured by the entrapping mucosa. However, a structure with the overall shape of the epiglottis remains present. In a proportion of cases the mucosa overlying the epiglottis is ulcerated.

EE may be intermittent and it is important, as a routine part of endoscopic procedure, to stimulate a series of deglutition sequences in an attempt to identify the condition.

Occasionally the free border of the soft palate is found to be dorsally displaced throughout the endoscopic examination of a horse showing abnormal respiratory sounds at exercise. Epiglottal entrapment and sub-epiglottic cyst are the two most common explanations for persistent DDSP in the horse at rest. Attempts should be made to stimulate swallowing while the endoscope is in place in the hope that the epiglottis can be brought into view because, in the face of persistent DDSP, it should be obvious that the epiglottis cannot be seen at all. Should this manoeuvre prove to be ineffective a lateral radiograph of the pharynx is indicated to determine whether or not entrapment is present. On this projection the epiglottis should measure at least 7.0 cm from tip to hyoid articulation; an epiglottis with a length less than 5.5 cm is indicative of hypoplasia and surgery is contra-indicated.

In very rare cases EE may be present only during vigorous exercise so that high speed treadmill endoscopy provides the best opportunity to establish a diagnosis of this form of intermittent entrapment.

**Treatment of epiglottal entrapment**

The treatment options for epiglottal entrapment are:

- resection of the entrapping mucous membrane via ventral laryngofissure
- axial division of the entrapping mucosa per os
- axial division of the entrapping mucosa per nasum
- transendoscopic Nd: YAG laser resection per nasum

Methods (1) and (2) require general anaesthesia but both (c) and (d) can be performed on the standing horse with the obvious advantage of minimal disruption of training and racing schedules.

Methods (2) and (3) utilise a hooked bistoury to make a longitudinal cut in the loose mucous membrane overlying the epiglottal cartilage. The most important practical limitation of these methods is the stability of the entrapment. Whenever the entrapment is insecure or intermittent it may not be possible to make a satisfactory section of the redundant mucosa because the forward pull of the bistoury may displace the entrapment from the apex of the epiglottis. An inadequate incision is likely to be followed by recurrence within a short time. However, if the entrapment is secure the hooked bistoury techniques are applicable. Obviously a sharp instrument introduced into the pharynx of a fractious horse in method (3) has the potential for causing serious injury and there is a very real danger of causing damage to the soft palate. Thus, it is essential that the patient is thoroughly sedated and the pharynx is desensitised by the infusion of local anaesthetic solution. The procedure of axial section of the EE is performed under endoscopic surveillance - the endoscope is passed through one nostril while the bistoury is introduced through the other. The aryepiglottic mucosa is quite tough and care is required to prevent a disastrous palatal injury when the hook suddenly cuts free from the entrapment. Method (2) is safer, albeit with the risk of anaesthesia, and represents the technique of choice in those clinics which do not possess a laser facility .

Surgical relief via laryngotomy uses a conventional approach through the crico-thyroid membrane. For the resection itself the endotracheal tube must be withdrawn so that the entrapped epiglottis can be reverted towards the incision. It is sensible to place a stay suture through the tissue to be excised at an early point in the procedure. An elongated oval section of mucosa 6 cm x 1 cm is removed and it is preferable to commence with incisions from lateral to medial through the doubled layer of mucosa. Care is taken not to traumatise or cut into the cartilage of the epiglottis. Haemorrhage is usually minimal and surgical closure of the defect in the sub-epiglottic mucosa is unnecessary.

Regardless of the technique used, endoscopy performed in the days after surgery should confirm that the entrapment has been successfully relieved although the epiglottis itself may appear swollen and flaccid. Hence anti-inflammatory medication should be provided routinely after treatment of EE.

### Prognosis

All of the methods of treatment mentioned above yield a high rate of uncomplicated resolution of EE. The possibility of recurrence of entrapment is greatest with the axial section techniques but even this is uncommon. Iatrogenic trauma to the epiglottal cartilage can provoke granulomas and other distortions which may compromise the relationship between the epiglottis and the soft palate. Chronic coughing associated with low grade dysphagia is a rare but recognised complication of resection of the glosso-epiglottal mucosa.

## Sub-epiglottal Cysts

### Aetiopathogenesis

Developmental cysts are occasionally recognised in the pharynx of horses and represent causes of dyspnoea and/or dysphagia. The most frequently encountered of these lesions is the sub-epiglottic cyst which is thought to be derived from the embryological remnants of the thyro-glossal duct, a structure which runs from the level of the epiglottis to the anterior mediastinum. However, it is strange that comparable cysts do not arise elsewhere in the course of this duct. It is believed that sub-epiglottic cysts are present from birth although they may not be discovered until the horse is mature and commences training. An overall incidence of 1.4 per 1000 Thoroughbreds born was indicated in the survey outlined above.

The cysts themselves consist of smooth-walled, sometimes multilobular, structures filled with straw-coloured slightly tenacious fluid. They arise from within the loose glosso-epiglottic mucosa lying between the base of the tongue and the epiglottis. It is uncommon for a sub-epiglottic cyst to be found which is not located within an epiglottal entrapment.

### Clinical signs

The age and manner by which the cysts cause clinical signs is dependant on their size. Large cysts can be a cause of dysphagia and respiratory obstruction in foals. Such foals may present within a few days of birth with reflux of milk from the nares and will require an endoscopic examination for differentiation from foals with palatal clefts. Often aspiration of fluids into the trachea will lead on to inhalation pneumonia and in addition to surgical relief, vigorous antibiotic medication will be required.

Older animals with cysts may present with a variety of respiratory signs including nasal discharge from dysphagia and abnormal respiratory sounds at exercise. Horses with small sub-epiglottic cysts may present with a history of choking up under exertion and will require differentiation from DDSP.

### Diagnosis

The diagnosis is easily established by endoscopy provided that the cystic lesions are available to be seen. In some horses with this condition persistent DDSP is present, in which case it will be necessary to resort to other diagnostic procedures such as plain and contrast radiography. The cysts may be intermittently obscured by the palatal arch when the cartilage of the epiglottis lies above the palate and the cyst is trapped below. Patient inspection with repeated stimulation of deglutition should always be part of the endoscopic routine for horses with suspected pharyngeal lesions such as entrapment or cysts. Endoscopy per os with the patient anaesthetised is a justifiable technique if endoscopy per nasum and radiography have failed to provide a definitive explanation for persistent DDSP, .

### Treatment

The objective of treatment is to ablate the cyst by sharp excision which can be achieved per os using an ecraseur provided that the mouth of the horse is large, and the hand of the surgeon is small, for accurate placement of the ecraseur loop around the cyst without risk of injury to adjacent structures, most notably the epiglottis itself. Resection by conventional ventral laryngofissure is generally simpler and safer. The technique is similar to that used for the treatment of epiglottal entrapment and, again, once the tip of the epiglottis has been retroverted towards the laryngotomy incision, stay sutures to stabilise the tissues before resection are a useful practical precaution. Haemorrhage is usually slight and there is no need for closure of the mucosal defect following excision.

Nd : YAG laser destruction of the cysts can also be used when the facility is available.

### Prognosis

The cystic lesions can often be excised intact and even if they should be ruptured during removal a favourable prognosis can be given. Unlike thyro-glossal cysts in man, recurrence is most unlikely to occur after excision in the horse.

## Fourth branchial arch defects (4-BAD)

Surveys of structural and functional abnormalities of the upper respiratory tract of the horse have previously been based upon selected populations for example those submitted to public sales or in training where unsaleable or untrainable individuals will have been excluded earlier. In addition, no survey of dynamic dysfunctional conditions, occurring only under exercise conditions, has been attempted to date. Thus, it is generally held that dorsal displacement of the soft palate (DDSP) and recurrent laryngeal neuropathy (RLN) are the two most frequently encountered disorders of the region. The prevalence of non-RLN URT disorders found during the video-endoscopic study of 3497 yearlings was as follows:

| | | |
|---|---|---|
| Fourth branchial arch defects (4-BAD) | 7 cases | 0.20% |
| Sub-epiglottal cyst (SEC) | 5 cases | 0.14% |
| Epiglottal entrapment (EE) | 2 cases | 0.06% |
| Right laryngeal malfunction | 5 cases | 0.14% |

It has been shown that in the development of the larynx in the early human embryo the extrinsic structures are derived from the fourth branchial arch and the intrinsic structures from the sixth arch. 4-BAD is a syndrome of irreparable congenital defects resulting from a failure of development of some or all of the derivatives of the fourth branchial arch. The structures involved are the wings of the thyroid cartilage, the crico-thyroid articulation, the crico-thyroideus muscles and the crico-pharyngeal sphincter muscles. Any permutation of aplasia or hypoplasia of these structures may arise uni- or bilaterally.

The condition has been identified in other breeds such as the Hanovarian, warmbloods, Welsh Section A ponies and the Haflinger. As often as not it has been reported under titles such as rostral displacement of the palato-pharyngeal arch (RDPA) or crico-pharyngeal-laryngeal dysplasia.

The author has reviewed the findings in 60 4-BAD-afflicted Thoroughbred horses and the results are summarised here. The presenting signs of horses with 4-BAD are variable and reflect the severity of the absence of the structures involved. However, in order of frequency, the signs are abnormal respiratory sounds at exercise (50 out of the 60 horses), belching (13), nasal discharge (10), coughing (10) and recurrent colic (5). The involuntary aerophagia and eructation sometimes may be confused with the noises produced by 'wind-suckers'.

There is no current evidence that the syndrome is genetically transmitted. A complete evaluation of the extent of 4-BAD can only be made at exploratory surgery or autopsy but the combined findings of palpation, endoscopy and radiography are generally sufficient to justify a diagnosis. When the cartilage components are defective, an unusually wide gap can be palpated between the caudal margin of the thyroid and the rostral edge of the cricoid whereas in the normal larynx the two structures overlap. The two endoscopic features to alert the clinician to the possibility of 4-BAD are RDPA (33 cases) where the caudal pillars of the soft palate form a cowl which partly obscures the corniculate processes dorsally and defective arytenoid motility (45 cases). **4-BAD is the commonest explanation for apparent right-sided RLN** and there is be a marked over-representation of right-sided cases. In the 60 cases 15 were bilateral, 39 were right unilateral and in 6 the defects were confined to the left side. In one instance 4-BAD was only detected as dynamic RDPA during treadmill exercise. RDPA itself is simply an endoscopic symptom of a major underlying laryngeal disorder and should not be regarded as a disease in its own right. It arises when the upper oesophageal sphincter muscles are not present to provide an anchor for the palatal arch caudal to the apices of the corniculate processes of the arytenoid cartilages. When the crico- and thyro-pharyngeus muscles are absent there is a failure to close the upper oesophagus so that lateral radiographs will reveal a continuous column of air extending from the pharynx into the oesophagus. The RDPA is seen as a 'dew drop' intruding into this air column from the dorsal wall. Repeated

aerophagia leaves those animals without an upper oesophageal sphincter susceptible to episodes of colic which may be life threatening. Three horses have died or been destroyed through tympanitic and one other has required surgical decompression.

The performance records of 51 of the horses have been traced: 22 were un-named; 29 were named but unraced; 11 raced but were unplaced; 4 were placed and 7 won albeit all in minor competitions. Thus, horses afflicted with 4-BAD are generally ineffective athletes.

There are currently no surgical techniques available to replace or reconstruct the absent laryngeal structures. Treadmill studies have shown that the dynamic collapse of the unstable structures of the larynx is not straightforward and axial deviation of the right ary-epiglottal fold (ADAEF) is the most common cause of airway obstruction. Thus, resection of the right ary-epiglottal fold offers a symptomatic remedy for a proportion of afflicted animals.

Five cases of right laryngeal malfunction, other than the 7 cases of 4-BAD, were identified in the stud survey of yearlings. Three of these were later subjected to surgery with a view to prosthetic laryngoplasty but were found to be inoperable by virtue of hypoplasia of the muscular process of the right arytenoid cartilage. Given that the arytenoid cartilages develop from the sixth branchial arch dare we contemplate adding a 6-BAD syndrome to the clinical vocabulary!?

## Conclusions

"Endoscopists are inclined to examine the hole in the patient and not the patient as a whole"

Endoscopic assessments must never be performed in isolation, least of all during pre-purchase examinations, and the findings must be placed in the context of the other observations which have been made during the external physical inspection. There is frequently a perception by horse owners that endoscopy represents an infallible method by which to diagnose all respiratory disease, but this is far from correct.

# HEAVES AND INFLAMMATORY AIRWAY DISEASE: THE DIFFERENCES

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## DEFINITIONS

**Large airways:** The nasal passages, throat, trachea, and major bronchi (~ 5 generations)

**Small Airways:** Small bronchi (~ next 15 generations) and bronchioles

**Heaves:** recurrent airway obstruction that is reversed by a change in environment or use of bronchodilators.

**Inflammatory Airway Disease (IAD)**: A syndrome of non-septic inflammation of the small and large airways particularly in young horses

**Reactive airway syndrome:** any horse with hyper-reactive airways (could be a horse with heaves, IAD, or bronchopneumonia)

**Pneumonia:** a pulmonary disease characterized by replacement of air in alveoli and alveolar ducts with an inflammatory exudate, and/or by inflammatory cell infiltration of the alveolar walls and interstitial spaces.

## Terms that are "valid" but probably better avoided in equine medicine

**COPD:** chronic obstructive pulmonary disease

**Small Airway Disease (SAD):** any non-infectious disease of the small airways

**Small Airway Inflammatory Disease (SAID):** inflammation of the small bronchi and bronchioles

**Chronic bronchitis / bronchiolitis:** inflammation of the small airways as confirmed by biopsy.

## HEAVES

### Etiology & Pathophysiology

- Equine inflammatory airway disease can have various causes and is observed in various degrees of severity. It is inappropriate to use one term such as COPD for all these syndromes.

- The term heaves or RAO should be used for the mature horse with airway obstruction that is reversed by a change in environment or use of bronchodilators.

- New information in human and equine medicine has revealed major differences between human COPD and equine heaves so that at present it is no longer appropriate to use the term COPD in equine medicine.

- The relationship between IAD (inflammatory airway disease) in young horses and heaves is unknown. The former may not necessarily progress to the latter.

- At present there is no way to identify a young horse that may develop heaves later in life.

- In horses with heaves, morphological changes are primarily located in the small airways, but functional changes may occur throughout the tracheobronchial tree.

- During exacerbations of heaves, the major cause of airway obstruction is bronchospasm.

- In addition to bronchospasm, airway obstruction is also a result of accumulation of mucus and inflammatory exudates in the airway lumen and thickening of the airway wall.

- During clinical remission in heavey horses, residual airway inflammation, obstruction or hyper-responsiveness may persist.

- During exacerbations of heaves, the predominant inflammatory cell in the lumen of the airway is the neutrophil. Eosinophilic inflammation is not typical of heaves. The presence of neutrophils does not imply a septic process.

- Bacterial infection is not a primary component in the etiopathogenesis of heaves.

- Heaves exacerbations are caused by exposure to inhaled organic dusts, which may contain molds, endotoxins and other pro-inflammatory agents. Organic dusts may be found indoors and outdoors.

- There is accumulating evidence to support an allergic component in the etiology of heaves.

- Other factors such as inhaled endotoxin may contribute to the inflammation of heaves.

- There is evidence that genetic factors play a role in the expression of heaves as a clinical disease.

### Treatment

- Environmental control is paramount for the management of heaves.

- In the majority of horses with heaves, corticosteroids are effective in reducing inflammation and in improving airway function and clinical signs. Corticosteroids do not provide immediate relief of bronchospasm.

- During acute exacerbations of heaves, bronchodilators rapidly improve airway function and reduce the signs of respiratory distress. Bronchodilatory therapy does not alleviate the underlying inflammatory process.

- Because no relationship has been demonstrated between airway and dermal reactivity, the use of skin testing to identify culprit aeroallergens is not justified.

- There is no scientific evidence demonstrating the efficacy of hypo-sensitization in the treatment of heaves.

- There is currently no scientific evidence to support the use of serum antibody testing for the diagnosis and treatment of heaves.

## INFLAMMATORY AIRWAY DISEASE (IAD)

- A syndrome of non-septic lower airway disease particularly in young horses

- The term IAD should be used until the etiology and principal anatomic site of lower respiratory disease is better defined.

- Occurs in 22-50% of Thoroughbreds and Standardbreds racehorses
    - Prevalence?
    - 22% of 965 Standardbred racehorses
    - 27% of 300 Thoroughbred racehorses
    - 55% of 2 year olds
    - 76% of horses referred for poor performance

- Is a common cause of impaired performance and interruption of training. Performance changes may go unnoticed at rest or during light work.

- Have variable clinical signs, including cough, nasal discharge, and abnormal lung sounds

- Abnormalities identified by endoscopic examination of IAD affected horses include mucopurulent exudate in the pharynx, trachea, and bronchi.

- Horses with these clinical findings have high total nucleated cells in bronchoalveolar lavage (BAL) fluid characterized by mild to moderate airway inflammation, which may involve neutrophils, mast cells, eosinophils or lymphocytes.

- May have normal lung function at rest, but show evidence of airway hyper-reactivity on exposure to non-specific agents such as histamine

- The proposed etiologies of IAD include recurrent pulmonary stress and / or persistent viral respiratory infection.

- Although allergic pneumonits may be involved in the pathophysiology of IAD, cytological profiles of BAL from IAD affected horse differs from that of heaves wherein BAL is characterized by marked neutrophilia, lymphopenia and normal total cell counts.

## Etiology

- Horses live, eat, and work in a **dust - laden** environment. Dust is composed of molds, bacteria and their toxins, minerals, insect parts, and various chemicals.

- Dust level in the horse's breathing zone can be as high as 25mg/m3 – level that would be considered unacceptable for any human workplace

- Some horses work in urban areas with air pollutants. Air pollutants are mostly gases, like ozone, sulfur dioxide, and nitrous oxide, but particulate matter, such as heavy metals, carbon, and smoke, contribute to pollution exposure in urban horses.

- Air within the stable can be polluted with ammonia, bacterial byproducts (e.g. endotoxin) molds and other chemicals which irritate the airways.

- Although previous **viral disease** is commonly invoked as a predisposing factor in the development of IAD, little evidence exists to implicate viral disease.

- As in human asthmatics, viral respiratory disease has been shown to cause airway hyperreactivity for a period of time after infection, perhaps due to denuding of the respiratory epithelium.

- However, no known virus was associated with poor performance and respiratory disease in 68% of cases in the United Kingdom.

- Role of **bacterial infection** in IAD, particularly in young racehorses.

- A strong relationship between inflammation of the lower respiratory tract and the presence of streptococcal species as well as positive correlation between bacterial counts in TTA fluid and measures of airway inflammation was shown in one study.

- It is important to maintain a distance between association and causation, and increased numbers of bacteria may reflect impaired airway clearance rather than a causative role for the bacteria.

- The role of bacterial and viral infection in IAD remains unclear at this time.

## Pathophysiology

- Dust particles enter airways and are trapped on their inner surface.

- Dust, gases, and chemicals severely irritate the sensitive airway lining.

- The airways, which are active living issue, mount a rapid and aggressive defense... inflammation.

- The extent of this reaction appears to depend on the susceptibility of the horse.

- Mucus and cellular debris accumulate within the airways; the airway walls thicken, constrict or collapse.

- The narrow, inflamed airways become very sensitive and "twitchy" to environmental challenges, including allergens, irritants, pollutants, viruses and inert particles. Their tendency to constrict is exaggerated. The airways become "hyper-reactive".

- Although heaves has been clearly shown to be an allergen mediated disease, in which horses manifest a Th2-type cytokine response, and environmental challenge can produce a consistent exacerbation of disease, there is no such convincing evidence of an allergic response in horses with IAD.

- However, the presence of elevated numbers of mast cells in BALF of horses with poor performance and the association of BALF mastocytosis with airway hyper-reactivity, as well as immunohistochemistry studies showing more IgA-containing cells and occasionally increased numbers of IgM and IgG-containing cells in the airways of horses with IAD is suggestive of a degree of allergic response and a heightened immune response.

## Clinical signs & Diagnosis

- Coughing: the typical cough is intermittent, deep, and productive. Some horses cough in a series of spasms, seemingly unable to dislodge mucus from their airways.

- Lung sounds: usually normal, but in more advanced cases there can be crackles or wheezes heard throughout the lung fields. A re-breathing bag should be used to stimulate deeper breathing for auscultation.

- Changes in Performance: complaint that horse is more "lethargic", "less responsive", "sluggish", "slowing down", or "exercise intolerant".

- Performance suffers. Typical complaints are that "racehorses slow down at the * pole", or sport horses are "sluggish" or "unwilling to move forward in their training".

- Exercise intolerance develops over days to weeks and gets progressively worse without treatment. Racehorses drop a class or two and finish poorly. Typically, the horses race well until the 3/4-mile (6 furlongs) when they "hit a wall".

- When scoped, horses with IAD have small white to gray globs or clear to white streams of mucus visible in their windpipe. After exercise, this is more readily seen.

- BAL cytology in horses with IAD has shown, variably: neutrophilia and lymphocytosis, neutrophilia and lymphopenia, neutrophilia and mastocytosis, or eosinophilia.

- This is distinguished from RAO by the relatively low percentage of abnormal cells in IAD-whereas horses with RAO may exhibit almost entirely neutrophils in the BALF, horses with IAD seldom have greater than 10-15% neutrophils.

- The **esophageal balloon/pneumotach method** for measuring lung function
    - Tends not to detect airway obstruction even in horses with RAO in remission.
    - The pressure-flow relationship is assessed as resistance, and the volume-pressure ratio as dynamic compliance.
    - The problem with this classical approach is the insensitivity of the test for sub-clinical disease.
    - Until clinical signs are evidence (presence of nasal flaring and / or abdominal pumping), there is little to no change in resistance or dynamic compliance.
    - It is clear that this method, although the gold standard due to its direct and simple elements is not particularly useful to diagnose IAD

- **Oscillometry**
    - Method for forced expiratory maneuvers to demonstrate that considerable flow limitations existed in a horse with mild heaves in which clinical examination and lung mechanics data were normal.
    - Measures of forced expiratory flow (FEF95%) was lower in horses with IAD than in normal horses

- **Bronchoprovocation**
    - Horses with clinical signs compatible with IAD also exhibit signs of airway hyper-reactivity when they are exposed to non-specific agents such as histamine aerosol

## How does IAD affect performance?

- A practitioner with a primarily pleasure horse or show hunter clientele might report a very low incidence of IAD in younger horses, because their level of exercise is not likely to force a diagnosis through signs of exercise intolerance.
- A racetrack practitioner, on the other hand, would be far more likely to detect exercise intolerance due to lower airway disease in young horses because the level of expected athletic output is much higher, and the horses have a greater likelihood of being examined endoscopically.
- Persson was able to demonstrate that oxygen uptake and pulmonary ventilation correlated inversely with the morphological grade of small airway disease and the height of the bronchiolar epithelium-the last finding suggesting that the extent of obstruction may determine the extent of exercise impairment.
- Couetil and Nyman found in separate studies that horses with IAD undergoing a treadmill stress test demonstrated a more severe impairment of gas exchange during peak exercise than did normal horses.

- Other studies have found that horses with obvious evidence of airway inflammation do not necessarily have a history of exercise intolerance.
- This may reflect the difficulty of diagnosing low-grade respiratory impairment, and the trainer's failure to recognize poorer performance than nature intended, however, rather than the benign nature of the underlying disease.

## Treatment

Medical therapy for IAD needs to achieve two main goals

- Treatment of airway inflammation: Anti-inflammatory agents Corticosteroids are the most effective drugs to reverse inflammation. Once there is significant improvement noted in clinical signs, aerosolized (inhaled) drugs can be started for long-term control to prevent recurrences.

- Relieve airflow obstruction: bronchodilators

  These remove the stimulus for cough and bronchospasm, thus enabling the horse to breathe easier immediately.

- Long-Term Control: the most important aspects of long-term control have nothing to do with drugs. Avoidance of dust, hay, and air pollutants, and lowering the incidence of viral infections by vaccination are the most important facets to prevent IAD.

- Most of the drugs and dosages recommended are based on studies performed on horses with heaves.

- Nonsteroidal anti-inflammatory and anti-histamine drugs are ineffective for the treatment of IAD.

## Aerosol Delivery Devices

- Administration of therapeutic substances via inhalation has the advantage of delivering high concentration of the drug directly into the lungs while minimizing the systemic effect.
- Therapeutic aerosols may be produced by nebulizing a solution, actuating prepackaged aerosols in metered-dose inhalers (MDI), and inhaling the drug using dry powder inhalers (DPI).
- Several types of devices are used to improve delivery of aerosol to the horse's lungs such as facemask, nosepiece, and extension tubing (spacer or holding chamber).
- The fraction of drug deposited into the lungs averages between 0.3-7.4 % for nebulizers and between 6.1-23.3 % for MDI delivery devices (6.1 %, Aeromask; 8.2 % Equine Haler, 23.3 % 3M Equine Inhaler).
- Spacers and holding chambers are designed to alter the size distribution of particle originating from the MDI or nebulizer resulting in a reduction in upper airway deposition and an increase in the mass of drug contained in respirable particles

- A valve is usually present between the spacer and the horse's nostril, therefore precise synchronization between MDI actuation and onset of inhalation is not required.

**Aerosolized corticosteroids**

- Five different inhaled corticosteroids are available in the US:
    - beclomethasone dipropionate
    - budesonide
    - flunisolide
    - fluticasone proprionate
    - triamcinolone acetonide.

- A common test of potency for inhaled corticosteroids allows relative ranking of the compounds from least to most potent:
    - flunisolide = triamcinolone acetonide < beclomethasone dipropionate = budesonide < fluticasone propionate.

- Beclomethosone
    - Clinical trials in horses with heaves indicate that beclomethasone dipropionate at dosages ranging from 500-1500 µg twice a day (3M Equine Inhaler) to 3750 µg twice a day (Aeromask) result in significant clinical and lung function improvement as well as reduction in pulmonary inflammation.
    - Therapeutic effects are measurable within 24 hours of administration.
    - Dosages between 500-1500 µg are recommended to treat IAD using commercially available MDI-delivery devices.
    - Such dosages result in significant systemic absorption evident by adrenal suppression, however administration of a low-dose (500 µg) to horses with heaves results in similar efficacy as high-dose but with less adrenal suppression.
    - In humans, the half-life of beclomethasone metabolites after inhalation treatment varies from 1.5 to 15 hours
    - Elimination times have not been reported in the horse.

- Fluticasone
    - Fluticasone propionate has been used successfully for the treatment of heaves in horses using 2000 µg twice daily (Aeromask).
    - Improved clinical signs, decreased airway hyper-responsiveness, and reduced pulmonary inflammation are detectable within 2 weeks of therapy in horses with IAD.
    - Treatment of horses inhaled fluticasone (Aeromask + spacer) using 3000 µg twice a day results in adrenal suppression, however no adrenal suppression is detectable with 2000 µg twice a day.
    - Elimination drugs studies have not been conducted in the horse.

- Cromones
    - Sodium cromoglycate (cromolyn) has been shown to improve clinical signs and to decrease bronchial hyper-responsiveness when administered to horses with type 2 IAD (high mast cell count in BAL).
    - However, it is ineffective for the treatment of IAD type 1 and 3.

- Bronchodilators
    - Bronchodilators are indicated to relax airway smooth muscle and relieve airflow obstruction.
    - Two main classes of inhaled bronchodilators have been used in the horse:
    - ß2-agonists
    - anticholinergics
    - Bronchodilators should not be used as the only therapy for IAD because they do not suppress airway inflammation and do not reduce airway hyper-responsiveness.
    - In addition, prolonged use of ß2-agonists without corticosteroids induces receptor down regulation, which renders the drug ineffective.
    - In horses with significant airway obstruction, bronchodilators should be administered prior to corticosteroids in order to optimize lung deposition.

- ß2-agonists
    - ß2-agonists induce airway smooth muscle relaxation regardless of bronchoconstriction mechanism and also inhibit mast cell degranulation.
    - Albuterol, pirbuterol, and fenoterol are short acting bronchodilators (1 hour) with rapid onset of action (5 minutes).
    - Salmeterol and formoterol are long-acting ß2-agonists suitable for twice daily dosing but with slow onset of action.
- Anticholinergics
    - Ipratropium bromide is an anticholinergic drug chemically derived from atropine but devoid of side effects when administered by inhalation.
    - Nebulization of 2 µg/kg causes bronchodilation for approximately 6 hours.
    - The effects of anticholinergic drugs on airway smooth muscle are additive to ß2-agonists.

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# ASSESSMENT OF UPPER RESPIRATORY TRACT OBSTRUCTION AND HIGHSPEED TREADMILL ENDOSCOPY

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## Summary

- Most endoscopy is performed on horses which are at rest any yet there are no races for horses standing still - endoscopy is often used to predict events which only occur when the subject is galloping at racing speed
- Highspeed treadmill endoscopy (HSTE) provides the best means to make a definitive diagnosis of dynamic obstruction of the upper respiratory tract in equine athletes
- Other aspects of respiratory and cardiac function can be assessed simultaneously
- HSTE is safe provided that careful screening and training programmes are used
- Malfunctions of the soft palate are the most frequently diagnosed dynamic disorders of the equine upper airways
- The findings of HSTE are frequently at odds with the conclusions reached by endoscopy in the horse at rest.
- Dynamic obstructions are often complex and do not involve single structures or causes

An inability to breathe freely provokes obvious performance-limitations for the athletic horse and the presenting signs in afflicted animals include reduced stamina through an inability to sustain aerobic muscle function and audible respiratory noises through increased turbulence within the upper respiratory tract. The minute volume of normal horses at rest is of the order of 250L but this is increased to as much as 2500L during fast exercise. The pressure gradients required to move such large volumes of air can be as much as 40cm water and these act across the walls of the airway so that considerable "collapsing forces" come into play during inspiration. In the nasal passages and the trachea the semi-rigid structure of the lining of the airway helps to resist this effect but the obvious areas of weakness lie at the nares and in the pharynx and larynx.. At rest 70-85% of the total airway resistance arises at these points but through muscular effort this is reduced to approximately 50% during forced breathing. For example, the action of the naso-labialis muscles dilates the nares, the pharyngeal and palatal musculature helps to maintain the patency of the mus-

cuio-membranous tube of the nasopharynx and the crico-arytenoideus dorsalis muscles abduct the cartilages of the larynx. Obviously there will be occasions when a structural deformity leads to compromised airflow, for example with epiglottal entrapment, sub-epiglottal cysts and arytenoid chondropathy, but it is a failure to withstand the collapsing forces of respiration which is responsible whenever horses show signs of dynamic obstructive dyspnoea.

Earlier comments were made regarding the limitations of endoscopic examinations made during quiet breathing, i.e. when the patient is standing in the stable or has recently returned from exercise. In fact, for the majority of equine clinicians in practice such examinations form the lynch-pin for diagnosis in horses showing signs suggestive of dynamic URT obstruction, but how dependable are the findings? The best means to achieve a definitive diagnosis is to assess the patient while it is exercising vigorously.

The advent of highspeed treadmills and modern instrumentation enables the simultaneous recording of endoscopy of horses exercising at speeds up to 16 m/sec. as well as respiratory sounds, volume flow loops, end-tidal oxygen and carbon dioxide, minute volume, and oxygen consumption on a breath by breath basis.

Horses take to treadmill exercise surprisingly well and the technique is very safe provided that a rigorous screening and familiarisation training routine is adopted. At the University of Bristol over 700 clinical cases have been investigated on the treadmill with an excellent safety record. On arrival all horses are carefully screened for lameness so that no horse with current orthopaedic disease is admitted to the treadmill training programme. A small number are also rejected on the grounds of temperamental unsuitability. Endoscopy of the URT is performed at rest and with the history and palpation findings a tentative diagnosis is made – this is compared later with the true diagnosis when the patient is tested at speed. A gradual process of introduction and training on the treadmill takes place in three sessions over a 48 hour period by which time horses are ready for a full test to the limit of their ability. Some centres use a test routine which is dictated by the speed at which maximum heart rate is reached. At Bristol a step test is used which starts at 6.5 m./sec for 90 secs before the speed is increased to 8 m./sec followed by increments of 1 m./sec every minute until the horse fails to keep up with the treadmill – the point of fatigue. All tests are conducted on a 10% slope and most horses reach the point of fatigue between 11 and 12 m./sec but exceptional athletes may reach 13 m./sec. The overall procedure of treadmill testing is noisy and trainers who ask whether a 'noise' was heard from the horse during the test would otherwise be disappointed if it were not for the simultaneous recording from a microphone placed in the face mask which also accommodates sensors to measure other respiratory parameters such as flow rates of the inspired and expired air as well as its content.

The data from five hundred and fifty horses undergoing highspeed treadmill endoscopy (HSTE) have now been reviewed and the diagnoses reached were as follows:

| | |
|---|---|
| No upper respiratory tract abnormality seen | 123 |
| Dorsal displacement of the soft palate (DDSP) | 179 |
| Palatal instability (PI) 145 | |
| Dynamic arytenoid cartilage collapse (ACC) | 57 |
| Vocal cord collapse (VCC) without ACC | 16 |
| Axial deviation of the aryepiglottal fold (ADAEF) alone | 15 |
| ADAEF with other forms of dynamic collapse | 55 |
| Fourth branchial arch defects | 7 |
| Right sided ACC / VCC | 3 |
| Epiglottal entrapment | 2 |
| Epiglottal retroversion | 2 |
| Posturally dependent laryngeal collapse | 1 |

The finding that malfunction of the soft palate constitutes the most frequent definitive diagnosis (324 out of 550 horses) is consistent with previous reports (Morris and Seeherman, 1991; Kannegieter and Dore, 1995). This should come as no surprise as it is now widely agreed that a diagnosis of DDSP cannot be made from endoscopic findings at rest. The report from the jockey is far more reliable!

Current opinion is that DDSP results from weakness of the palatal musculature and an inability on the part of the horse to maintain an efficient oro-pharyngeal seal between the ventral aspect of the palate and the dorsal aspect of the tongue. Once air enters the oro-pharynx the palate becomes destabilised like a wet sail and eventually spontaneously moves to a position dorsal to the epiglottis. There is some evidence that DDSP arises following upper respiratory tract infections and it is conjectured that the innervation to the palatal muscles – the pharyngeal branch of the vagus – is damaged by infection where it passes through the auditory tube diverticulum and lies immediately under the respiratory epithelium. In treadmill terms a predictable sequence of events occurs leading up to DDSP. This commences with billowing movements by the more rostral portion of the soft palate corresponding to separation of the oro-pharyngeal seal, followed by flattening of the epiglottis against the caudal soft palate representing the opposition between the contraction of the hyoepiglotticus muscle and the elevation of the palate by the air ventral to it. Eventually the free border of the palate slips from under the epiglottis and vibrates causing obstruction particularly during expiration and there is an immediate dramatic reduction in ventilation.

One aspect of the study of the horses referred to the University of Bristol's Equine Sports Medicine Centre was to compare the RLN gradings of horses examined at rest on arrival with the diagnosis achieved by endoscopy on the treadmill - 459 horses were reviewed for this aspect of the study. (Franklin, 2002).

| | | |
|---|---|---|
| 82 Grade 1 at rest yielded | 1 | Arytenoid cartilage collapse (ACC) on treadmill |
| | | 3 Vocal cord collapse (VCC) |
| 256 Grade 2 at rest yielded | 11 | ACC |
| | 4 | VCC |
| 82 Grade 3 at rest yielded | 20 | ACC |
| | 9 | VCC |
| 23 Grade 4 at rest yielded | 19 | ACC |
| | 0 | VCC |
| 6 Grade 5 at rest yielded | 6 | ACC |
| | 10 | Others |

Thus, in a population of horses which were referred for the investigation of poor performance, often with a history of abnormal respiratory noise, only 29 / 82 (35%) animals with Grade 3 RLN at rest were confirmed to show dynamic collapse of the left arytenoid cartilage and/or vocal fold during treadmill exercise. Four horses out of 23 (17.4%) with a Grade 4 score at rest and would have been expected to show dynamic collapse on the left side of the larynx showed consistent near-symmetrical abduction throughout HSTE and were spared unnecessary laryngeal surgery. These results also show that there were isolated cases (19 / 338 – 5.6%) where horses showing 'normal' laryngeal motility – Grades 1 and 2 - during quiet breathing showed dynamic arytenoid or cord collapse under exercise conditions. In all cases there was palpable wasting of the laryngeal musculature on the left side and the patients made progressive 'roaring' noises at exercise.

To the unaided human ear the inspiratory noises produced by horses afflicted with axial deviation of the ary-epiglottal folds (ADAEF) is not distinguishable from the 'roaring' or 'whistling' associated with RLN. In this series of 550 horses there were 15 horses with ADAEF and no other concurrent form of dynamic collapse in the URT, 44 where it was associated with palatal malfunction, 7 where there was concurrent dynamic collapse of the arytenoid or vocal fold and 4 where it was the major source of airway obstruction in cases of fourth branchial arch defect. The cause of ADAEF is not known. The tissue which shows dynamic collapse is not muscular and represents the lateral extensions of the glosso-epiglottic mucosa. Thus, simple stretching of the mucosa seems to be responsible although it is unclear why some horses show ADAEF and others exhibit epiglottal entrapment.

There is a tendency in clinical practice to make simple single diagnoses but HSTE has shown that dynamic collapse in the equine upper respiratory tract is often complex with multiple sources of obstruction. On the other hand the traditional assumption that RLN and DDSP often go together holds little water. Only 17 of the 73 (23.3%) horses with collapse of the arytenoid cartilage and / or vocal fold showed concurrent palatal malfunction – DDSP or PI.

HSTE has led to an improved understanding of the dynamics of previously recognised disorders such as recurrent laryngeal neuropathy, epiglottal entrapment and dorsal displacement of the soft palate. However, it has also led to a broadened repertoire of diagnoses so that dynamic obstructions of the URT which cannot begin to be identified during quiet breathing can now be recognised. Terms such as dynamic collapse of the pharyngeal walls or axial deviation of the ary-epiglottal folds (ADAEF), retroversion of the epiglottis, posturally dependent laryngeal collapse and dynamic rostral displacement of the palatal arch can now be added to the equine clinician's vocabulary, and no doubt there will be more to come!

An understanding of the aetiopathogenesis of palatal instability and dorsal displacement of the soft palate is far from complete. A combination of analysis of pharyngeal dynamics in naturally occurring cases of DDSP and PI during HSTE and experimental surgery suggests that weakness of the palatal musculature and a failure to maintain an effective oro-pharyngeal seal is responsible. The epiglottis appears to be an innocent party in the onset of DDSP and surgical techniques which interfere with the epiglottis are irrational. The role of palate resection surgery (staphylectomy) is also dubious other than as a means to reduce the volume of tissue contributing to the overall expiratory obstructive effect of the disorder. The use of techniques which increase the tension in the oral aspect of the soft palate by partial thickness resection (Ahern procedure), cautery or the injection of sclerosis chemicals has become fashionable. These may improve the stability of contact between the underside of the palate and the upper surface of the tongue but they only involve a thick layer of glandular tissue. No technique has been devised which will make the muscles of the palate stronger and this being the case all are doomed to have a have a "success" rate of about 60%. One group of patients worthy of specific comment consists of those which "quit out" abruptly in races and on the treadmill develop PI. It has been suggested that these horses have experienced DDSP in the past and are pre-empting the partial asphyxiation which accompanies it by stopping once the initial stage of palatal weakening has been sensed.

Clearly not all horses which show poor or deteriorating performance and/or which make untoward respiratory noises at exercise can or should be referred for HSTE. The selection of horses for this technique should include those patients where the noises heard at exercise cannot be explained by the findings of palpation or endoscopy; horses which fade abruptly in races without abnormal respiratory sounds; horses where previous airway surgery has been unsuccessful; and horses which have been subjected to nerve-muscle pedicle graft surgery to ameliorate RLN and where the outcome cannot satisfactorily be evaluated during quiet breathing.

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# EQUINE PLEUROPNEUMONIA

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## Introduction

Bacterial pleuropneumonia, frequently referred to as pleuritis, is a common and often severe disorder of horses (1-8). The condition involves bacterial colonization of the pulmonary parenchyma, development of pneumonia and/or pulmonary abscesses, and subsequent extension to the visceral pleura and pleural space. In humans it is noted that up to 40% of patients with bacterial pneumonia have accompanying pleural effusions (9). While similar data is not available for the horse, the increased use of thoracic ultrasound has documented that pleural effusion is not uncommon in any horse with pneumonia and is not restricted to those horses with severe pleuropneumonia (10).

## Pathogenesis

The first stage of bacterial pleuropneumonia is an exudative stage characterized by rapid outpouring of sterile pleural fluid into the pleural space in response to inflammation of the pleura. The associated pneumonic process is usually contiguous with the visceral pleura and results in increased permeability of the capillaries in the visceral pleura. If appropriate antimicrobial therapy is initiated at this stage the pleural effusion may progress no further.

With progression, the bacteria invade the pleural fluid from the contiguous pneumonic process and the second, fibropurulent, stage evolves. This stage is characterized by the accumulation of large amounts of pleural fluid with many neutrophils, bacteria, and cellular debris. Fibrin is deposited in a continuous sheet covering both the visceral and parietal pleural in the involved area. As this stage progresses, the tendency is to loculation and the formation of limiting membranes. These loculations prevent extension of the empyema, but make drainage of the pleural space with chest tubes increasingly difficult.

The last stage is the organization stage in which fibroblasts grow into the exudate from both the visceral and parietal pleura surfaces and produce an inelastic membrane called the pleural peel. This inelastic pleural peel encases the lung and renders it virtually functionless. At this stage the exudate is thick.

Although pleuropneumonia can occur spontaneously, it is often associated with a stressful event such as transportation over an extended distance (1) or recent illness from acute viral disease. It is most commonly seen in Thoroughbred and Standardbred racehorses. Aspiration of pharyngeal secretions may play a significant role in the etiology of pleuropneumonia, as suggested by the bacterial populations responsible for pleuropneumonia. Transportation of horses usually involves an elevation in environmental temperature and relative humidity and increase in the number bacterial organisms within the air. These changes, combined with the stress of transportation, may predispose the animal to development of lower respiratory disease. The aerobic bacteria most commonly involved in equine pleuropneumonia include ß-hemolytic *Streptococcus spp., Pasteurella spp., Actinobacillus spp., E. coli* and *Klebsiella pneumoniae.* The majority of the horses have a mixed infection, with both aerobic and anaerobic bacteria. Commonly isolated anaerobes include *Bacteroides spp.* and *Clostridium spp.* A wide variety of other anaerobes are commonly found in these horses (2).

## Clinical signs

Clinical signs include fever, anorexia, depression, cough, respiratory distress, stiff gait, weight loss, sternal or limb edema and colic. In the acute stage of pleuritis, pain in the thorax may be elicited by palpation over the thoracic wall. Pain is demonstrated by grunts, intercostal muscle spasm, or even escape maneuvers by the patient. Horses may abduct their elbows and have a ``catch'' to inspiration. As more fluid accumulates in the pleural space and the disease becomes chronic, pain is less evident. Auscultation of a horse with pleuropneumonia reveals a normal lung sound in the dorsal lung field with no sounds or only bronchial tracheal sounds heard ventrally. Pleural friction rubs are often not heard because they are present only in the acute stage of the disease. If they are heard, friction rubs are present predominantly at the end of inspiration and the early part of expiration. They disappear as inflammation decreases or as pleural fluids accumulate. Cardiac sounds are often heard over a wider area of the chest than normal, probably as a result of enhanced conduction of sound through the pleural fluid. Thoracic percussion frequently confirms the impression gained from auscultation. Pleural effusion causes a dullness of the ventral aspects of the lung field and is often delineated by a horizontal line.

## Diagnostic procedures

### Thoracic ultrasonography

Thoracic ultrasonography (10-14) is currently regarded as the preferred method to diagnose pleuropneumonia in the horse. While the value of the art of thoracic auscultation and percussion should not be undermined, clinicians managing horses with thoracic disease recognize the limitations of these tools. With the widespread use of thoracic ultrasound, the equine practitioner currently has the ability to determine not only the presence of pleuropneumonia, but also the location and the extent of the disease. Although sector scanners are superior (preferably 3.5-5.0 MHz transducers), linear probes can also be used to evaluate the thorax in practice.

Thoracic ultrasonography in horses with pleuropneumonia allows the clinician to characterize the pleural fluid and to evaluate the severity of the underlying pulmonary disease (14). The appearance of the pleural fluid may range from anechoic to hypoechoic, depending on the relative cellularity. This fluid is usually found in the most ventral portion of the thorax and causes compression of normal healthy lung parenchyma with retraction of the lung toward the pulmonary hilus. The larger the effusion, the greater the amount of compression atelectasis and lung retraction that occurs.

The presence of adhesions, pleural thickening, pulmonary necrosis and compression atelectasis can also be detected (14). Fibrin has a filmy to filamentous or frond-like appearance and is usually hypoechoic. Fibrin is deposited in layers or in web-like filamentous strands on surfaces of the lung, diaphragm, pericardium, and inner thoracic wall limit pleural fluid drainage (14). Dimpling of the normally smooth pleural surface results in the appearance of 'comet-tail' artifacts, created by small accumulations of exudate, blood, mucus, or edema fluid. Pulmonary consolidation varies from dimpling of the pleural surface to large, wedge-shaped areas of sonolucent lung. Atelectic lung is sonolucent and appears as a wedge of tissue floating in the pleural fluid. Necrotic lung appears gelatinous and lacks architectural integrity. Peripheral lung abscesses are identified ultrasonographically by their cavitated appearance and the absence of any normal pulmonary structures (vessels or bronchi) detected within. While detection of a pneumothorax may be easy for the experienced ultrasonographer it is not as easy for the less experienced. The gas-fluid interface can be imaged moving simultaneously in a dorsal to ventral direction with respiration, the "curtain sign" reproducing the movements of the diaphragm. The dorsal air echo moves ventrally during inspiration, similar to the lowering of a curtain, gradually masking the underlying structures. A pneumothorax without pleural effusion is even more difficult to detect ultrasonographically. While free bright gas echoes within the pleural fluid can occur following thoracentesis, they are more often seen with anaerobic infections or when sufficient necrosis has occurred in a segment of parenchyma to erode into an airway and form a bronchopleural fistula. The absence of gas echoes in pleural fluid does not rule out the possibility that anaerobic infection may be present.

Ultrasonography is a valuable diagnostic aid in the evaluation of the pleura, lung, and mediastinum of horses with pleuropneumonia. The detection and further characterization of the above abnormalities improves the clinician's ability to form a more accurate prognosis. Adhesions can be detected which ultimately may affect the horse's return to his previous performance level. Horses with compression atelectasis and a non-fibrinous pleuritis have an excellent prognosis for survival and return to performance. The detection of areas of consolidation, pulmonary necrosis, or abscesses all increases the probable treatment and recovery time and the prognosis for survival decreases as they become more extensive. Ultrasonography can be used as a guide to sample or drain the area with a large fluid accumulation or the least loculation. These patients often benefit from progressive scanning to assess response to treatment and the need for drainage.

## Thoracocentesis

If pleural effusion is suspected, thoracocentesis should be considered. In the acute stages of pleuropneumonia with small volumes of pleural effusion, thoracocentesis is not necessary if the horse is improving or is not showing signs of respiratory distress. Moderate amounts of pleural effusion may be resorbed quite readily. However, if fluid accumulates rapidly, if the horse is in respiratory distress, or if its condition deteriorates, thoracocentesis should be performed. The preferred site is the sixth or seventh intercostal space just dorsal to the palpable costochrondral junction. Choosing a site farther caudal may provide a sample but does not allow adequate drainage of the chest. When attempting to aspirate pleural fluid from a horse with a minimum amount of effusion, one should choose a space no farther back than the sixth or seventh intercostal space. Thoracic ultrasound aids the site selection. If the procedure has caused some trauma, the first fluid obtained may be blood tinged, but this clears as more fluid is withdrawn. If the pleural fluid is blood tinged because of the underlying disease process, the red coloration persists throughout the entire procedure. An aliquot of pleural fluid is transferred from the syringe into tubes containing anticoagulant solution (EDTA) so that appropriate laboratory evaluation may be performed. Part of the fluid

should be saved in sterile containers with transport media for subsequent Gram stain and culture. Fluid should be removed as long as it flows freely. Both sides of the thorax should be tapped.

## Examination of pleural fluid

The color, turbidity, viscosity and odor should be noted. Normal pleural fluid is clear and yellow; cloudiness reflects an increased number of WBCs. Putrid-smelling pleural fluid is a hallmark of anaerobic infection; however, the absence of odor does not exclude anaerobic infection. In addition to the odor of the pleural fluid, the odor of the horse's breath should be noted, particularly after coughing. The majority of horses with anaerobic infections have a putrid odor associated with the pleural fluid or breath. These horses have a low survival rate.

The WBC count of normal pleural fluid is generally less than 10,000/ul. WBC count of pleural fluid in pleuropneumonia can range from 1600 to 300,000 cells/ul, varying in the same pleural fluid sample between the beginning and the end of the thoracocentesis. There is no association between the WBC count in pleural fluid and survival. Pleural fluid protein is greater than 3 g/dl in horses with pleuropneumonia, but this is also not a prognostic indicator.

Pleural fluid should be Gram stained and cultured for bacteria. The Gram stain may provide tentative identification until the culture results are obtained. Both aerobic and anaerobic cultures should be performed. Anaerobes occur in 46% of horses with pleuropneumonia. The pleural fluid used for anaerobic cultures should be transferred to the laboratory immediately after collection in a manner that prevents or minimizes exposure to air. Anaerobic transport media is commercially available and should be routinely used. Specimens submitted for isolation of anaerobes should not be refrigerated, since many anaerobes are intolerant to cold. Isolation of anaerobic bacteria from either the pleural fluid or tracheobronchial aspirate provides a poor prognosis.

While pleuropneumonia is the most common cause of pleural effusion in the horse, the second most common cause is neoplasia. Differentiating between the two conditions is a challenge for the equine clinician as there are similarities in the clinical signs and physical examination findings.

Pleuropneumonia effusions are more likely to have abnormal nucleated cell count greater than 10,000/ ul (usually greater than 20,000) with greater than 70% neutrophils. Bacteria are frequently seen both intra and extracellularly. A putrid odor may be present. Neoplastic effusions have variable nucleated cell count. If caused by lymphosarcoma, there may be a predominance of abnormal lymphocytes. However, neoplastic cells are often not readily apparent and a definitive diagnosis may be difficult. Rarely do neoplastic effusions have a putrid odor nor have bacteria seen cytologically.

## Pleural drainage

Following selecting of an appropriate anti-microbial agent, the next decision to be made is whether to drain the pleural space (15). Ideally the decision is based on an examination of the pleural fluid. If the pleural fluid is thick pus, drainage using a chest tube should be initiated. If the pleural fluid is not thick pus, but the Gram stain is positive and WBC counts are elevated, pleural drainage is recommended. Another indication for therapeutic thoracocentesis is the relief of respiratory distress secondary to a pleural effusion.

There are many options for thoracic drainage including the following: intermittent chest drainage, indwelling chest tube, pleural lavage, pleuroscopy and debridement, open chest drainage / debridement - no rib resection (standing), open chest drainage /debridement - rib resection (standing), open chest drainage and debridement (general anesthesia), and lung resection (general anesthesia). Drainage of a pleural effusion can be accomplished by (1) using a cannula, (2) indwelling chest tubes or (3) thoracostomy. Thoracostomy (16) is reserved for severe abscessation of the pleural space. Thoracocentesis is easily accomplished in the field and may not need to be repeated unless considerable pleural effusion re-accumulates. Indwelling chest tubes are indicated when continued pleural fluid accumulation makes intermittent thoracocentesis impractical. If properly placed and managed, they provide a method for frequent fluid removal and do not exacerbate the underlying pleuropneumonia or increase the production of pleural effusion. The chest entry site and end of the drainage tube must be maintained aseptically. A one-way flutter valve may be attached to allow for continuous drainage without leakage of air into the thorax. If a chest tube is placed aseptically and managed correctly, it can be maintained for several weeks. It should be removed as soon as it is no longer functional. Heparinization of tubing after drainage helps maintain patency. Local cellulitis may occur at the site of entry into the chest, but is considered a minor complication. Bilateral pleural fluid accumulation requires bilateral drainage in most horses. Open drainage or thoracostomy may be considered when tube drainage is inadequate. It is important not to begin open drainage too early in the disease. An incision is made in the intercostal space exposing the pleural cavity and causing a pneumothorax, unless the visceral and parietal pleura adjacent to the drainage site have not been fused by the inflammatory process. The wound is kept open for several weeks while the pleural space is flushed and treated as an open draining abscess.

## Pleural lavage

Pleural lavage may be helpful to dilute fluid and remove fibrin, debris and necrotic tissue. Lavage appears to be most effective in sub acute stages before loculae develop; however, pleural lavage may help break down fibrous adhesions and establish communication between loculae. Care must be exercised that infused fluid is communicating with the drainage tube. Lavage can be performed by infusing fluid through a dorsally positioned tube and draining it through a ventrally positioned tube. Ten liters of sterile, warm lactated Ringer's solution is infused into each affected hemithorax by gravity flow. After infusion, the ventrally placed chest tube is opened and the lavage fluid is allowed to drain. Pleural lavage is probably contraindicated in horses with bronchopleural communications because it may result in spread of septic debris up the airways. Coughing and drainage of lavage fluid from the nares during infusion suggests the presence of a bronchopleural communication.

**Tracheobronchial aspirate** provides an excellent specimen for Gram stain and bacterial culture.

**Thoracic radiography** is often limited by the availablity of facilities. Lateral thoracic radiographs can often show small amounts of pleural effusion not detectable by either auscultation or percussion.

**Pleuroscopy,** a procedure in which a flexible or rigid endoscope is introduced into the pleural space, is rarely indicated in bacterial pleuropneumonia. This technique is better reserved for equine patients with pleural effusion of undiagnosed etiology.

**Hematologic** findings in horses with pleuritis are usually nonspecific and do not predict the outcome of the case. A low hematocrit (less than 30%) usually reflects an anemia of chronic disease, whereas elevated total plasma proteins (greater than 8 g/dl) are probably caused by hypergammaglobulinemia. Both these findings suggest that the pleuropneumonia is chronic. WBC counts can be misleading, since not all affected horses have a leukocytosis. Plasma fibrinogen appears to be a more sensitive indicator of inflammation because it is elevated in almost all cases of pleuropneumonia.

## Management

The primary goals of managing a horse with pleuropneumonia are to stop the underlying bacterial infection, remove excess inflammatory exudate from the pleural cavity, and provide supportive care. Ideally an etiologic agent is identified from either the tracheobronchial aspirate or pleural fluid, and antimicrobial sensitivity determined. Without bacterial culture results, broad-spectrum antibiotics should be used because many horses have mixed infections of both gram-positive and gram-negative and aerobic and anaerobic organisms. Commonly used therapy is penicillin combined with an aminoglycoside such as gentamicin, enrofloxacin, trimethoprim and sulfamethoxazole, or chloramphenicol. Because of the need for long-term therapy, initial intravenous or intramuscular antimicrobials may need to be followed by oral antimicrobials. Preferably the oral antimicrobials are not administered until the horse's condition is stable and improving because blood levels obtained by this route are not as high as those achieved following intramuscular or intravenous administration.

Treatment of anaerobic pleuropneumonia is usually empiric, since antimicrobial susceptibility testing of anaerobes is difficult because of their fastidious nutritive and atmospheric requirements. Thus familiarity with antimicrobial susceptibility patterns is helpful in formulating the treatment regimen when an anaerobe is suspected. The majority of anaerobic isolates are sensitive to relatively low concentrations (22,000 IU/Kg bwt IV four times per day) of aqueous penicillin. *Bacteroides fragilis* is the only frequently encountered anaerobe that is routinely resistant to penicillin, although other members of the Bacteroides family are known to produce ß lactamases and are potentially penicillin-resistant. Chloramphenicol (50 mg/kg orally four times per day) is effective against most aerobes and anaerobes that cause equine pleuropneumonia. However, because of human health concerns, the availability of chloramphenicol may decrease. Metronidazole has in vitro activity against a variety of obligate anaerobes including *B. fragilis.* Pharmacokinetic studies indicate a dose of 15 mg/kg intravenously or orally four times a day is necessary to maintain adequate serum levels. Oral administration rapidly results in adequate serum levels and thus is an acceptable route of administration for horses with pleuropneumonia. Metronidazole is not effective against aerobes and therefore should always be used in combination therapy, at a dose of 15 mg/kg three or four times per day. It is important to recognize the side effects of metronidazole, including loss of appetite and lethargy, and to stop the use of the drug when these signs are observed. Aminoglycosides and enrofloxacin should not be considered for the treatment of pleuropneumonia caused by an anaerobe unless it is used in combination therapy (i.e., with penicillin).

Other therapies: Antiinflammatory agents help reduce pain and may decrease the production of pleural fluid. This in turn may encourage the horse to eat and maintain body weight. Flunixin meglumine (500 mg once or twice daily) or phenylbutazone (1 to 2 g twice a day) is commonly used for this purpose. I believe that corticosteroids are contraindicated in bacterial pleuropneumonia. Rest and the provision of an adequate diet are important components of the treatment of pleurop-

neumonia. Because the disease course and period of treatment are usually prolonged, attempts should be made to encourage eating. Intravenous fluids may be indicated in the acute stages of the disease to treat dehydration from anorexia and third-space losses into the thorax.

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# RECENT ADVANCES IN THE DIAGNOSIS AND MANAGEMENT OF EQUINE SINUS AND NASAL DISORDERS

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## Introduction

The clinical signs associated with diseases of the paranasal sinuses of the horse include nasal discharge, which may be mucoid, purulent, haemorrhagic or a combination of these, facial swelling and obstructive dyspnoea. Expansive lesions in this area may produce facial swellings or obstruct nasal breathing and on occasions will displace orbital tissues resulting in exophthalmos. It is exceptional for a sinus disorder to extend caudally into the cranium to provoke central nervous signs. Nevertheless, the discomfort associated with sinu-nasal disorders may be responsible for behavioural changes in some horses. The intranasal structures are richly vascular and it is not surprising that trauma and destructive conditions frequently lead to epistaxis.

The purpose of this contribution will be to present an overview of "sinusitis" as it afflicts the horse, in terms of its diagnosis and management and to comment on three more recent developments which are applicable in clinical practice: (1) *** the diagnostic value of direct sinus endoscopy, (2) *** the technique of frontal flap surgery performed on the standing horse and (3) *** intralesional treatment of progressive ethmoidal haematomas.

## Anatomical Considerations

The paranasal sinuses of the horse are extensive air-filled spaces lined by mucoperiosteum. The human nasal mucosae produce in excess of 0.5 litres of mucus daily and thus it is reasonable to suggest that the paranasal sinus lining of the horse secretes several times this volume. **The normal removal of mucus is a dynamic process depending on mucociliary flow to the drainage ostia** which do not lie at the lowest points in the sinuses. Once the nasal meati are reached mucus is lost by a combination of evaporation and further mucociliary flow towards the nasopharynx. The aetiopathogenesis of primary sinusitis in horses hinges on stagnation of mucus in the sinus cavities through inhibition of dynamic clearance. Other sinu-nasal disorders such as space-occupying conditions produce secondary empyema by physical obstruction of the drainage ostia.

The five paired paranasal sinuses of the horse are:

1) Frontal/conchofrontal
2) Caudal maxillary
3) Rostral maxillary
4) Ethmoidal
5) Sphenopalatine

The frontal sinus is divided into conchofrontal (CFS) and frontal (FS) portions. Drainage takes place through the fronto-maxillary foramen into the caudal maxillary sinus (CMS). The ethmoidal and sphenopalatine sinuses also drain via the CMS into the middle nasal meatus. The rostral maxillary sinus (RMS) has an independent drainage ostium, again into the middle nasal meatus. The RMS is divided into a lateral bony and a medial turbinate portion within the ventral conchus (ventral conchal sinus, VCS). These are separated by the infra-orbital canal and a sheet of bone joining it ventrally to the roots of the cheek teeth. It can be seen that in the young horse the lateral bony compartment is largely occupied by the roots of the cheek teeth and that, regardless of age, the VCS is not easily accessible for surgery or for direct endoscopy other than via the floor of the CFS. A thin-walled bulla of this compartment extends caudally into the CMS and can be appreciated on radiographs.

The roots of cheek teeth 4, 5 and 6 lie within the maxillary sinuses: these are most prominent in young horses and recede towards the floor of the sinuses with age. The roots of CT3 form the rostral wall of the RMS. When dental periapical suppuration is the cause of sinusitis, the tooth roots are approached with a trephine site located as directly over the roots as vital structures such as the nasolacrimal canal, infra-orbital canal, vein and artery of the angle of the eye will permit. It should be noted that repulsion of maxillary CT6 is performed through the frontal sinus, but fortunately extraction of this tooth is rarely indicated.

## Objectives of Management

The management of sinus diseases of the horse depends upon a decision-making process with clearly defined objectives**: to identify and eliminate the cause of disease, and to restore function.** At the onset of 'sinusitis', once dynamic mucociliary clearance has been compromised, usually by a viral infection, mucus removal reverts from an active to a passive process with greater or lesser opportunist bacterial infection through pooling and stagnation of mucus in the dependent spaces. Traditionally it was proposed that the drainage ostia were physically obstructed at an early stage but this cannot be the case when the earliest sign of sinusitis is a nasal discharge. Passive overflow of mucus/mucopus through patent ostia is a more rational explanation. In the face of chronic empyema the sinus lining may develop hyperplasia and necrosis. At this stage passive drainage may be obstructed by thickening of the tissues bordering the ostia and/or by inspissation of pus. A facial swelling develops as pressure within the sinus cavities increases.

**1) Identification of the primary sinus disorder:**

The principle diagnostic procedures to investigate sinu-nasal conditions comprise external assessment, radiography, endoscopy via the nostrils, direct endoscopy through the sinus wall, exploratory surgery and biopsy. An accurate case history is invaluable and particular note should be made of possible contact with infectious respiratory disease and of the duration and nature of any nasal discharge. It is unusual for sinusitis to be bilateral and it is logical that the discharge will be largely unilateral whenever its origin lies rostral to the caudal limit of the midline septum. Even when a horse is presented with unilateral epistaxis enquiries should be made regarding possible associations with exercise to eliminate a diagnosis of EIPH. Epistaxis due to guttural pouch mycosis may be acute and, even if episodic, the course of the history is unlikely to exceed 3 weeks. Progressive ethmoidal haematoma (PEH) is a more likely explanation when episodes of epistaxis span a longer period, especially if the blood is not fresh. A foetid nasal discharge points to suppuration but this could arise from a wide range of chronic sinus lesions. Horses involved in steeplechasing, hurdling, show jumping or eventing may become unreliable jumpers in the face of sinusitis, presumably reflecting a painful jarring sensation on landing.

A External assessment

The facial area should be inspected for evidence of deformity of the supporting bones through swelling or trauma. Percussion of the walls of the paranasal sinuses is an unreliable technique but increased resonance may be perceived when the walls become thin or dullness may develop when the sinuses are completely filled by fluid or soft tissue. The airflow at each nostril should be checked to assess obstruction of the nasal meati. The clinical crowns of the cheek teeth are examined for the presence of fracture, displacement or impaction of degenerate ingesta. The patency of the nasolacrimal duct can be checked by catheterisation and infusion of saline solution from either end.

B Radiography (discussed above)

C Endoscopy

The endoscopic examination of the nasal area is performed in two ways. First, by conventional passage of the instrument into the nasal meati and, second, by direct inspection of the sinus contents with the endoscope passed through a small trephine hole. It is not exceptional for major abnormalities to be identified by direct endoscopy into the sinuses when conventional examinations per nasum have revealed little untoward. All endoscopy of this region is best performed on the standing horse because orientation is straightforward and the nasal tissues of the recumbent animal become discoloured and engorged.

Endoscopy via the nose may reveal changes such as narrowing of the nasal meati; distension of the ventral or dorsal conchi reflecting filling of the ventral conchal and concho-frontal sinuses respectively; the presence of a soft tissue mass such as an ethmoidal haematoma; discharge streaming from the sinus drainage ostium; and mycotic plaques on the surface of the conchal mucosa.

## *** Direct sinus endoscopy (DSE)

It is generally not possible to pass an endoscope through the drainage ostia of the paranasal sinuses unless there has been distortion by surgery or disease. Thus, it is perfectly possible for sinus disorders to remain undetected by endoscopy per nasum and for an incomplete diagnosis to be made unless the contents of these airspaces are inspected directly.

### *Indications for direct sinus endoscopy*

The sinus compartments which lend themselves best to DSE are the caudal maxillary (CMS), the true frontal (FS) and the concho-frontal (CFS) sinuses. These are inter-connected via the fronto-maxillary foramen and, coincidentally, share a common drainage ostium to the middle nasal meatus.

Most disorders of the paranasal sinuses lead to the presence of free fluid accumulation and/or a soft tissue mass within the sinus airspaces and both of these changes can readily be seen on erect lateral radiographs of the head. The presence of blood, pus or mucopus emerging from the caudal area of the middle meatus establishes the sinuses as the source of a nasal discharge and yet there may be no other evidence of disease to identify by endoscopy of the nasal structures. For example, in cases of ethmoidal haematoma or mycosis confined to the sinuses. PEHs may arise from the nasal aspect of the ethmoturbinates, in which case they will be available for diagnosis by endoscopy per nasum, or on the sinus surfaces, in which case they will not. A complete diagnostic evaluation for PEH requires that four sites are checked, i.e. endoscopy via left and right nares, and left and right DSE even when the clinical signs are unilateral. Mycotic infections of the nasal region produce destructive changes and may be confined to the lining of the sinuses. Surprisingly, the changes on radiographs are often minimal and endoscopy via the nares can be unrevealing unless the mycotic plaques have destroyed through the conchi to the nasal surfaces. Thus, DSE is indicated for those horses which show a persistent, unilateral, low-grade, malodorous nasal discharge with non-specific findings on radiographs. DSE is not normally necessary in the face of primary empyema but should be considered if the condition fails to respond to more conservative methods of management.

### *Technique and equipment for DSE*

Although there are reports of the use of rigid endoscopes, i.e. arthroscopes, for DSE and a variety of entry points have been suggested, the author has found that flexible equipment passed into the CMS provides a simple and effective approach. A flexible gastroscope with an external diameter of 7.5 - 9.0 mm is suitable but, of course, a trephine with a slightly larger diameter is also needed.

A linear rather than circular incision is used so that it can be closed with sutures if there are negative findings within the sinuses, but a treatment catheter can still be placed if required. The incision is made over the CMS, bisecting the angle between the bony margin of the orbit and the facial crest. The periosteum is cleared in the usual way. Care is required to make sure that the bony edges are smooth for fear of damage to the outer covering of the endoscope. Strict asepsis may not be possible but the distal section of the endoscope should be thoroughly cleaned and surgical spirit is applied immediately before introduction into the sinus.

***Endoscopic findings during DSE***

The normal landmarks which are readily identifiable include the roots and reserve crowns of the fifth and sixth cheek teeth in the foreground ventrally; the infra-orbital canal running horizontally above the tooth roots; the bulla of the RMS rostrally; the wide fronto-maxillary foramen in the mid-dorsal field and through this the leaves of the ethmoturbinates. The FS and CFS can be inspected by advancing the endoscope through the fronto-maxillary foramen.

DSE is not normally required in cases of primary sinusitis, but inflammation of the mucosal lining and the pooling of a mucus-based discharge ventrally would confirm such a diagnosis. Dental periapical suppuration produces such gross proliferative changes around the tooth roots that the view for DSE is likely to be obscured and a specific diagnosis may not be possible. Ethmoidal haematomas are usually obvious although the colour of the lesions is very variable. Care should be taken to inspect the dorsal aspect of the ethmoids in the FS where small lesions may be overlooked. Large lesions on the nasal surfaces of the ethmoids may distort the medial walls of the sinuses. Again, there is no diagnostic subtlety regarding mycotic infections - these lesions produce obvious plaques from which material can be taken for culture and typing. Less common lesions which may be seen by DSE include sinus cysts and neoplasms - both conditions are more likely to be diagnosed on the basis of radiographs and exploratory surgery. However, in horses with suspected neoplasia DSE offers a means to obtain representative tissue samples for pathological interpretation.

The rostral maxillary sinus (RMS) is not easily amenable to DSE because of the position of the infra-orbital canal as well as the reserve crowns and roots of the cheek teeth. Also, there are minimal indications to inspect the contents of this compartment. The exception might be chronic empyema but frankly this is far better dealt with using the standing frontal flap technique described below.

**2 Return of a normal drainage system or creation of alternative drainage through a sinu-nasal fistula:**

Non-surgical methods to manage sinusitis include antibiotics, mucolytics, steam inhalations, volatile inhalations and continued exercise. The objective is the return of normal mucociliary clearance mechanism. Simple trephination has similar intentions with the addition of irrigation, possibly including topical antibiotic agents, to clear stagnant mucus and eliminate secondary infection.

In the face of chronic sinusitis, sinus cyst and PEH the natural drainage system may be physically obstructed. Fistulae can be made by removal of a medial section of the floor of the CFS or medial wall of the VCS so that there is free communication between the sinus cavities and the nasal meati. Fistulation surgery can be highly haemorrhagic and should not be attempted in the standing patient.

**3 Adequate visibility for accurate diagnosis and surgery**

The miserable visibility conveyed by a traditional trephine hole can be greatly enhanced by direct endoscopy as described above. However, frontonasal or maxillary flap surgery is required for more extensive excisional procedures.

## *** Standing facial flap surgery

Good access to the concho-frontal, frontal and maxillary sinus compartments can be achieved through a laterally based frontal flap approach performed with the horse standing, under sedation and local anaesthesia. Such an approach is particularly suitable to manage empyema of the RMS in horses of all ages. The bulla of the RMS may bulge caudally into the CMS when it is inflated by pus and this is easily punctured and partly excised. There is less haemorrhage with the standing approach because the postural change of anaesthesia is avoided and orientation is simple. A standing approach might also be used to ablate those ethmoid haematomas which prove inaccessible by DSE and small cysts may also be removed by this approach.

### *Technique for standing sinus flap surgery*

The frontal area is clipped and prepared for clean surgery in the normal manner and at the same time the site for CMS trephination is also prepared for the later placement of an irrigation catheter. Heavy continuous sedation is provided using a detomidine and butorphanol combination administered in an intra-venous drip. Local anaesthetic is infiltrated between the skin and periosteum over the surgical site which is located over the CFS, avoiding the line of the naso-lachrymal duct. An incision is made through the skin and periosteum together and this should be fashioned at least 3 mm. wider than the intended bone incision. A 5.0 cm. trephine is ideal to make the bone window but if this is not available a series of smaller overlapping holes can be used in the style of Olympic-rings. On entry into the CFS topic local anaesthetic is sprayed onto the lining. Landmarks such as the ethmoidal turbinates, the infra-orbital canal and RMS bulla are identified. The bulla can be punctured with the point of a finger and as much of this structure as possible is resected with scissors and rongeurs. Haemorrhage should be no more than slight but trauma to the medial wall of the exposed VCS should be avoided. Inspection of the sinus contents is followed by ablation of localised lesions, lavage of inspissated pus and the placement of an irrigation catheter. The skin/periosteum incision is closed in a single layer with a combination of sutures and staples. Although the disc of trephined bone is rejected, the cosmetic results should be good.

Facial flap surgery under anaesthesia

Fronto-nasal or maxillary flap surgery under general anaesthesia is required for more extensive and haemorrhagic procedures such as the removal of sinus cysts, large PEHs and selected tumours as well as for fistulation techniques. Fistulae can be made between the sinus cavities and the nasal meati but this may provoke major haemorrhage and cannot be performed safely with the horse standing. In surgical practice the fistulae described provide convenient routes by which to lead sock-and-bandage pressure packs to the nostrils (see below).

Radical exposure of the nasal chambers, paranasal sinuses and their contents can be achieved through the bony walls of the supporting bones. The major surgical options are (a) between frontonasal and lateral (maxillary) approaches and (b) whether to reject or preserve the bone flap. Lesser decisions to be made relate to the shape of the bone flap and the direction in which the skin/periosteal flap is raised.

The lateral approach into the maxillary sinuses provides limited access and it should be reserved for those instances where the disease process is restricted to the maxillary compartments and

older horses are most suitable. The decision to use this approach is generally based on radiographic confirmation on ventro-dorsal projections that the disorder does not extend medial to the infra-orbital canal. The argument against rejection of the bone flap is that a cosmetic blemish in the form of a slight depression will result. However, this author favours rejection because a replaced flap provides a potential focus for infection or sequestration.

The surgical procedures are straightforward. Incisions are made through skin and periosteum in the same plane before the periosteum is peeled away from the underlying bone using an elevator. If the bone flap is to be replaced the skin, periosteum and bone are sectioned in staggered planes but the periosteum should not be separated from the bone. The bone flap is best fashioned with an oscillating saw but in practice hand saws or a series of overlapping trephine holes are alternatives. The bone flap should again be smaller than the overlying skin/periosteal incision. Care must be taken that the bone edges are not devitalised by heat generated by the saw blades as localised osteomyelitis may occur. Once the sinus contents are exposed , the disease focus is identified and removed. Closure of the incision can be achieved with a single layer of mattress sutures but accurate alignment is required for the best cosmetic result in horses with natural facial markings. When staggered incisions have been made and when bone is to be replaced the periosteum and skin are closed as separately and the periosteal layer holds the bone in place.

**4 Control of Haemorrhage**

Sinus surgery is bloody surgery - haemorrhage in a highly vascular area is exaggerated by the postural change in an anaesthetised horse. Care must be taken to provide circulatory support by aggressive intravenous fluid administration although whole blood replacement is rarely necessary. Hypotensive anaesthetic agents are not desirable when prolonged surgery renders horses vulnerable to rhabdomyolysis of the dependent limbs.

Temporary bilateral carotid occlusion can reduce haemorrhage and improve visibility during the intraluminal stages of sinus flap surgery and comprises the pre-placement of snares around each common carotid artery which are drawn tight only for the 15-20 minutes required to complete the intranasal stages of the surgery. However, the advantages of slight haemorrhage reduction must be weighed against the disadvantages of prolonging the surgery and also the risk during placement and removal of the snares of insult to the recurrent laryngeal nerves which run within the carotid fascia.

Pressure packing within the sinus cavities and nasal chambers is essential to control haemorrhage on completion of the surgery and during the initial recovery period, i.e. 48-72 hours. Sock-and-bandage packing consists of lengths of cotton bandage packed into tubular stockinet socks. These are prepared ahead of surgery and 1-4 may be needed for each operation. The open end of the stockinet is led to the nostril through a fistula created between the sinus cavity and the nasal meati. On the third or fourth day after surgery the bandage is withdrawn and a day later the stockinet is removed.

**5 Provision of a safe airway during surgery and recovery**

The use of a cuffed endotracheal tube is essential for sinus surgical procedures performed under general anaesthesia. This protects the lower airways from the inhalation of blood, pus, surgical debris and irrigation fluids during surgery. Vigorous lavage, possibly from a hosepipe, is frequent-

ly used to displace inspissated pus from the recesses of the sinuses and for this inflation of the cuff should be checked and the head lowered. During anaesthetic recovery, until the horse regains its feet, a nasopharyngeal tube should be placed through the non-surgical nasal chamber and secured at the nostril. Tracheotomy intubation is not necessary.

**6 Facilities for topical post-operative treatment and monitoring of progress**

The implantation of a Foley balloon catheter, typically into the CMS, offers a convenient route for post-surgical irrigation and medication. It must be appreciated that blood clots and devitalised tissue are inevitably left after all sinus surgery and opportunist infections, frequently mycoses, are likely to become established. Balloon catheters are recommended as a routine to permit physical displacement of debris by irrigation and for topical medication which should include an antimycotic agent - a portable pressurised garden spray is a practical means of administration which is well tolerated by the patients. Inspection of the sinus contents to monitor progress after sinus flap surgery can be achieved per nasum through the iatrogenic fistulae or laterally through the trephine hole into the CMS.

**7 Early return to exercise**

In spite of the radical nature of some sinus surgical procedures an early return to exercise is to be commended as an integral part of treatment. Forced nasal ventilation increases the evaporation of residual discharges when stagnation might otherwise encourage post-operative infections to become established.

**8 Pleasing cosmetic result**

The cosmetic results of trephination and facial flap surgery are both satisfactory as no more than a slight depression at the operation site should result from either. However, it has been conceded above that when a replaced bone flap heals without complication, the cosmetic result is marginally superior. Occasionally after all sinus surgery proliferative suture periostitis is seen, presumably as a result of vibrations in the suture lines during trephination or sawing.

**Specific indications for sinus surgery in horses:**

1 Dental Extraction

The traditional means to extract the cheek teeth is by repulsion and for maxillary CT 4 - 6 this means trephination through the paranasal sinuses. Checking the oro-antral fistula and sinus content is an important part of nursing and thus the trephine holes should be prevented from closure until the floor of the sinus has healed and secondary infection has resolved. Dental extraction is one indication for sinus surgery where trephination is preferred and facial flaps should be avoided. Once a flap has been closed there is no access to the alveolus for inspection or curettage unless the flap is re-opened.

2 Acute Sinusitis

The normal progress in the treatment of sinusitis is to use medical regimes initially. When these fail and the diagnosis of sinusitis is confirmed by endoscopy and radiography and dental disease has been ruled out, the treatment should progress to the next level with trephination into the CMS in

the corner between the facial crest and orbital margin. This is performed with the horse standing under local anaesthesia. A Foley balloon catheter is placed for direct irrigation and medication and the horse should continue in moderate exercise.

3 Chronic Sinusitis

Failure of simple irrigation is seen as continued nasal discharge, possibly malodorous and arises through necrosis of the sinus lining, inspissation of pus and occlusion of the natural drainage ostia. Comprehensive radiographic and endoscopic investigation aims to eliminate other disease processes and if none is found standing facial flap surgery is indicated. Chronic primary sinusitis must be differentiated from sinusitis secondary to dental periapical suppuration.

4 Removal of Sinus Cysts

They are expansive lesions, usually in the CMS or CFS, with a distinct wall and characteristically contain vivid yellow fluid. At facial flap surgery they can be peeled away from the inside of the sinuses. In the event of incomplete ablation small areas of residual cyst tissue do not appear to cause complications and the condition carries a good prognosis.

5 *** ***Treatment of progressive ethmoidal haematomas***

Progressive ethmoidal haematoma (PEH) is a multifocal disease of unknown aetiology which produces expanding, discrete, soft tissue masses filled with clotted blood which are attached to the surfaces of the ethmoturbinates. Intermittent splitting of the overlying mucous membrane produces the spontaneous leakage of dark blood which is seen at the nostril. Although one mass may be larger than the others and account for the presenting clinical signs which may include obstructive dyspnoea as well as epistaxis, it is rare for lesions to be solitary. Thus, most published surgical series on the treatment of PEH report a significant recurrence rate but whether the secondary lesions are recurrences at the original site or new lesions altogether is far from certain. PEH is probably best viewed as a lifelong affliction for susceptible horses requiring tactical treatment as and when required. Thus, there has recently been a move away from surgical resection of PEH, unless the lesions are very large, to ablation by intra-lesional infusion of euflavin or 10% formalin. Such treatments are performed trans-endoscopically using a bevelled catheter passed through the biopsy channel. the treatments are applied directly via the nose or in combination with DSE. Most lesions require more than one treatment so that horses are re-examined and treated at monthly intervals until clear of lesions. Even so it is quite likely that episodes of recurrence will arise indefinitely.

6 Primary mycotic rhinitis and sinusitis

Although mycotic opportunism is common after surgery or secondary to other suppurative conditions such as dental periapical abscessation horses are encountered where these infections arise on the sinu-nasal tissues without obvious underlying disease. Topical medication with the benzimidazole agent enilconazole has proved to be effective in the author's experience and primary mycosis carries an excellent prognosis. A Foley balloon catheter is placed into the CMS in the usual manner and the sinus cavity acts as a reservoir for the medication which is infused twice daily.

7 Trauma

Fractures of the walls of the paranasal sinuses are common injuries to horses through kicks, falls, etc. They are generally comminuted and sometimes compound. The direct trauma causing the fractures produces a depression of the fragments involved. Haemorrhage, emphysema, sequestration, secondary sinusitis and disruption of the nasolacrimal duct are potential sequels. Each case must be individually assessed and treatment ranges from conservative antibiotic cover to aggressive surgery to control haemorrhage, to remove detached bone fragments and to elevate depressed plates of bone into their normal alignment. A balloon catheter for irrigation/medication should be considered when severe haemorrhage has been sustained and for all cases where the sinuses have been contaminated in compound fractures.

## Complications of Sinus Surgery

The short-term complications which may follow surgical invasion of the nasal chambers and paranasal sinuses have largely been mentioned already together with measures for prevention or control:

1) Haemorrhage
2) Rhabdomyolysis
3) Wound dehiscence
4) Secondary infections, typically mycotic
5) Sequestration/osteomyelitis of the bone flap
6) Meningitis

Meningitis is a rare occurrence and the author's only experience of this complication arose in a horse with an extensive neoplasm which had compressed the cribriform plate. The horse died 3 days after surgery.

Long-term persistence of sinus empyema is again uncommon and is likely to arise from inadequate fistulation between the infected compartments and the nasal meati.

Many of the procedures described above are not subtle but with diligent planning and application of the surgery, general anaesthetic and intensive care, extensive lesions which in former times would have been deemed inoperable, can be brought to a successful conclusion.

# *RHODOCOCCUS EQUI* PNEUMONIA IN FOALS

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## Definition

- *Rhodococcus equi* is one of the most important causes of disease in foals between 1 and 6 months, with most foals showing clinical signs before the age of 4 months.

## Etiology and Epidemiology

- Gram positive, pleomorphic, rod-shaped bacterium
- Is a facultative intracellular opportunistic pathogen capable of surviving within phagocytic cells.
- Saprophytic opportunist, found in equine feces, prospers in herbivore manure and may be aerosolized from soil
- Avirulent *R. equi* are widespread in the feces of horses and their environment on every farm; Plasmid-cured derivatives of virulent *R. equi* strains lose their ability to replicate and survive in macrophages, and fail to induce pneumonia in foals, confirming the absolute necessity of the large plasmid for the virulence of *R. equi*.
- Natural infections in foals are principally by virulent *R. equi*, Unlike most environmental *R. equi*, isolates from pneumonic foals typically contain an 80-90 kb plasmid encoding a family of seven closely related virulence-associated proteins designated VapA and VapC through VapH.1
- The total number of *R. equi* in the environment may be similar on farms with or without a history of *R. equi* infections. However, farms with enzootic disease are more heavily infected with virulent (VapA positive) *R. equi* than those where the disease is not present.
- Although the virulence plasmid is well conserved between isolates, considerable chromosomal variability exists among isolates of *R. equi* from a given farm and multiple strains can be cultured from a sick foal.
- Foals bred on a farm with endemic disease are exposed more frequently to virulent *R. equi* in their environment than those of a farm without the problem.
- Infected foals which constantly shed large quantities of virulent *R. equi* in their feces are the major source of virulent *R. equi*, which may be the mechanism of progressive development of infection on farms with a history of the disease.

- At present, farms with a potential for endemic infection can be distinguished on the basis of the contamination with virulent *R. equi*, so regular examination of foals and their environment by virulence markers might be the most practical approach to control *R. equi* infection on endemic farms.

## Pathogenesis

- Route of infection: inhaled or ingested from soil contamination
- Inhalation of dust particles laden with virulent *R. equi* is the most important route of pneumonic infection in foals.
- Ingestion of the organism is a significant route of exposure and immunization but does not lead to hematogenous pneumonia unless the foal has multiple exposures to very large numbers of bacteria.
- Increased # of virulent organisms in the soil, increased # of foals with *R. equi*
- *R. equi* is a facultative intracellular parasite
- *R. equi* establishes residence there and multiplies in host macrophage
- *R. equi* inhibits phagosome-lysosome fusion
- The ability to replicate within the macrophage is associated with virulence, and correlates in horses with a large plasmid that expresses VapA.

## Clinical signs

### Pulmonary manifestations

- *R. equi* causes an insidious chronic bronchopneumonia in foals characterized often by multiple pulmonary abscesses.
- Slow spread of the lung infection coupled with the remarkable ability of foals to compensate for the progressive loss of functional lung make early clinical diagnosis difficult.
- Early clinical signs may only include a slight increase in respiratory rate and mild fever. These subtle signs are either missed or ignored, allowing the condition to progress.
- Often insidious during its early stages, foals often present with advanced disease at the time clinical signs are first noted.
- Mild lethargy, decreased appetite, dyspnea, tachypnea, nasal discharge, fever, moist crackles and wheezes, flared nostrils
- Most foals are in good body condition.
- Foals may be found dead or more commonly present in acute respiratory distress with a high fever and no previous history of clinical respiratory disease.
- Lung sounds vary considerably and the findings can be confused by referred sounds from the upper airway.
- Inspiratory and/or expiratory wheezes or crackles may be audible over the affected areas which are more commonly located cranioventrally.
- In more severely affected foals, auscultation may only reveal large airway sounds suggesting consolidation. Lung sounds are diminished over areas of severe consolidation or extensive abscess formation.
- Findings on auscultation do not correlate well with the severity of the pneumonia.

## Intestinal manifestations

- Approximately 50% of pneumonic foals present for necropsy also had intestinal manifestations. Only 4% of foals had GI infections without pneumonia
- The majority of foals with *R. equi* pneumonia do not show clinical signs of intestinal disease.
- GI: multifocal ulcerative enterocolitis and typhlitis over the areas of the Peyer's patches with granulomatous or suppurative inflammation of the mesenteric and/or colonic lymph nodes
- Occasionally a single large abdominal abscess
- Peritonitis +/-

## Non-Septic polysynovitis

- Immune mediated polysynovitis seen in some cases
- All or any joint can be affected
- Joint effusion variable.
- Lameness is not apparent or limited to a stiff gait
- Cytologically: non-septic mononuclear pleocytosis
- Bacteriologically: joint fluid negative
- Histologically: lymphoplasmacytic synovitis
- Local therapy of the joints is not needed as the effusion usually resolves as pneumonia does

## Septic arthritis and osteomyelitis

- Bacteremic spread of the organism from the lungs or gastrointestinal tract may occasionally result in septic arthritis and/or osteomyelitis.
- Foals can occasionally develop septic arthritis or osteomyelitis without apparent lung or other source of infection.
- Lameness distinguishes them from foals with immune-mediated polysynovitis.
- Not sure?? Culture and cytology of joint fluid
- Usually primary metaphysitis with bone infection extending through the metaphyseal cortex and into surrounding tissue
- In addition to usually *R. equi* treatment, local lavage may be needed.
- Vertebral body osteomyelitis also seen

## Other manifestations

- Ulcerative lymphangitis
- Cellulitis
- Subcutaneous abscesses
- Uveitis
- Panophthalmitis
- Nephritis and renal abscessation

## General comments

- Foals without clinical disease exposed to contaminated environments may have *R. equi* in their trachea as a result of inhalation of contaminated dust.
- For this reason, bacteriologic culture or PCR detection of *R. equi* from a TBA should be interpreted in the context of cytological evaluation, physical examination and laboratory results.
- A light growth of *R. equi* from a foal with no clinical signs of respiratory disease, normal fibrinogen concentrations and WBC count, and no cytological evidence of airway inflammation is likely an incidental finding.
- Nucleic acid amplification based on the VapA gene sequence by PCR is a slightly more sensitive mean of identifying *R. equi* in TBA samples than bacterial culture, especially if the foal sampled is concurrently being treated with antimicrobial agents.
- PCR amplification may be done in association with, but should not replace, bacterial culture because it does not permit identification of concurrent bacterial pathogens and *in vitro* antimicrobial susceptibility testing.
- Other pathogens are often isolated along with *R. equi*.

## Serology

- Because of the need for less invasive and more practical means of diagnosis, several assays detecting antibody to *R. equi* have been developed and are commonly used by practicing veterinarians for the diagnosis of *R. equi* infections.
- The performance of four ELISA assays (ELISA-6939, ELISA-33701, ELISA-VapA, ELISA-California) and an agar gel immunodiffusion test for diagnosis of *R. equi* pneumonia in foals was evaluated at U of Florida.
    - Antibody concentrations of foals with culture-confirmed *R. equi* pneumonia (n=41) were compared to that of age-matched pasture mates that remained clinically healthy during the entire breeding season (n=24).
    - For each serological assay evaluated, selection of a low cut-off resulted in high sensitivity but low specificity.
    - Increasing the cut-off value resulted in better specificity but to the detriment of sensitivity.
    - They concluded that current serological assays **do not differentiate** between diseased and clinically healthy foals on farms with enzootic *R. equi* infections.
- In an independent study, serologic assays, whether performed on single or paired samples, also failed to reliably establish, confirm, or exclude a diagnosis of *R. equi* pneumonia in foals.
- Serologic testing is problematic because the widespread exposure of foals to this organism at a young age leads to antibody production without necessarily producing clinical disease.
- In addition, maternally-derived antibody may cause positive reactions in some serological assays which further confound the interpretation of the test.
- Summary
- Unfortunately *R. equi* antibodies are widespread even in healthy foals.

- These tests may be more helpful at the farm level to detect overall clinical infection with *R. equi* than for an individual foal.
- If you simply rely on serology to diagnose *R. equi* infections, you will over-diagnosis the disease and miss infections in the early stage.

**Clinical Laboratory**

- CBC is characterized by leukocytosis with a mature neutrophilia.
- Fibrinogen is increased consistently.
- Serum protein may be increased.
- Foals with diarrhea may have electrolyte abnormalities including hyponatremia and hypochloremia.

**Tracheobronchial aspirate(TBA): Culture**

- Still the only acceptable way to arrive at a definitive diagnosis of *R. equi* pneumonia
- Returns??- 62%, 64%, 100% of affected foals
- Recent study: Comparison of tracheal aspiration with other tests for diagnosis of *R. equi* pneumonia in foals
    - Pneumonia was induced by spraying of virulent *R. equi* into the trachea of foals.
    - Radiography, serodiagnosis and fecal culture were demonstrated to be valuable diagnostic methods, but to be limited compared with tracheal aspiration.
    - Indirect fluorescent antibody technique (IFA) using VapA Ab of *R. equi* and PCR targeting the structural gene of VapA detected bacteria in tracheal aspirates less sensitively than the isolation technique although they were more rapid.
    - Conclude that a combination of tracheal aspiration and bacterial isolation was the most valuable method for routine diagnosis of *R. equi* pneumonia in foals.
- Remember that on a farm with enzootic *R. equi* pneumonia 35% of 216 foals sampled had positive TBA culture and no signs of respiratory disease due to inhalation of contaminated dust.

**Tracheobronchial aspirate(TBA): Cytology**

- Cytology reveals gram positive to gram variable pleomorphic ("Chinese character") intracellular rods.
- Returns?? 61% of affected foals

**Blood culture for *R. equi***

??? Maybe in the future

## Imaging

### Ultrasonography

- Very useful in evaluating the severity of pneumonia and in assessing response to therapy especially for equine practitioners who do not have access to thoracic radiography. A sector scanner equipped with a 7.5 or 5.0 MHz transducer is preferred, but the linear scanner commonly used in equine reproduction can also be used.
- In *R. equi* infected foals, ultrasonographic evaluation of the chest often reveals extensive consolidation with well-defined abscesses.
- Ultrasonography is also a useful tool for detection of abdominal abscesses in some cases.

### Scintigraphic perfusion

- Shows major pulmonary perfusion defects characterized by a decrease or absence of radioisotope activity in affected areas of the lungs in foals.

### Radiography

- Useful to evaluate the severity and assess response to therapy.
- A prominent alveolar pattern characterized by ill-defined regional consolidation is the most common radiographic abnormality.
- The consolidated lesions are often seen as more discrete nodular and cavitary lesions compatible with pulmonary abscessation.
- Evidence of tracheobronchial lymphadenopathy characterized by nodular densities displacing the trachea dorsally.
- In foals presented with acute respiratory distress and having a prominent bronchointerstitial pattern on radiographic evaluation, the major differential diagnosis is sporadic severe bronchointerstitial pneumonia.
- Radiographs may be useful in monitoring response to therapy and determining the severity of pulmonary involvement.

### Treatment:

- The combination of erythromycin and rifampin has been the treatment of choice for *R. equi* infections in foals for more than ten years.
- Although combined therapy with erythromycin and rifampin has dramatically improved the survival rate of foals infected with *R. equi*, this treatment regimen is not without problems.
- In a recent study, 13% of *R. equi* isolates were found to be resistant to erythromycin.
- Treatment of foals infected with erythromycin-resistant isolates is problematic due to the lack of effective alternatives.
- Erythromycin
    - 25 mg/kg TID or QID or 37.5 mg/kg BID PO
    - Erythromycin can be stearate, estolate, phosphate or ethylsuccinate

- Azithromycin
    - 10 mg/kg SIB for 5 days than every other day
    - Lower incidence of diarrhea but not as effective for pneumonia as erythromycin or clarithromycin
- Clarithromycin
    - 7.5 mg/kg bid – most effective for pneumonia
- Rifampin –
    - 5.0 mg/kg BID or 10mg/kg SID PO
    - Rifampin should never be used alone due to the rapidity of development of resistance.

**Treatment points**

- Continue until CBC, fibrinogen and clinical presentation are normal. If possible, pneumonia should be resolved radiographically.
- If soft feces, usually self-limiting. Some foals may develop severe diarrhea. Decreasing the erythromycin dose may resolve the problem. If not, give IV erythromycin (5 mg/kg diluted QID).
- Idiosyncratic hyperthermia and tachypnea has been reported in foals receiving erythromycin. Use of the estolate form of the drug may decrease this adverse response. Placement of foal in a cool environment and antipyretic drugs work
- Watch for diarrhea in mare of foal if due to cophrophagy.
    - A fatal enteritis in mares to *R. equi* foals treated with erythromycin is a risk in Sweden
    - 2 articles: "*Clostridium difficile* associated with acute colitis in mares when their foals are treated with erythromycin and rifampicin for Rhodococcus equi
    - pneumonia", "The association of erythromycin ethylsuccinate with acute colitis induction in horses in Sweden"Assumption: ingested small amounts of erythromycin disturbs the gut flora of the mare
    - Erythromycin ethylsuccinate in mixture or tablets given orally is the most widely associated with this problem.
- Aminophylline should not be use in combination with erythromycin due to potential toxicity.

**Other therapies**

- Cool well ventilated environment
- Oxygen
- NSAIDs... use judiciously

**Prognosis**

- Pre-rifampin and erythromycin: 20% survivors
- Post-rifampin and erythromycin era: 80% survivors Standardbreds
  61% survivors Thoroughbreds
- Post infection may be less likely to get to the racetrack, but if they do they race as well

**Prevention**

- *R. equi* pneumonia is often not recognized until it is well advanced and, therefore, difficult to treat. Even severely affected foals may appear to suckle and behave normally to a casual observer.
- The diagnostic performance of WBC counts was significantly higher ($P = 0.03$) than that of fibrinogen concentrations.
- The sensitivity and specificity of WBC counts
- Using a cutoff of 13,000 cells/μl: 95.2% sensitivity and 61.2% specificity.
- Using a cutoff of 15,000 cells/μl, 78.6% sensitivity and 90.8% specificity.

**Decreasing the size of infective challenge**

- Enzootic farms more heavily infected with virulent (VapA positive) organisms than those where the disease is not present.
- Don't overcrowd foal paddocks
- Avoid loss of grass, allowing for dusty loafing areas.
- We have little information of what, if anything, might be practically done to decontaminate heavily infected paddocks. There is potentially fruitful work to be done in this area.

**Earlier recognition of the disease**

- Improve early detection by monitoring rectal temperature daily and plasma fibrinogen levels. Closely monitor those that have increases.
- Twice weekly complete physical examination with auscultation of the lungs
- Every 2 weeks, AGID testing from birth to 5 months

**Passive immunization**

- Immune plasma from adults given during the 1st month of life to foals on endemic farms
- Not 100% effective
- When: first 1-2 weeks of age and/or beginning of warm season
- Maybe should be first 1-2 weeks followed 3-4 weeks later
- Although it may not prevent *R. equi* colonization of the airway in all foals, its immune enhancement mechanism appears to prevent serious disease in virtually all foals if the plasma is given prior to exposure.
- Although plasma with high levels of specific antibody against *R. equi* has been used in these studies, it is not proven if it is the specific antibodies that are responsible for the efficacy or other non-specific components of plasma that may enhance opsonization.
- If it is a specific antibody, then the isotype of antibody responsible for protection must not be passed in colostrum since vaccination of mares in the U.S. has failed to protect foals against the disease.
- Efficacy of hyperimmune plasma might be the result of antibody to EHV2??
- Decrease likelihood of failure of passive transfer with good colostrum ingestion and evaluating immune status.

**Passive immunization**

- Intravenous administration of hyperimmune (HI) plasma obtained from horses vaccinated against *R. equi* using various antigens has consistently proved effective in significantly reducing the severity of *R. equi* pneumonia in foals following experimental challenge.
- However, results of studies evaluating the efficacy of various hyperimmune plasma preparations under field conditions have given contradictory results.
- This suggests that various factors such as the method of immunizing plasma donors, the amount of hyperimmune plasma used, the timing of hyperimmune plasma administration, management conditions, as well as the number of virulent bacteria in the environment may influence the effectiveness of a particular hyperimmune plasma product.
- Recent research suggests that antibodies to VapA and VapC proteins, present in most hyperimmune plasma preparations, are important in protection.
- Administration of HI plasma prior to infection with *R. equi* is essential.
- However, early administration may result in the decline of passively transferred antibody to a non-protective level at a time when foals are still susceptible to *R. equi* and environmental challenge is high.
- Because a recent study indicates that foals become infected early in life, intravenous administration of 1 L of HI plasma within 24-48h of birth is recommended, followed by a second administration at approximately 25 days of age.
- USDA licensed *R. equi* antibody (hyperimmune plasma) for the prevention of *R. equi* pneumonia is commercially available in North America (Lake Immunogenics Inc., Ontario, NY; Veterinary Dynamics, Templeton, CA).
- Hyperimmune plasma is expected to slightly decrease the incidence of the disease (by 30-40%) but will not prevent infection in all foals and should not lull farm owners into a false sense of security and reduce the need for continued vigilance.
- Whether this strategy is cost effective will vary from farm to farm.
- If used for the control of control of *R. equi* infections on enzootically infected farms, administration of hyperimmune plasma should always be combined with other control strategies, aimed at early identification and treatment of infected foals.

**Active immunization**

- No effective vaccine yet
- In Europe, EHV-2 may precede *R. equi* infection and vaccination for EHV-2 may prevent foals developing *R equi*.
- This study demonstrates that VapA may be an important antigen involved in humoral protective immunity in *R. equi* infections caused by foal virulent strains.

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# DIAGNOSIS AND TREATMENT OF DISORDERS OF THE AUDITORY TUBE DIVERTICULA (GUTTURAL POUCHES)

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## Introduction

A number of publications, mostly by Cook, during the late 1960s drew attention to diseases of the auditory tube diverticula (ATDs), notably mycosis. As a result of these, it became popular to diagnose ATD diseases practically whenever a horse was presented with a nasal discharge and yet, in reality, conditions arising within these structures are not common. Nevertheless, the location of the pouches and the nature of the vital structures which traverse them render the diseases of the ATDs important clinical entities.

## Anatomical Considerations

The ATDs are balloon-like structures lined by mucous membrane which lie between the base of the cranium and atlas dorsally and the pharynx ventrally. Rostrally they extend to abut onto the pharyngeal recess and caudally they lie ventral to the atlantal attachments of the longus colli muscle and the atlanto-occipital joint. As the term implies there are diverticulae of the pharyngo-tympanic tubes but their role has not yet been convincingly explained. Each balloon has an approximate volume of 300 ml and medially they are in contact with one another, divided only by a thin layer of areolar tissue, but in the dorsal part of the median septum they are separated by the paired rectus capitus ventralis muscles and their tendinous attachments to the basi-sphenoid bone. The stylo-hyoid bone intrudes into the caudal wall of each pouch and thus divides it into lateral and medial compartments. The vault of the pouch is in contact with the base of the skull so that those structures which enter and leave the cranium through the foramen lacerum must cross the pouch: internal carotid artery, cervical sympathetic nerve and cranial nerves IX (glosso-pharyngeal), X (vagus) and XI (accessory). The facial nerve (VII) lies in the submucosa of the lateral compartment dorsally. The internal maxillary artery and vein cross the wall of the lateral compartment antero-ventrally and just posterior to this the pouch lies beneath Viborg's triangle, the external landmark of the throat formed by the linguo-facial vein, the tendon of the sterno-mandibularis muscle and the caudal border of the mandible. The wing of the atlas marks the postero-dorsal limit of each pouch. Laterally, the ATDs are bordered by the pterygoideus and palatini muscles, the vertical ramus of the mandible and temporo-mandibular joint, the occipito-mandibular part of the digastricus muscle and the mandibular and parotid salivary glands.

The walls of the ATDs are composed of ciliated columnar epithelium with numerous mucous glands and lymph nodules. A dynamic mucociliary clearance system moves the normal mucus secreted by the lining of each ATD towards its drainage ostium which does not lie at the most ventral site. The drainage ostia are slit-like openings covered by cartilaginous flaps which lie on the dorso-lateral wall of the pharynx and they are quite close together, separated by the pharyngeal recess. Thus, although slight discharges from one pouch may produce a predominantly unilateral nasal discharge, the more copious the discharge, the more likely it is that it will be to some extent bilateral. The openings to the pouches lie on a level with the lateral canthus of the eye, and for the purposes of endoscopy or catheterisation they are best approached through the ipsilateral ventral nasal meatus. Running parallel with the ostia are the levator palatini muscles which contract to raise the palate during deglutition and so the ostia shorten and open during swallowing allowing a free movement of air between the lumen and the nasopharynx. This can be observed endoscopically when the cartilage flaps open and as they bow towards the midline they are almost in contact with one another.

There is no reason to suppose that the true pharyngo-tympanic tubes serve a different function in the horse to that which they fulfill in other species, i.e. to equilibrate pressure between the middle ear cavity and the external environment across the eardrum, and to provide drainage from the middle ear. However, the rationale for the ATDs is obscure. Amongst the suggestions on offer is to act as a source of pharyngeal mucus and to modify vocalisation and to warm inspired air. This latter proposal seems unlikely because, in the normal animal, reflex opening of the pharyngeal ostia and medial deviation of their cartilaginous flaps only occur at deglutition.

The **surgical approaches** used to gain access to the pouches are:

1. Hyovertebrotomy. An incision is made parallel and immediately cranial to the wing of the atlas. The parotid salivary gland is reflected forwards and the superficial branch of the second cervical nerve may be sectioned. The deeper loose connective tissue is separated and held open with retractors. An endoscope introduced into the ATD per nasum serves to illuminate the membranous lining at the surgical site once the loose connective tissue has been bluntly separated. The lumen is entered through the lateral wall of the medial compartment caudal to the stylo-hyoid bone. This approach is limited dorsally by the paramastoid process. In the foal the hyovertebrotomy route provides an adequate exposure for the creation of a medial septal fistula and for disruption of a defective ostium to treat cases of congenital tympany. The same approach may be used for the unilateral removal of aggregates of chondroids.

2. Viborg's Triangle. This was the traditional landmark for the drainage of ATD empyema but the indications for such a measure are practically non-existent nowadays because techniques for drainage through the natural ostia are superior. Access to the ATDs by this approach is very restricted although it has been recommended as the route of choice for medial fenestration surgery to relieve tympany. However, in this condition, stretching of the tissues which overlie the distended pouch increases the overall size of Viborg's triangle.

3. Paralaryngeal (Whitehouse) approach. With the horse in dorsal recumbency, a ventral midline incision is made over the larynx. The sterno-hyoid muscles are divided and the dissection continues lateral to the larynx, trachea and cricopharynx to reach the pouches ventro-medially. Entry to the ATD is made medial to the stylo-hyoid bone. However the depth of incision limits the value of this approach.

4. Modified Whitehouse approach. Although the surgery is again performed with the patient in dorsal recumbency, the site of the incision corresponds to that used for prosthetic laryngoplasty, i.e. it lies ventral to the linguo-facial vein and then follows the same route to enter the pouch. The Whitehouse approaches are advocated because they allow access to the roof of the ATD, the possibility to explore the lateral compartment digitally and access to both ATDs through the same incision.

## Diagnosis

External **palpation** in the parotid area is helpful to detect swellings produced by tympany, empyema, abscessation of adjacent lymph glands or neoplastic foci particularly in the parotid lymph glands. On occasions guttural pouch mycosis may produce painful foci deep to the base of the ear when the head of the stylo-hyoid bone or the tympano-hyoid articulation have become involved. Similarly the site may be painful to palpate in horses with tympano-hyoid osteitis.

The ATDs are normally filled by air and the contrast thus provided makes for good diagnostic radiographs. The **radiology** of the region is discussed elsewhere in these proceedings.

The simplest way to pass an **endoscope** into the pouch is by using a wire leader protruding from the biopsy channel. This channel is invariably eccentric and thus, the wire can be used to raise the cartilage flap before advancing into the duct beyond. However, this manoeuvre is invariably unsuccessful unless the ostium is approached via the ventral nasal meatus. In many horses both pouches can be entered by way of one nostril. Endoscopy should check for laryngeal and pharyngeal evidence of ATD disease as well as inspecting the inside of each pouch. Depression at the pharyngeal recess and partial obscuring of the larynx may result from distension within the ATD and an endoscopic assessment of potential respiratory obstruction is essential if a general anaesthetic is being considered for subsequent surgery. Pharyngeal and laryngeal hemiplegia can result from mycotic infections. Chronic diverticulitis is sometimes accompanied by stenosis/stricture of the ATD ostium which inhibits passage of the endoscope.

## Tympany of the ATD (Tympanitis)

This is a condition of foals which usually manifests itself within a few days of birth and it is thought to arise from a congenital malfunction of the pharyngeal opening of pouch. The disorder appears to be more common in fillies than colts and is almost invariably unilateral. In the United Kingdom Arabian horses are more susceptible than other breeds. In the condition the ostium acts as a non-return valve so that air can enter the pouch but cannot escape. It would seem that the obstruction to the escape of air is functional rather than physical because there is never any resistance to the passage of a catheter through the ostium to deflate the affected pouch. The result of the disorder is that air accumulates and produces a tympanitic swelling in the parotid region which is initially non-painful and not inflamed. Established cases invariably show evidence of opportunistic infection because a mucopurulent nasal discharge is generally present by the time afflicted foals are submitted for corrective surgery. On external examination the swelling may show an indentation where

the sternocephalicus tendon passes across it Also, the laxity of the medial septum may lead to swelling on the normal side, and hence it is sometimes tempting to conclude that the condition is bilateral. However, this possibility can only be confirmed if unilateral catheterisation of the worse side fails to relieve the swelling completely. Dysphagia and dyspnoea may be exhibited by virtue of the size of the distension. In addition, some features of ATD tympany suggest that the relationship between the opening of the ATD ostia and deglutition is more subtle, possibly because the condition results in part from a failure of the levator palatini muscle to shorten and open the ostium. Cases have been reported where afflicted foals have presented with nasal reflux of ingesta and where the competence of pharyngeal stripping waves has been compromised as well as the function of the ATD ostium. By the same token dysphagia may be a sequel to attempts at surgical relief regardless of the technique used and this cannot always be attributed to iatrogenic neuropathies. Occasionally ATD tympany is an acquired disorder of the adult horse.

**Treatment.** A simple conservative technique to remedy the disorder consisting of the long-term implantation of an in-dwelling mushroom-tipped catheter is usually successful. A 30 ml long, 8.7 mm diameter Foley catheter inflated with water is placed through the defective pharyngeal ostium per nasum and left in place for up to 8 weeks.

Only when the conservative method fails is it necessary to resort to surgery and the most effective method is the creation of a fistula between the normal and distended pouches by the removal of a section of the medial septum. The purpose of the medial wall fistulation technique is to facilitate the egress of air from the abnormal ATD through the pharyngeal ostium of the normal side. The author's preference for fistulation is by the hyovertebrotomy route but through the non-affected pouch. The logic for this technique is that the vital structures passing in the medial septum are more safely negotiated where the tissues were less stretched and distorted. The surgery is aided by the presence of an endoscope in the underside (diseased) pouch to provide trans-illumination of the field. Although afflicted foals may later become effective athletes a small number are rendered chronically dysphagic as mentioned above, and the owner should be warned of this.

Trans-endoscopic laser surgery provides an option for non-invasive fistulation through the medial septum when this facility is available. possibility before surgery is attempted.

## Diverticulitis

There are a group of poorly defined and poorly understood conditions which cause, or possibly result from, inflammation of the mucous membrane lining of the ATDs and which can loosely be termed 'diverticulitis'. These include strangles abscessation in the lymphoid tissue of the walls of the pouches, empyema, chondroid formation and chronic diverticulitis. A catarrhal inflammation of the ATD mucosae probably accompanies most upper respiratory tract bacterial and viral infections and the condition of tympany described above is also likely to be accompanied by inflammatory changes, hence the term tympanitis.

**Strangles:** This is an infectious condition of horses caused by **Streptococcus equi** which occurs world wide and which consists of a suppurative lymphadenitis particularly of the lymph glands associated with the upper respiratory tract including the ATDs. While primarily a disease of young equidae, horses of any age may become afflicted if they have no previous immunological experience of the infection. An initial stage of pyrexia is followed by inflammation of the pharyngeal and

ATD mucosae and their associated lymph nodes. The patient will be inappetant partly because of fever and partly because of pharyngeal discomfort. As the lymph nodes abscessate the dysphagia may become more severe with coughing and, in extreme cases, with the nasal return of food material. The head is often held in extension and there is generally soft tissue swelling dorsal to the larynx. Palpation of the region is resented and may provoke coughing. Occasionally the free movement of air through the drainage ostia of the ATDs is obstructed by the physical presence of the lymphadenopathy and a variable degree of tympany may be exhibited. In the earlier stages of the infection a bilateral serous nasal discharge will be present, which only becomes purulent when the abscesses burst into the lumen of the upper respiratory tract. If endoscopy were to be performed it would show soft tissue swelling of the postero-dorsal wall of the pharynx so that the larynx may be partly obscured. Introduction of the endoscope is difficult and resented but within the ATDs some landmarks may also be obscured by an irregular thickening of the pouch walls, especially caudally. However, endoscopy is not recommended as a routine for the investigation of suspected strangles cases because there is an obvious risk that the endoscope itself will act in the spread of the infection to other patients. Thus, scrupulous hygiene must be practised in the care of instruments used on horses which may be infected with this highly contagious disorder which is otherwise spread by the insufflation of discharges from infected animals. Pus released from a typical strangles abscess passes from a lymph node in the caudal wall of the ATD, into the pouch lumen, through the drainage ostium, into the nasopharynx and out via the nostril.

Once the abscesses have matured and ruptured, symptoms regress. Thus the onset of a purulent nasal discharge is accompanied by a reduction in the coughing, and eating is resumed. Treatment in these cases must always be directed towards encouraging the lymph node abscesses to burst with warm fomentation of the throat and steam inhalation. Supportive therapy by multivitamin injections plus nursing in a warm, well-ventilated box are indicated. Affected horses may find freshly cut grass easier to swallow than hard foods. Systemic antibiotic therapy should be avoided during the maturation stage of abscessation. Temporary tracheotomy is indicated for those horses which show life-threatening airway obstruction.

The primary aetiological factor in ATD **empyema** is a failure of mucociliary clearance followed by stagnation of mucus, opportunist bacterial infection and finally purulent exudation. Regardless of the precise aetiology of empyema, pus which is stagnant within the pouch eventually becomes inspissated and progressively leads to the formation of solid concretions **(chondroids).** In recent years it has been established that **Streptococcus equi** is the most common cause of chondroids and that the presence of these solid masses is a feature of the carrier state of the infection.

The clinical signs of empyema include a bilateral purulent nasal discharge and swelling of the parotid region. The distension of the affected pouch may produce obstructive dyspnoea and on endoscopy the larynx may again be partly obscured. The nasal discharge is sometimes malodorous. Lateral radiographs confirm the loss of air contrast from within the ATD, and if the pus is still fluid, an air/fluid interface will be demonstrable. Although leakage of pus from the ostia may be seen on endoscopy, it is usually difficult to introduce the endoscope because of the gross swelling. Care should be taken when attempting to drain or irrigate the ATDs with a catheter and this is best performed under general anaesthesia with a cuffed endotracheal tube in place. An indwelling self-retaining Foley balloon catheter may be used for prolonged irrigation in the management of chronic cases. Chondroids are either removed trans-endoscopically with a wire snare if the number is

small or by open surgery if there are many concretions present or if the pus is in a semi-solid state and cannot be flushed free.

A major consideration in the organisation of the surgical treatment of horses afflicted with restriction of the pharyngeal and laryngeal airways through ATD distension is the provision of a temporary alternative airway until the dyspnoea is relieved. Tracheotomy should be seriously considered before embarking onto general anaesthesia as intubation per os may be difficult after conventional induction. Plain lateral radiographs in the standing position can be helpful for anaesthetic decision-making.

**Chronic ATD diverticulitis** Chronic diverticulitis without the presence of empyema may come to light because of a syndrome of neuropathies where any combination of deficits of the glosso-pharyngeus, vagus, facial, spinal accessory and sympathetic nerves may be present. It is assumed that the nervous pathways are damaged as an extension of the inflammatory process through the ATD walls with which they are intimately associated. The diagnosis is established by a functional assessment of the cranial nerves mentioned combined with the endoscopic identification of a roughened thickening of the ATD lining. The nervous form of diverticulitis carries a poor prognosis.

## Trauma

The rectus capitis ventralis muscles insert onto the basi-sphenoid bones which form a narrow bridge between the foramina lacera on the ventral aspect of the skull in the dorso-medial aspect of the ATDs. Fractures at the junction of the basi-sphenoid and occipital bones may occur in horses which have sustained violent head trauma by rearing over backwards. Some patients with this injury are never able to stand again but show collapse and epistaxis. If such a patient can humanely be transported to an equine hospital, endoscopy can be used to confirm that the origin of the epistaxis lies in the ATDs and radiography can also make a valuable contribution to diagnosis. A step in the base of the skull will be visible on radiographs. Some horses do stand again and, with supportive management, make a full recovery .

## Guttural pouch mycosis (GPM)

The development of an invasive fungal plaque on the mucosal wall of the auditory tube diverticulum of a horse will produce consequences ranging from nil to fatal. Spontaneous epistaxis at rest is the most frequent sign noted by owners and this usually consists of a small quantity of fresh blood at one nostril in the first instance. A number of further minor haemorrhages may follow but, if untreated, exsanguination is a probable final outcome. It is unusual for the first episode of epistaxis to be fatal, but **the course of the disease from first to final haemorrhage rarely spans more than three weeks** and is more likely to be a matter of days. The reason why mycoses develop on the lining of the ATD is not known and the disease has not, so far, been reproduced experimentally. The fungal plaques of GPM are usually found in one of two characteristic sites; the great majority on the roof of the medial compartment and a small number of others on the lateral wall of the lateral compartment. Hence, there is a close association between these sites and the underlying internal carotid (ICA) and external carotid (ECA) arteries respectively. There is considerable variation in the relative frequency with which mycotic lesions have been found overlying these two major vessels. On the suspicion that the mycotic infection might be secondary to a primary arterial disease Colles and Cook undertook angiographic studies of the carotid circulation of normal horses and those afflicted with GPM. They confirmed aneurysmal dilatations in the majority of

diseased horses but did not comment on whether the remainder showed other radiographic changes such as filling defects or occlusion due to thrombosis. In a later study arterial abnormalities were identified in all 10 horses on which angiography was performed; eight horses had changes suggestive of an aneurysm of the ICA.

A definitive diagnosis of GPM is straightforward with modern flexible endoscopes, but two caveats should be heeded. First, the stress of handling the horse may precipitate a fatal epistaxis. Secondly, endoscopy may be unrewarding after a recent haemorrhage - visibility within the affected pouch may be poor because of the presence of a haematoma, and accurate location of the lesion may not be possible. However, it is generally possible to identify whether or not the ECA is healthy as it passes through the lateral compartment. Care is required because the endoscope may dislodge the haematoma, again provoking a severe haemorrhage. If the epistaxis has been recent, it may be sufficient to identify the stream of blood flowing from the pharyngeal drainage ostium. However, in all cases of mycosis, the contra-lateral pouch should be checked for extension of the disease through the medial septum and for concurrent bilateral mycosis. True bilateral infection is uncommon.

Medical treatment of GPM, be it by the use of topical or systemic antimycotic drugs or by a combination of both, provides poor results. In an early study Cook reported that in his series of 32 horses, eight of 14 which presented with epistaxis died or were destroyed as a result of haemorrhage when conservative therapy was employed. Three of 12 horses with nasal catarrh sustained a fatal epistaxis and only 13 of the 32 animals survived. The natural progress of many primary arterial disorders is towards thrombosis and this phenomenon explains instances where spontaneous recovery from GPM has been observed as well as the mechanism of successful medical treatments.

The concept of surgical occlusion of the branches of the carotid artery is not new and precedes the angiographic studies mentioned above. It has long been appreciated that the ICA is not an end-artery but placement of a distal ligature on the cerebral side of the lesion is difficult because the site is not easily accessible and it is often obscured by a mycotic plaque or haematoma. Although balloon catheter occlusion represents a more logical approach, the simplicity of the proximal ligation technique is not only appealing but there have been occasional reports of favourable results. The technique does not necessitate entry into the lumen of the ATD and consists of the placement of single ligatures at the origin of the ICA followed by topical and/or systemic antimycotic medication. Clearly this procedure includes a calculated gamble on two counts. First, no measure is taken to prevent retrograde haemorrhage from the Circle of Willis and, secondly, there is the assumption that the internal carotid artery is invariably the source of haemorrhage. The odds appear to be in favour of this because the site of mycosis has been on the ICA in at least 120 of the 134 cases reported in the literature over a 20 year period. Nevertheless an unacceptable number of horses die through exsanguination in the weeks following this surgery. Techniques have been devised to overcome this limitation comprising the ligation of the ICA proximally and occlusion of the vessel distal to the diseased segment with a balloon-tipped When catheters are used there is a divergence of opinion whether it is necessary to perform a second surgery to remove it after sufficient time has elapsed for thrombosis of the ICA to have occurred. Initially, surgical attention was directed to the ICA only, but it had already been shown that occlusion of other branches of the carotid circulation may be required in some cases of GPM. Rarely aneurysms of the ECA and pos-

terior auricular arteries do occur and with this in mind a modification of the balloon technique using venous thrombectomy catheters has been devised for the occlusion of branches of the ECA. A second catheter occludes the maxillary artery having been inserted through the greater palatine artery using an incision through the mucosa of the hard palate. Anomalous variations in the branches of the carotid tree are more difficult to identify, even at surgery, and, in the author's experience, are the most common cause of recurrent post-operative epistaxis often leading to a fatal outcome.

Most reports of the surgical management of GPM mention the use of topical antimycotic medication and yet the value of this component of the treatment regime is dubious. The objective of such medication is to accelerate resolution of the secondary mycotic infection once the diseased artery has been occluded or has become thrombosed, and yet the necessity to provide antimycotic agents to achieve this is not proven. It is conjectured that the surgical elimination of the substrate upon which the mycotic plaque thrives is sufficient.

Pharyngeal paralysis or, more accurately, hemiplegia, is the neuropathy which most frequent accompanies GPM and the inclusion of ingesta in a nasal discharge from any horse is an indication to inspect both ATDs by endoscopy as well as to assess pharyngeal function. Strangely, it is unusual to encounter a horse with GPM which concurrently shows epistaxis and neurological deficits. Endoscopic evidence of pharyngeal paralysis includes persistent dorsal displacement of the palatal arch, the presence of saliva and ingesta in the nasopharynx, weak pharyngeal contractions and a failure of one or both of the pharyngeal ostia of the ATDs to dilate during deglutition. Approximately a third of all cases of GPM recorded to date have been afflicted with pharyngeal paralysis or hemiplegia; this has generally been the most difficult aspect of the condition to manage. Recovery of competent pharyngeal function follows elimination of the mycosis in approximately a quarter of cases but with the period from the onset of dysphagia to the resolution of coughing and nasal reflux of ingesta ranging from three to 14 months. Horses with pharyngeal paralysis should be fed from ground level to reduce the risk of aspiration pneumonia and regular bronchoscopy and chest radiography should be undertaken to monitor any tendency to aspirate ingesta or to develop inhalation pneumonia. Obviously, vigorous supportive therapy to maintain the nutritional requirements of the afflicted animals is required and this may necessitate nasogastric or oesophagotomy intubation in isolated cases.

Laryngeal hemiplegia is the second most frequent cranial nerve deficit encountered in horses with GPM but it is rarely responsible for the only signs observed by the owner. It is not clear from the literature how frequently normal laryngeal function returns because in reports of horses with multiple signs including laryngeal hemiplegia, the authors tend to describe the overall condition as 'recovered' or 'resolved' without specifying whether the horses simply survived the life-threatening components of the disease, such as epistaxis and dysphagia, or whether each and every sign was resolved. Clearly a full endoscopic evaluation of the upper respiratory tract is indicated in cases of GPM so that the owner can be appraised of the significance of the findings before treatment commences. The presence of laryngeal hemiplegia may mitigate against a successful return to an athletic career without additional corrective surgery. Thus, unless the horse is valuable for breeding, treatment may not be economic.

already mentioned. These include other neuropathies such as facial palsy and Horner's syndrome; reluctance to lower the head to the ground and stiffness in the upper neck; parotid pain; otorrhoea; epiphora and photophobia. Abnormal head posture may be associated with pain in the atlanto-occipital joint when the mycosis extends directly to produce an infectious osteoarthritis. Thus, GPM should be included in the differential diagnosis of a wide range of disorders of the region and inspection of the lining of each ATD should be an integral part of the diagnostic procedure of each case. However, it should be noted that swelling of the parotid area is an unusual feature of GPM unless the pouch has become distended by blood from a recent major haemorrhage or by submucosal extravasation between the soft tissue fascial plains of the region.

Conclusions

In ideal circumstances, the objectives of the management of horses with suspected GPM should be:

- Urgent attention
- Accurate diagnosis to identify which branch(es) of the carotid artery is diseased by endoscopy and/or angiography.
- Full endoscopic assessment of secondary neuropathies.
- Secure occlusion of the diseased vessel proximal and distal to the lesion preferably by balloon catheter occlusion. (Simple proximal ligation of the ICA will provide a high level of success but, occasionally, horses will succumb to fatal haemorrhage, either by a retrograde flow via the Circle of Willis or when a vessel other than the ICA is damaged.)
- Elimination of the mycotic plaque by topical medication.
- Supportive therapy to allow resolution of associated neuropathies.

## Diseases of the middle ear, petrous temporal bone and hyoid apparatus

A syndrome has been reported which is thought to arise from chronic otitis media in the horse and it is thought that this leads to proliferative osteitis of the tympanic bulla which extends to the proximal stylo-hyoid bone. Ankylosis of the temporo-hyoid articulation follows but with the stress of repeated tongue movements fracture of the petrous temporal bone precipitates neurological deficits. Unilateral facial and vestibulo-cochlear dysfunction forms the most frequent presenting syndrome, although glosso-pharyngeal and vago-sympathetic deficits as well as seizures may occur in isolated cases. Endoscopy and radiography of the ATDs are indicated as part of the diagnostic assessment of suspected cases. When a diagnosis of temporo-hyoid osteitis is made before a pathological fracture of the stylo-hyoid occurs pre-emptive surgery – osteotomy of the stylo-hyoid is indicated to remove a short section of bone and to allow free movement of the hyoid apparatus as a whole.

## Neoplasia

All regions of the horse richly endowed with lymphoreticular tissue are potential sites for lymphosarcoma development and this tumour can arise in the tissues abutting onto the ATDs.

Ectopic melanosis is a common feature of the mucosa of the ATD of grey horses. The usual site for this is in the lateral compartment over the internal maxillary vessels. Primary melanomas can arise here and this is the site from which it is proposed that the familiar secondary parotid melanomas spread.

### Topical therapy of the ATDs

A 35 cm long 30 ml Foley balloon catheter offers a suitable indwelling device for repeated topical infusion of medication. The balloon also has the additional advantage that it provides a means to check that the catheter is correctly sited. The drainage ostium is the only structure in the equine head which will retain the inflated balloon. The catheter is passed up the ventral nasal meatus using a metal stiffener slightly bent at the leading end. This is rotated under the cartilage flap of the ostium which lies on the lateral pharyngeal wall at the same level as the eye. Long-term catheterisation may lead to weakening of the ostium and erosion of the cartilage flap. The repeated use of 10% iodide solutions produces severe inflammatory changes in the ATD mucosa and is not recommended as this has been shown to produce chronic hyperplastic changes in the mucosa.

# EQUINE INTERSTITIAL LUNG DISEASE

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## Introduction

Interstitial lung disease comprises an ill-defined group of pulmonary disorders that are chronic with insidious progression to pulmonary fibrosis (1). These diseases are characterized morphologically by a derangement of alveolar structure and a loss of functional gas exchange units. It may lead to life threatening respiratory distress due to hypoxemia which results from a progressive limitation of oxygen transfer from air to blood (2-4). The causes of interstitial lung disease are numerous and include infectious agents and toxins. There are probably two types of equine interstitial lung disease: one in foals and one in the adult horse. The form in foals appears as an acute respiratory distress syndrome in foals aged 1 to 8 months. Adult horses with the interstitial lung disease may have clinical signs resembling those of heaves. Most cases of interstitial lung disease in adult horses are of unknown causes. The diagnosis is based on history, clinical signs, thoracic radiographs, serology, and cytologic evaluation of tracheal bronchial fluid. A lung biopsy may provide a definitive diagnosis. Most often the cause of the interstitial lung disease is undetermined.

## Etiology

A variety of agents can be responsible for causing interstitial lung disease in animals. Fewer than 20 agents have been implicated in horses, the most common of which are infectious agents and ingested toxins (1-3). In horses specific syndromes have not been identified and the suggested classification has been made to differentiate interstitial lung disease in foals from that of adult horses (5). Viruses, immune complexes, infectious agents, and abnormalities of lung defense mechanisms have all been implicated in humans with pulmonary fibrosis (1). The difficulty in determining the causative agent is due to the fact that the lung responds in a single manner to most injuries.

Infectious agents are the most common known causes of interstitial lung disease in domesticated animals (2). The lung disease is acute and often progressive causing severe damage to the interalveolar space. Reported infectious causes of interstitial lung disease in horses include viral, bacterial, parasitic, protozoal, and fungal agents (1, 6-8). Viruses have been implicated as a cause of acute severe bronchointerstitial lung disease in many foals and adult horses (1, 6-8). In most of the reported cases of interstitial lung disease, despite the use of histology, serology, and viral isola-

tion, a viral agent was rarely confirmed. Many have speculated that there are unknown equine respiratory viruses responsible for this interstitial damage (6). The most commonly recognized respiratory viruses, including equine influenza virus, equine herpes virus Types I and IV, rhinovirus, equine viral arteritis, equine herpes virus Type II, and equine adenovirus during natural infection can cause mild clinical signs and well may be responsible for contributing to the development of secondary bacterial infection of the lower respiratory tract, but have rarely been implicated in interstitial lung disease. While bacterial infections of the lung most often result in a bronchopneumonia, bacterial agents have been isolated in horses with interstitial lung disease. Most likely these are opportunistic infections rather than the primary cause of the interstitial lung disease. It is suggested that bacteria in conjunction with another insult may induce interstitial lung disease. The protozoan *Pneumocystis carnii* has been isolated sporadically from foals with acute interstitial lung disease (6, 7, 9, 10). Its role as a primary agent for interstitial lung disease is unknown and it may well be a secondary invader of some other infectious lung disease. Parasitic infestation of the lung can cause an interstitial lung disease. This would include migration of ascarid larvae in foals and lungworms in adults.

Toxins either ingested or inhaled can be responsible for diffuse lung injury in horses. Plants are the most commonly ingested pneumotoxin. Perilla ketone derived from the plant *Perilla frutescens*, is a potent pneumotoxin in many animals and has been demonstrated to cause acute restrictive lung disease in horses (2, 11). Toxicity requires further metabolism of the 3-substituted furans by a mixed-function oxidase system which probably occurs in the lungs. Ponies present with respiratory distress within a week of ingesting the plant (22). Lesions include a diffuse alveolitis and Type II cell proliferation. This is unlike 3-methylindol which causes primarily an obstructive lung disease in horses. Chronic ingestion of Crofton weed, *Eupatorium adenophorum,* has caused severe chronic interstitial respiratory disease in horses in Australia and Hawaii (12). Horses initially cough and are exercise intolerant with gradual progression to respiratory distress and failure. Lesions include alveolitis, alveolar septal fibrosis, and epithelial cell proliferation.

Pyrrolizidine alkaloids (*Crotalaria* spp, *Senecio* spp.) cause pulmonary as well as hepatocellular injury, although the amount of alkaloid required to cause lung injury is much less than the dose which is hepatotoxic (13). The metabolically activated alkylating agent is produced in the liver and distributed to the lung via the blood stream primarily damaging capillary epithelial cells (1, 13).

Inhalation of pneumotoxic chemicals is thought to be an infrequent cause of diffuse lung injury in horses. Smoke inhalation causes acute, diffuse interstitial lung disease and respiratory distress in horses (14, 15). Prolonged inhalation of greater than 80% oxygen causes acute alveolitis which is a potential problem in neonatal foals. Such changes tend to occur in an already compromised lung after several days of oxygen therapy and are believed to be due to reactive oxygen species that damage macromolecules in some membranes (16). Agrichemicals or herbicides have the potential to cause acute interstitial lung disease in horses.

Silicosis: A chronic granulomatous lung disease has been associated with inhalation of forms of silican dioxide crystals by horses from the Carmel valley region in California (17). Silicone dioxides, which are inorganic dust, commonly found in the earth crust, are cytotoxic to macrophages as well as fibrogenic. Cristobalite is the most fibrogenic form of silican dioxide identified. Once inhaled the particles are ingested by macrophages causing lysis of the macrophage, persistent alveolitis, and

subsequent fibrosis. Multiple granulomas are present with submicron intra-cytoplasmic crystalline particles present in macrophages (17, 18). The disease in horses is similar to the chronic or accelerated form of silicosis in humans (18).

Hypersensitivity reactions: Hypersensitivity pneumonitis is a chronic, lymphocytic bronchointerstitial lung disease with granuloma formation and fibrosis (2, 3). It is caused by an inhalation of organic antigens such as microorganisms or animal tissues to which the animal has become sensitized (2, 3). In humans this disease includes farmer's lung and chicken breeder's syndrome. Hypersensitivity pneumonitis is rare in horses. Fungi in chicken dust were implicated in causing severe chronic bronchointerstitial lung disease in six horses (19-21).

Metabolic and toxic conditions have been associated with acute pulmonary damage in humans and animals. The pulmonary lesions are associated with endotoxemia (3, 11).

Idiopathic: Most cases of interstitial lung disease in horses remain undiagnosed. There may be a different pathologic process for the interstitial lung disease in foals versus adult horses.

## Pathogenesis

The pathogenesis of interstitial lung disease involves progression through four phases (3, 4, 6, 22). In phase I, an initial insult causes injury to parenchymal cells as well as to acute alveolitis. Phase II is a proliferative phase with cellular and connective tissue changes involving lung parenchyma. In cases of chronic infection, there is progression to phase III, which is a development of irreversible interstitial fibrosis. Phase IV is the end stage, irreparable fibrosis of the lung (1).

All of these structural changes in the lung that occur with interstitial lung disease reduce the number of functional alveoli, thus altering pulmonary function (1). There is reduced lung compliance due to reduction in total volume of the lungs and increased transpulmonary pressure at total lung capacity (22). Pulmonary gas exchange is impaired and this is primarily due to mal-distribution of ventilation and profusion (23). Total and vital lung capacity is decreased due to a loss in gas exchange units and the change in the elastic properties of the lung (11). Stiff fibrotic lungs increase the work of breathing, resulting in exercise intolerance and dyspnea. Pulmonary hypertension and cor pulmonale are occasional sequela to interstitial lung disease in other species. It should be noted that these changes in the lung result in restrictive pulmonary disease in comparison to obstructive disease which occurs with primary airway pathology.

## Diagnosis

Horses with interstitial lung disease may present with an acute to chronic history of weight loss, recurrent cough, nasal discharge, fever and respiratory distress Chronically affected adult horses may be bright and alert, despite dyspnea, and have a variable appetite (18, 19, 23, 24). The lung disease is generally unresponsive to antimicrobial and anti-inflammatory therapy in both adult and foals. Physical examination findings include tachypnea, tachycardia, and variable fever. Mucous membranes may be cyanotic in severe cases. There is a rapid, shallow respiratory pattern with occasional respiratory dyspnea. Diffuse crackles and wheeze may be heard on auscultation of the lung or there may be an absence of lung sounds in the presence of severe pulmonary edema. Hematologic findings may include neutrophilic leukocytosis and hyperfibrinogenemia. Peripheral

eosinophilia may accompany hypersensitivity or parasitic pneumonitis. Arterial blood gas analysis reveals a spectrum of disorders, but hypoxemia is usually present.

Thoracic radiographs are useful to determine the presence of pulmonary disease and to monitor its course. The most common radiographic finding is severe, diffuse, nodular interstitial disease (5, 7, 18, 24). Pulmonary infiltrates were often present with fungal or parasitic pneumonia. Radiographs are not a specific or sensitive method to monitor alveolitis because the degree of radiographic changes to the lung does not correlate well with the severity of the disease.

Culture of tracheal wash or bronchoalveolar lavage fluid in horse with interstitial lung disease often yields no significant growth of bacteria or fungal pathogens (5, 7). Ultrasonographic findings of the thorax may demonstrate changes suggestive of severe fibrosis if a sub-pleural distribution exists (24). A trans-thoracic lung biopsy is the definitive test for diagnosing chronic interstitial lung disease (1). A representative sample can be obtained when the lung disease is diffuse or when a lesion can be identified ultrasonographically. Histopathological evaluation of the biopsy during the acute phase of the disease may demonstrate coagulation necrosis of alveolar walls, hyaline membrane formation and focal hemorrhage and edema (5, 7, 25). In chronic cases the biopsy may show evidence of severe fibrosing alveolitis with minimal airway involvement (5, 8, 23). The interlobular septa may be thickened, alveolar replaced with collagen and reticulum fibers and there may be evidence of Type II cell hypoplasia. Multi-focal granulomas and bronchiolitis may also be seen. Except in cases of silicosis and pneumocystis infections, biopsy really defines the cause of the disease (17, 18).

## Treatment

Corticosteroids have been used to treat interstitial lung disease in horses (5, 7-9, 24). The goal of therapy is to limit inflammation and to prevent further fibrosis. In the acute phase of interstitial lung disease antimicrobial drugs are also recommended to prevent secondary bacterial infections. While bronchodilating agents have been used in horses with interstitial lung disease their effectiveness is limited to the fact that this is a restrictive and not an obstructive condition. While corticosteroids have been recommended as a course for treatment there is no evidence to suggest that these drugs or any other anti-inflammatory agents effectively suppress the alveolitis which occurs. Preventive measures are difficult in as much as the inciting cause is rarely known.

In summary interstitial lung disease in horses is a recognized clinical entity. The condition has been recognized in Europe and in the United States and has evaded detection as to its cause(s) (5).

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# CURRENT TRENDS IN THE DIAGNOSIS AND TREATMENT OF EQUINE RECURRENT LARYNGEAL NEUROPATHY

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## Introduction

There can have been few conditions in the entire veterinary diagnostic and surgical repertoire which have received more attention than recurrent laryngeal neuropathy (RLN) of the horse and therefore it is beyond the scope of this presentation to provide a comprehensive review of all aspects of current knowledge on the subject. The objective will be to outline how techniques incorporating exercise testing have improved diagnostic specificity for RLN and have brought into question the tendency to over-reliance on endoscopy during resting breathing. Spiers in 1987 made the observation that no new concept for the treatment of RLN had been introduced throughout the 20th century - even prosthetic laryngoplasty and nerve transplantation had been suggested in the late 19th century - and there are no significant advances to report during the past 16 years.

**Definition:** Recurrent laryngeal neuropathy is the cause of a permanent dysfunction of those intrinsic muscles of the larynx which receive their motor innervation through the recurrent laryngeal branch of the vagus nerve and which leads to partial obstruction of the airway, especially during exercise. Thereby, it is likely that the performance potential of affected horses will be compromised. The condition almost invariably involves the left side of the larynx and very rarely right-sided or bilateral cases are encountered. The disease manifests itself as a failure to achieve or to maintain full symmetrical arytenoid abduction, especially under conditions of greatest respiratory demand. The resistance to normal airflow provoked by an inadequately abducted arytenoid cartilage causes turbulence in the airstream which is the source of a characteristic abnormal inspiratory sound when the horse is exerted.

RLN has been recognised as a cause of adventitious respiratory noise in the exercising horse since classical times and, on account of the sounds produced, the disorder has generally been referred to as 'roaring' or 'whistling' in the English-speaking world. In continental Europe the terms 'cornage', 'siffler', 'kehlkopfpfeifen' and 'pfeiferdampf' convey the same message. In more recent times the condition has also been termed idiopathic laryngeal hemiplegia (ILH) but this term is not satisfactory for reasons given below.

The objectives of corrective surgery for RLN should be to prevent the dynamic collapse of the arytenoid cartilage into the airway and to eliminate the obstructive effect of the disorder. Estimations of the prevalence of RLN suggest that approximately 2% of Thoroughbred horses are afflicted with dynamic collapse of the left arytenoid cartilage during vigorous exercise and a similar incidence is likely in other larger breeds of horse. A further 20% of Thoroughbreds show anomalous laryngeal motility during quiet breathing and highspeed treadmill endoscopy (HSTE) helps to discriminate between the majority of these which subsequently show normal full symmetrical abduction of the larynx at exercise and the minority which sustain dynamic airway obstruction and require surgery. It also helps to identify those horses where the vocal cords collapse without obstruction by the arytenoid cartilages themselves and such patients would not benefit from prosthetic laryngoplasty.

**Pathophysiology.** During exercise the arytenoid cartilages are normally held in full symmetrical abduction which is sustained throughout all stages of the respiratory cycle and this abduction will be present for a period after completion of the exercise dependent on the duration and degree of the exertion imposed, and the fitness of the horse. Failure of the neuromuscular unit which abducts the left arytenoid cartilage results in an inability to maintain full abduction of the rima glottidis and the result is partial obstruction of the airway. Video-endoscopic studies of horses with RLN galloping on highspeed treadmills have revealed progressive dynamic collapse of the vocal fold and arytenoid cartilage, sometimes to the point where they cross the midline and contact the abducted cartilage. Sound generally results initially from air passing across the entrance to the open ventricle of the affected side, producing a whistle sound by resonance within the ventricle. As the cross-sectional area of the rima glottidis is reduced the pressure differentials within the larynx must increase to maintain the airflow necessary to sustain exercise. The collapsing forces rise and the paralysed cord is drawn even further across the airway (Venturi effect) producing the harsh roaring noise which typifies the condition of hemiplegia. As the demands for inspiratory flow reach their peak, afflicted horses show flow limitation, increased inspiratory resistance and depression of arterial oxygen tension. Finally complete dynamic collapse of the left arytenoid occurs which will have a devastating effect on respiratory function. The horse is obliged to reduce speed to reduce demand and the paralysed arytenoid gradually falls back to its neutral position.

## Diagnosis

### Presentation.

The circumstances in which veterinary assessments of laryngeal function may be required are as follows:

1. At an examination for a prospective purchaser. This examination should include examinations at rest and at exercise. An unusual respiratory noise, poor exercise tolerance or the findings of laryngeal palpation may suggest a diagnosis of RLN, in which case it is essential that the examining veterinarian points out the significance of these findings and recommends further investigations including endoscopy to clarify the situation. Failure to identify and to report evidence of previous laryngeal surgery during a pre-purchase examination may lead to malpractice litigation.

2. An owner/trainer has noticed an unusual respiratory noise at exercise. Horses afflicted with RLN usually produce consistent inspiratory sounds which can be heard throughout the period of exertion at the canter and gallop. Disappearance of the sounds can be expected with-

in a short period of pulling up. Occasionally horses with RLN produce adventitious respiratory noises only under extreme exertion and, unless examined by endoscopy during high-speed treadmill exercise, it is not possible to determine whether the noise stems from abrupt dynamic collapse of the paralysed arytenoid cartilage or from secondary DDSP.

3. The athletic performance of the horse is disappointing. It is important to distinguish between 'loss of performance' and 'an inherent inability ever to perform well'. If RLN is sufficiently severe to be responsible for diminished performance, there will invariably be adventitious respiratory noises at exercise, although the rider or owner may not have observed these.

4. At the examination of horses which "gurgle" or "choke up" at fast work. Some of these horses are primarily cases of RLN which show secondary dorsal dislocation of the soft palate.

5. A horse which shows changed vocalisation. Horses which have previously been subjected to surgery such as ventriculectomy and/or prosthetic laryngoplasty for RLN usually show voice changes.

**Diagnostic procedures:** The static data relating to the breed, age and conformation of the patient may provide useful pointers to the possibility of RLN. Provided that the information is available, the case history should take note of the age of onset of clinical signs, the nature of abnormal respiratory sounds at exercise, details of previous respiratory infections and performance data before and after the onset of signs.

**Palpation** of the larynx should look for:

(1) A **complete cartilage skeleton**: the spacing between the cricoid and thyroid cartilages becomes widened when there are deformities of the thyroid laminae. These are usually present as part of the fourth branchial arch defect syndrome and particular vigilance is required in cases of apparent right-sided RLN.

(2) **Atrophy of the intrinsic laryngeal musculature** - more likely on the left side

(3) Strength of the **'slap' response** is, in the author's hands, more accurately judged by palpation than by endoscopy

(4) The **arytenoid depression test**, whereby the right side of the larynx is forced into adduction by pressure on the right arytenoid at the muscular process, is more likely to provide a convincing increase in stridor at the conclusion of exercise than at rest.

(5) **Evidence of previous surgery** - a cicatrix from an earlier ventral laryngofissure: the most reliable technique to find such scarring is to roll the skin over the crico-thyroid membrane between the first finger and thumb of both hands.

(6) The area ventral to the left linguo-facial vein should be checked for a **laryngoplasty scar**.

(7) The **patency of the jugular veins** should be checked for evidence of previous perivascular injection reactions which may impact on laryngeal function.

**Laryngoscopy** should be performed at rest during quiet breathing but, although frequently recommended, the value of endoscopy immediately after exercise is very questionable.

The asymmetry of the rima glottidis in cases of true left laryngeal hemiplegia is usually obvious, although the perspective distortion which arises from the eccentric position of the endoscope in the nasopharynx must be taken into account. Whenever doubt exists, the endoscopy should be performed through each nostril in turn. Most equine endoscopists have evolved a grading system to quantify laryngeal motility during quiet breathing and while these may be unique for each and every one of us the fundamental requirement should be one of repeatability. The system used by the author for many years comprises a modification of the scheme proposed by Ducharme, Hackett et al (1991) and is as follows:

**Grade 1**. Overall symmetry when even the smaller movements by the vocal folds are synchronised. Perspective artefacts cancelled when viewed via contra-lateral nostril - 'mirror' effect.

**Grade 2.** All major movements are symmetrical and a full range is achieved. Transient asynchrony, flutter or delayed/biphasic abduction.

**Grade 3.** Asymmetry of the rima glottidis at rest due to reduced ('lazy') motility by the left arytenoid cartilage and vocal fold but can achieve full symmetrical abduction, for example in response to the nostril closure manoeuvre or after swallowing.

**Grade 4.** Consistent asymmetry but with some residual movement. However, full abduction never observed

**Grade 5.** Complete absence of abductory and adductory movement on the affected side.

In general terms horses with Grades 1 and 2 motility prove to be normal and produce no untoward respiratory sounds at exercise. It is very unusual for horses showing such motility during quiet breathing to show dynamic left-sided collapse during HSTE but in these exceptional cases the patient will produce characteristic inspiratory noises and have palpable atrophy of the intrinsic musculature. Grade 3 motility is equivocal because treadmill studies have shown that at least 70% of horses with this grading at rest show full symmetrical abduction during exercise. Grades 4 and 5 almost always represent clinical disease and afflicted horses produce abnormal inspiratory noises when exerted..

Video-endoscopic recordings were made of the upper respiratory tracts of 3497 yearlings during a 15 year study and the findings have been reviewed. Although the subjects represent an elite group of horses on the basis of genetic selection, unlike previous investigations (Pascoe, et al, 1981; Raphael, 1982; Baker, 1983; Lane et al, 1987; Sweeney, et al, 1991), these were unbroken and therefore untried animals. Initially approximately half of the yearlings had been purchased, some privately and some at auction, and half were homebred. In recent years the majority of the yearlings examined have been homebred and yet the distribution of the findings has remained constant. The findings in regard to the distribution of gradings was as follows:

| RLN Grading | 1 | 2 | 3 | 4 | 5 | Not Graded |
|---|---|---|---|---|---|---|
| **Total: 3497** | 784 | 2006 | 617 | 69 | 9 | 12 |
| | 22.4% | 57.4% | 17.6% | 2.0% | 0.26% | 0.34% |

The twelve yearlings for which no grade was assigned included seven animals afflicted with the fourth branchial arch defect syndrome and an additional five where there was mal-function on the right side and where no physical explanation was discovered

The prevalence of clinically significant RLN, i.e. Grades 4 and 5, was found to be 2.26% and this can be regarded as a base level for an unselected population of Thoroughbred horses. It compares with previous reports of selected groups:

| Authors | Group Size | Population | RLN Incidence (sic. Grades 4 and 5) |
|---|---|---|---|
| Pascoe et al (1981) | 235 | Horses in training | 2.6% |
| Raphael (1982) | 479 | " | 3.3% |
| Baker (1983) | 537 | " | 4.7% |
| Lane et al (1987) | 6860 | Yearlings at sale | 0.96% |
| Sweeney et al (1991) | 678 | Horses in training | 1.8% |

Endoscopy also has a role to play in the investigation of horses with RLN after surgery. Whatever the limitations of the ventriculectomy procedure, endoscopic inspection of the ventricular openings provides a dependable means to establish whether a horse has previously been the subject of this surgery. Endoscopy is helpful to confirm the degree of abduction which has been achieved by abductor prosthesis implantation, to check whether it is maintained in the long-term and to identify signs of complications such as dysphagia or chondroma formation.

Other laryngeal disorders which should be considered in the endoscopic investigation of stridor at exercise and which may be identified during quiet breathing include:

(1) Epiglottal entrapment
(2) Sub-epiglottic cyst
(3) Fourth branchial arch defects (4-BAD)
(4) Arytenoid chondropathy

When facilities for treadmill endoscopy are not available a ridden exercise test is the best practical alternative. This should consist first of a collected canter in small circles around the examiner, followed by an extended canter on both reins and finally a full gallop within the limits imposed by the conditions underfoot, the size of the exercise area, the fitness of the horse and the competence of the rider. Normal horses make no detectable inspiratory noises but expiratory noises may be normal, e.g., those which result from turbulence at the false nostril. At the canter and gallop, the horse performs one complete respiratory cycle per stride and expiration occurs as the forefeet meet the ground and the rider moves forward in the saddle. This synchronisation allows accurate timing of abnormal noises in the respiratory cycle which is not possible at the walk and trot. Horses which are unfit or which have upper airway obstructions may attempt to adapt by taking one breath for every two strides – this slows down the flow of air and reduces dynamic collapse during light exercise. Horses with RLN typically produce an abnormal inspiratory sound ranging from a musical whistle to a harsh sawing roar. Severely afflicted horses may show premature exhaustion with difficulty in co-ordinating the locomotion and respiratory cycles. On pulling up, palpation of the larynx may reveal fremitus and this is the best time to employ the arytenoid depression test.

## Treatment

A number of procedures has been proposed to relieve the obstructive effects of RLN : the most notable of these are the various ventriculectomy and cordectomy procedures; prosthetic laryngoplasty; nerve/muscle pedicle grafting; temporary tracheotomy and permanent tracheostomy; and subtotal arytenoidectomy.

### Ventriculectomy (Hobday or Williams procedure)

Ventriculectomy was first performed in Germany in the mid-19th century by Günther Junior but in the English-speaking world it is generally known as the Williams or Hobday operation. Recent reports have brought into question the efficacy of the operation and yet the procedure has been performed on hundreds of thousands of horses world-wide during the past 100 years and owners and trainers frequently request that their horses are 'Hobdayed'. The damning evidence against the Hobday operation is derived from studies of airflow mechanics on horses with experimentally induced Grade 5 RLN then treated by left ventriculectomy alone. It is not yet known to what degree horses with Grades 3 and 4 RLN are physiologically compromised let alone whether ventriculectomy with or without cordectomy is in any way helpful. However, the benefits of the Hobday procedure are at best slight, and the technique should be reserved for horses with Grade 3 and possibly 4 RLN only, or for those which show dynamic collapse by the vocal cords alone during HSTE. Recent studies of the sounds produced by horses with RLN have shown that there is a marked reduction in noise after ventriculo-cordectomy when this is performed alone or in combination with prosthetic laryngoplasty.

**Technique**: For ventriculo-cordectomy the laryngeal lumen is accessed via the crico-thyroid ligament. Removal of the ventricle is achieved by introducing a burr which is rotated until it picks up the mucosa. The handle of the burr is eased to-and-fro like an aircraft joy-stick until the mucous membrane can be completely everted. It is then grasped by two pairs of clamps which are used to lever out any residual mucosa before the intact ventricle is excised with scissors Although there is little physiological rationale for removing the right ventricle many surgeons perform the technique bilaterally and the justification for this is based upon HSTE which frequently shows collapse of both cords towards the midline. The vocal cord is removed by section of its attachment to the

body of the thyroid and stripping it dorso-caudally towards the vocal process of the arytenoid where the excision is completed. A 30 day period of box rest is prescribed before normal exercise is resumed.

**Abductor Prosthesis Operation (Prosthetic laryngoplasty, "Tie-back")**

An alternative approach to the management of RLN was resurrected by Marks and Mackay-Smith in 1970 and over thirty years later prosthetic laryngoplasty (PL) remains the treatment of choice in most countries. The procedure and its variants aim to implant a ligature which mimics the action of the CAD muscle as if it were in a semi-contracted state. However, the procedure should be regarded as a gross physiological disturbance because when the rima glottidis is fixed in an abducted position, the ability of the larynx to protect the lower airways during deglutition is compromised and a degree of dysphagia is inevitable. Nevertheless, most horses subjected to PL show relief of laryngeal obstruction and are not clinically dysphagic. Studies of airway mechanics in horses exercising on the treadmill have confirmed that PL is effective in the restoration of normal respiratory function and in the prevention of dynamic collapse of the paralysed arytenoid. Although such studies imply 100% success in terms of respiratory physiological function, they take no account of the effect of PL on deglutition or of recurrence of laryngeal obstruction. Therefore, the overall "success" rate must be adjusted to include failures through a variety of complications. The reasons offered for the cough and dysphagia include excessive abduction by an over-tight prosthesis but other reports have suggested that dysphagia can occur without hyper-abduction. It has been suggested that pharyngeal nerve or muscle injury, or neuromuscular inco-ordination may be responsible. Dysphagia has been induced experimentally in 'sham' operations when no implant was used, implying that the insult of surgery was the important factor. In order to eliminate the tissue insult associated with separation and retraction of the crico- and thyro-pharyngeus muscles, the caudal approach to the muscular process provides good visibility and good access with little trauma.

Apart from persistent coughing the next most frequent cause of failure of the PL procedure is recurrence of collapse of the left arytenoid cartilage. There are three possible explanations for such failures: (1) breakdown of the implant, (2) avulsion at the cricoid cartilage attachment and (3) avulsion at the attachment through the muscular process. The view that avulsion from the muscular process is the usual cause of failure has been supported by experimental data and yet it seems illogical that most of the discussion relating to the siting of the implant(s) has focused on the cricoid attachment and the use of multiple prostheses.

The choice of material for the prosthesis has also been the subject of debate. It is generally agreed that the elastic properties of lycra, which was originally recommended, are not necessary and this material has been incriminated as a likely cause of wound dehiscence, the formation of sinus tracts and delayed healing. However, in the author's experience such complications are much more common when other multifilament non-absorbable materials such as Mersilene and braided nylon have been used. Some authors advocate the use of absorbable implants such as heavy gauge chromic catgut but no results of the use of this material have been published. For the past fifteen years the author has followed the recommendation above to use the least reactive materials available and has used a single prosthesis of 4 metric monofilament nylon in a double strand. Over 2000 procedures have been performed with this implant and the complication rate has been reduced, but it is conceded that the frequency of recurrence of collapse remains unknown. A monofilament steel laryngeal prosthesis is favoured by some surgeons.

In summary, PL is far from ideal as a treatment for RLN but it remains the best option currently available. Refinements are required to provide consistent and enduring abduction without dysphagia. Although it is recognised that this surgery can produce complications in the forms of coughing, nasal reflux of ingesta or recurrence of dyspnoea, the risks are justifiable in horses which cannot otherwise be effective athletes.

**Technique**: The patient is provided with non-steroidal anti-inflammatory and antibiotic medication from 24 hours ahead of surgery. Strict aseptic precautions should be observed for the operation. The patient is placed in right lateral recumbency with the neck extended. The skin incision is made ventral to the linguo-facial vein from the level of the vertical ramus of the mandible extending 10 cm caudally where it is limited at the musculo-tendinous junction of the sterno-mandibularis muscle. The fascial plane between the linguo-facial vein and the omo-hyoideus muscle is identified and divided. Retraction of the soft tissues dorsally permits palpation of the caudal border of the cricoid cartilage. A wide bladed retractor also helps to protect the oesophagus and carotid artery. The dorsal spine and midline indentation of the cricoid cartilage are identified by digital palpation. The prosthesis is introduced from the caudal border of the cricoid 1 cm lateral to the midline. Traction on towel clips applied to the caudal margin of the cricoid helps to achieve a near-midline placement. The prosthesis itself consists of a double strand of 0 gauge monofilament nylon swaged onto a trochar needle.

The muscular process of the arytenoid cartilage can be skylined by displacing the crico- and thyropharyngeus muscles rostrally using the index finger of the left hand. With this still in place a hole is tunnelled through the muscular process of the arytenoid using a 14 SWG needle at 90º to the line of the CAD muscle fibres and the cartilage plug is dispelled with a longer, narrower needle. The cartilage plug should be identified and retrieved. A 27 SWG wire loop is used to withdraw the medial end of the prosthesis is retrieved through the tunnel before the two ends of the prosthesis are tied. Tension in the prosthesis is assessed by endoscopy with the endotracheal tube withdrawn. Monocryl is preferred for routine layer-by-layer closure of the soft tissues and a Penrose drain is rarely required in the dead space. Ventriculo-cordectomy may be performed on those animals which have not previously been Hobdayed (see above). A five day course of prophylactic penicillin is provided and strict box rest is prescribed for 35 days. A normal training programme can be resumed at that stage but an endoscopic reassessment of the laryngeal abduction should be performed before fast exercise begins. Food and water are provided from ground level indefinitely.

### Nerve/muscle pedicle grafting

During the 1990's the possibility to restore motor function to the abductor musculature of the larynx was re-explored. Nerve/muscle pedicle grafting aims to transplant cubes of muscle taken from the omo-hyoideus together with their motor supply through the first and second cervical nerves into the atrophied CAD muscle in order to restore abductory function to the larynx. Following surgery the grafts grow in response to mechanical stimulation so that at least a year must be allowed to achieve optimum results. Abduction of the arytenoid cartilage occurs only during exertion because the omo-hyoid is an accessory muscle of respiration and the technique has the advantage over prosthetic laryngoplasty that no complications can arise from aspiration through a permanently abducted rima glottidis.

The surgical approach is similar to that of prosthetic laryngoplasty, i.e. immediately ventral to the linguo-facial vein, and dorsal to the omo-hyoideus muscle. The branches of the cervical nerves enter the muscle from the dorsal margin, slightly medially and tend to run rostrally in the muscle fascia before entering the muscular tissue itself. These are meticulously followed so that 2 - 4 grafts measuring 3 mm. cubed can be isolated with their nerve supply intact. The grafts are placed into the CAD muscle which is exposed by displacing the crico-pharyngeus muscle forwards. Splits are created in the CAD muscle parallel with the fibre alignment and each graft is secured with absorbable sutures in the CAD fascia.

Controlled exercise can commence after 3 weeks and is continued over the following 8 months before serious training resumes. At least 12 months should be allowed between the time of surgery and a first race and therefore this technique must be reserved for horses with patient owners and where the athletic career can withstand a prolonged recovery. Clearly, the earlier a definitive diagnosis can be made the more likely it is that nerve/muscle pedicle grafting will be the technique of choice.

**Temporary tracheotomy intubation and permanent tracheostomy**

In some countries including the UK and Ireland it is still permitted to race horses under rules with a self-retaining tracheotomy tube in place. The purpose of these tubes is to provide an alternative airway and to by-pass the site of airway obstruction. They may be used when other surgical techniques have failed but their major virtue is that intubation is performed under local analgesia and disruption of the training programme is minimal.

Although permanent tracheostomy, the creation of a fistula between the tracheal lumen and the skin surface of the ventral neck, has been described as a feasible procedure for horses and ponies the results are generally not acceptable to the owners and trainers of animals used for racing. In contrast to a tracheotomy tube which is normally plugged at all times other than when the horse is at exercise, the hole of a tracheostomy leaves the airway unprotected and the effectiveness of nasal and lower airway clearance mechanisms is reduced. Thus, there is a regular requirement for nursing to remove exudation from the skin adjacent to the stoma and to maintain local hygiene.

**Total, partial and sub-total arytenoidectomy**

In historical terms the wheel has almost turned full circle because partial arytenoidectomy was first proposed by Günther and Günther in 1845 and it was revived towards the end of the 20th century as a means to relieve laryngeal airway obstructions. The objective is to remove from the glottic airway those structures which are causing obstruction Thus, the usual indications for arytenoidectomy are the removal of infected cartilage in cases of chronic chondropathy and the removal of the left arytenoid cartilage when other techniques to treat RLN have failed. However, a recent study showed that it is preferable to repeat PL surgery when collapse of the arytenoid has returned than to opt for arytenoidectomy.

Total arytenoidectomy implies that the entire arytenoid cartilage together with the corniculate process is removed. In partial arytenoidectomy the muscular process and articular facet are left in situ and in sub-total arytenoidectomy part or all of the corniculate process is also left in place. The results of the various forms of arytenoidectomy suggest that these procedures are of limited

value in the restoration of horses to their full athletic potential and that their place is in the salvage of horses for breeding or for more sedentary pastimes.

**Technique:** Anaesthesia is maintained by direct intubation into the trachea; a tracheotomy incision is made between adjacent cartilage rings or by the removal of semi-circular cartilage discs from adjacent rings. The patient is placed in dorsal recumbency and the larynx is entered through the crico-thyroid ligament before the body of the thyroid cartilage is split along the ventral midline. It is not advisable to split the cricoid cartilage. The ventricle and cord on the affected side are removed with a burr in the conventional manner. A sub-mucosal resection of the arytenoid cartilage is made leaving a 2 mm rim at the corniculate process and the muscular attachments to the cartilage are divided as close to the arytenoid as possible. The arytenoid cartilage is then removed piecemeal but the muscular process and articular facet are left in place. The edges of the mucosal wound are debrided but no attempt to close the mucosa is necessary and the defect is left to granulate. The laryngotomy incision is left to heal by second intention. A tracheotomy tube is generally not required for the post-operative period.

## Future developments in equine laryngeal surgery

In his comprehensive review of equine laryngeal surgery Spiers (1987) comments that when the historical aspects of this subject are considered " it is humbling to note how little progress has been achieved in the 150 years since Günther Junior began his investigations, and even more so to realise that the methods employed today had been explored by the year 1900."

In the years since that review was published there have been no revelations and, as predicted, most effort is being directed towards the refinement of existing techniques. The failure rate of PL surgery remains significant and most exponents of this surgery for RLN are conscious of the need to provide enduring abduction and to eliminate dysphagia. It is probable that alternative cordopexy techniques will evolve to fulfil these requirements. Recent years have undoubtedly seen advances in the specificity of diagnosis and improved facilities for assessing the impact of RLN on the athletic horse will permit a continuing critical appraisal of corrective surgical procedures.

The potential for laryngeal functional restoration by re-innervation of the atrophic intrinsic musculature has already been mentioned. Clinicians are pressed to provide corrective measures that are immediately effective because the athletic careers of horses are brief. However, this must not be a deterrent to the development of re-innervation techniques.

Ultimately, equine veterinary science is being asked to make a silk purse out of a sow's ear and the profession should look to prevention of RLN by selective breeding programmes as the enlightened way forward.

# UTILIDAD DE LA ENDOSCOPIA ENDOTRAQUEAL RETROGRADA EN EL DIAGNOSTICO DE TUMORES DE VIAS RESPIRATORIAS ALTAS

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## Resumen

Se describe la técnica de endoscopia endotraqueal retrógrada y se ilustra su utilidad práctica en dos casos de carcinoma de células escamosas en vías respiratorias altas: un tumor laríngeo en un caballo cruzado de 10 años y un tumor faríngeo en una yegua P.R.E. de 3 años. En ambos casos la endoscopia endotraqueal retrógrada permitió una exploración más detallada de las lesiones y posibilitó el acceso a áreas lesionales con menores procesos inflamatorios secundarios, en las que es más fácil realizar el diagnóstico histopatológico de neoplasia.

## Introducción

Aunque no son muy frecuentes, los procesos neoplásicos de vías respiratorias altas constituyen una causa importante de obstrucción respiratoria en caballos. Estas neoplasias pueden ser muy variadas, habiéndose descrito carcinomas de células escamosas, adenocarcinomas, fibrosarcomas, linfomas, osteosarcomas, tumores de origen dental, etc . En cuanto a su localización anatómica, pueden afectar a cavidad nasal, senos paranasales, faringe y laringe. Debido a que, en la mayoría de los casos, la sintomatología no es evidente hasta que se produce una obstrucción respiratoria grave, cuando el animal se presenta a consulta, suele mostrar disnea muy acentuada. Esto determina que, en algunos casos, no sea posible avanzar el endoscopio por la vía habitual (endonasal, en sentido rostrocaudal).

Por otra parte, en estas circunstancias en las que existe dificultad respiratoria grave y riesgo de obstrucción completa, que conllevaría la muerte del animal por asfixia, está indicado realizar una traqueotomía. De esta forma, se asegura el paso de aire por una vía alternativa. Además, de su utilidad terapéutica, la traqueotomía proporciona una vía de acceso endoscópico transtraqueal que posibilitará realizar la endoscopia retrógrada.

A continuación se presentan dos casos clínicos en los que se ilustra la utilidad de esta técnica para el diagnóstico de tumores de vías respirtorias altas.

## Caso 1

Reseña: Caballo cruzado, macho entero, 10 años

Anamnesis: Desde hace 3 días respira con dificultad, el animal está empeorando progresivamente de forma rápida.

Exploración: T=38.4 °C, P= 60/min, R=29/min

Ligera deshidratación, mucosas pálidas

El caballo manifiesta disnea inspiratoria muy acusada, acompañada de ruido respiratorio. Existe riesgo de asfixia, por lo que se realiza una traqueotomía de urgencia. Tras establecer la traqueotomía, el caballo pasa a respirar normalmente. Se realiza un examen endoscópico que permite observar que la laringe se encuentra cerrada casi por completo y que está comprimida por una masa que presiona sobre el aritenoides izquierdo. La laringe está tan cerrada que no es posible introducir el endoscopio por la rima de la glotis. Radiológicamente se confirma la existencia de una masa en la zona de proyección aritenoidea. Con el fin de confirmar si la masa se localiza sólo extraluminal o si, por el contrario, también afecta a la parte interna de la laringe se lleva a cabo una endoscopia endotraqueal retrógrada. Mediante esta técnica se descarta la presencia de una masa intralaríngea.

Se realiza biopsia transendoscópica de la masa perilaríngea y se instaura un tratamiento antiinflamatorio y antibiótico.

El análisis anatomopatológico informa que la masa perilaríngea es un carcinoma de células escamosas.

Diagnóstico: Carcinoma de células escamosas

Pronóstico: Muy grave

Tratamiento: Ante la gravedad del pronóstico el propietario declina realizar tratamiento. Dado que el animal ha mejorado considerablemente durante el tiempo de hospitalización y respira confortablemente a través del tubo de traqueotomía, el propietario decide aplazar la eutanasia hasta que el animal empeore.

## Caso 2

Reseña: Yegua P.R.E., 3 años

Anamnesis: Hace 2 meses empezó a presentar mucosidad por el ollar izquierdo y a manifestar ruido respiratorio. Progresivamente ha disminuido su capacidad de trabajo. Cada vez respira con mayor dificultad y hace más ruido. Ha recibido diversos tratamientos, observándose mejoría parcial tras la administración de Dexametasona.

Exploración: T=38.1 °C, P= 44/min, R=11/min

La yegua muestra disnea muy acusada, acompañada de ruido respiratorio (inspiratorio) estridente y de respiración bucal. Existe exudado nasal mucopurulento, más abundante por el ollar izquierdo. También elimina el exudado vía bucal. Se observa epifora en ojo derecho y deformación ósea del área frontal, en la zona interocular, más acentuada en el lado izquierdo. El hueso no es deprimible ni aparentemente doloroso. Examen endoscópico: obstrucción completa de la cavidad nasal izquierda a unos 15 cm de los ollares. Se observa una masa con aspecto inflamatorio crónico y mineralizada. Obstrucción casi completa de la cavidad nasal derecha, deja pasar algo de aire ventral y dorsolateralmente. No es posible introducir el endoscopio hasta la faringe. Radiológicamente se observa la existencia de una masa en cavidad nasal con afectación simultánea del seno frontal. Se realiza una traqueotomía tras la cual

desaparecen la disnea y el ruido respiratorio Con el fin de explorar faringe y laringe, así como para buscar una zona más apropiada para tomar biopsia se realiza una endoscopia endotraqueal retrógrada. Mediante esta técnica se observa una gran masa que obstruye prácticamente por completo las coanas y hace algo de prominencia en faringe. No existe afectación faríngea ni laríngea.

Se realiza biopsia transendoscópica.

El análisis anatomopatológico informa que la masa es un carcinoma de células escamosas.

Diagnóstico: Carcinoma de células escamosas.

Pronóstico: Muy grave.

Tratamiento: Dado que la yegua respira sin dificultad a través del tubo de traqueotomía, el propietario solicita el alta. Tras conocer el resultado de la biopsia, el veterinario referente realiza eutanasia.

## Discusión

Los dos casos que aquí se presentan ilustran la utilidad de la endoscopia endotraqueal retrógrada como técnica diagnóstica que permite acceder a zonas que, debido a la existencia de procesos patológicos (en este caso neoplasias malignas), no son accesibles mediante la técnica endoscópica normal (avance endonasal en sentido rostro-caudal).

Además, en estos casos, en los que el diagnóstico definitivo debe basarse en el estudio de una biopsia, la endoscopia retrógrada permite el acceso a áreas que están menos alteradas por procesos inflamatorios secundarios y, de esta forma, mejora considerablemente la sensibilidad diagnóstica a la hora de detectar neoplasias.

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# CASO CLÍNICO DE ASFIXIA PERINATAL CON ENCEFALOPATÍA NEONATAL MUY GRAVE

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El objetivo de este caso es destacar la metodología diagnóstica, terapéutica y el pronóstico de un caso de asfixia perinatal y encefalopatía neonatal en un potro prematuro.

Se describe el caso de una potra PRE prematura, de 6 horas de vida con historia de incapacidad para levantarse, falta de ingestión de calostro y alteración respiratoria desde el nacimiento.

El animal presentaba signos marcados de ser prematuro, con fases de depresión y estupor, debilidad marcada, ausencia del reflejo de succión y midriasis. Además, el patrón respiratorio era variable, con fases bradipnéicas y apneas. El estado cardiovascular reflejaba ligera hipotensión y signos marcados de septicemia (IS 17). En la analítica sanguínea, destacaba una marcada leucopenia con neutropenia, hipoglucemia e hipercapnia. También presentó periodos de hipoxia.

Se diagnóstico una encefalopatía neonatal grave asociada a un síndrome de asfixia perinatal, así como una septicemia y fallo en la transferencia de inmunidad pasiva. Se estableció un tratamiento de urgencia consistente en oxigenoterapia, fluidoterapia de reanimación y estimulantes respiratorios, junto con antibioterapia y antiinflamatorios. También se administró plasma y se mantuvo con nutrición enteral.

En las primeras horas, la potra evolucionó presentando convulsiones y alteración de pares craneales, siendo controladas con anticonvulsivantes (diazepam y fenobarbital), DMSO y diuréticos osmóticos. En las siguientes horas, el patrón respiratorio se normalizó. Las alteraciones neurológicas mejoraron progresivamente a lo largo de 3 días. Durante las 3 semanas de su hospitalización, la evolución fue muy favorable, normalizándose su comportamiento y estado general.

Como conclusión, realzamos la posibilidad de éxito en casos de encefalopatía neonatal de pronóstico muy grave mediante un procedimiento diagnóstico y terapeútico adecuados, junto con una hospitalización intensiva especializada.

# ECOGRAFÍA TORÁCICA NO CARDÍACA EN EL CABALLO

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Aunque la ecografía se utiliza cada vez más en medicina equina, su aplicación está bastante limitada a los sistemas musculoesquelético y reproductor. En estos campos se trata de una técnica prácticamente imprescindible para establecer el diagnóstico y el pronóstico de muchos procesos.

La calidad de los ecógrafos de que disponen los veterinarios de campo ha mejorado mucho en los últimos años y esto hace que los mismos aparatos que se utilizan para Ecografía de tendones o reproducción, permitan obtener imágenes aceptables de otros órganos o sistemas.

Aunque exista la tendencia a pensar que el pulmón es una estructura llena de aire y que, por lo tanto, la ecografía tiene una utilidad limitada en la evaluación del sistema respiratorio, lo cierto es que la ecografía pulmonar permite el diagnóstico y seguimiento de numerosos procesos clínicos y, en ocasiones, puede ser importante para establecer un pronóstico.

El objetivo de esta comunicación consiste en familiarizar al veterinario con las imágenes ecográficas normales del campo pulmonar, así como las principales alteraciones que puede encontrar en su práctica diaria y su significado clínico. Para ello nos hemos ayudado de diversos vídeos y fotografías en los que se pueden ver imágenes en movimiento de pulmones sanos y de las lesiones más frecuentes (consolidación pulmonar, abscesos pulmonares, neumotórax, derrames pleurales) que pueden observarse en procesos relativamente frecuentes como las neumonías y pleuroneumonías post-transporte o las infecciones por *Rhodococcus equi* en potros. Todas las imágenes corresponden a casos tratados en el Hospital Clínico Veterinario de la UAB.

# IMPORTANCIA DEL EXAMEN CLÍNICO EN NEONATOS. ESTUDIO COMPARATIVO DE LA TASA DE SUPERVIVENCIA DE NEONATOS HOSPITALIZADOS RELACIONADA CON LAS CONSTANTES DE ENTRADA

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## Introducción:

El hecho de realizar de forma correcta un examen físico completo del neonato en el campo, permitirá al clínico valorar su estado y poner en marcha la más adecuada estrategia de manejo y tratamiento, que garantice en mayor grado, la supervivencia del animal.

Entre dichas estrategias, la toma de decisión de "referir a tiempo" el potro a un centro donde pueda recibir medicina intensiva de urgencia, profundizar en el diagnóstico y terminar de elaborar un pronóstico; es de vital importancia.

De ello dependerá la supervivencia de aquellos animales cuyo tratamiento sería difícil de realizar en otras condiciones distintas a las que ofrece un centro de estas características, que permite una monitorización continua del animal, y tratamiento intensivo, así como la realización de determinadas pruebas diagnósticas complementarias.

El siguiente estudio ha sido realizado con los casos de neonatos referidos (n=30) al Servicio de Medicina Interna y Neonatología del Hospital Veterinario de Aznalcóllar, Sevilla (España). Nuestro objetivo fue evidenciar, la relación existente entre las constantes físicas con que ingresaron los potros y la tasa de supervivencia de los mismos.

Se estudiaron 25 parámetros, obteniendose marcadas diferencias en 3 de ellos: actitud, presencia de reflejo de succión y consumo de calostro.

## Discusión:

Los datos que necesitará el clínico de campo para emitir un posible diagnóstico precoz del estado clínico del potro y sus posibles complicaciones, se asientan sobre 3 pilares básicos:

1. HISTORIA DE LA YEGUA: una anamnesis exhaustiva aportará una valiosa información. Incluirá detalles de sus antecedentes reproductivos (incluso de otras yeguas en la misma explotación),

evolución de la gestación, duración y/o complicaciones del parto, así como las condiciones en que se produjo (factores ambientales), lactaciones preparto, actitud respecto al recién nacido, alimentación, vacunaciones, desparasitaciones... etc.

2. HISTORIA DEL POTRO desde su nacimiento: medicaciones o procedimientos realizados, cuidado del ombligo, ¿mamó? (cuánto y a qué hora después del nacimiento), ¿pasó meconio?, ¿orinó?...etc.

3. EXAMEN FíSICO.
   - Actitud
   - Temperatura (37.2-38.8 °C)
   - Frecuencia Cardíaca (80-120 ppm)
   - Frecuencia Respiratoria (30-40 rpm, hasta 60 la primera semana)
   - Tiempo de Relleno Capilar (1 -2 seg.)
   - Estado de las Mucosas (rosadas y húmedas)
   - Grado de Hidratación
   - Sonidos intestinales
   - Heces (¿meconio?)
   - Orina (¿lo hace?, ¿por dónde?)
   - Ombligo (¿tumefacción?, ¿humedad?, ¿orina?)
   - Articulaciones (¿inflamación?, ¿cojera?, ¿calor?)
   - ¿Malformaciones congénitas?

Nos aporta información básica (aunque nunca definitiva) para establecer posibles diagnósticos diferenciales y decidir a tiempo un tratamiento agresivo a seguir. Ofrece la ventaja de poderse efectuar con independencia de las condiciones de manejo, instalaciones, experiencia del clínico, equipamiento, posibilidades económicas del propietario o valor del animal.

Son también de máxima utilidad determinadas pruebas diagnósticas laboratoriales como: hematología, bioquímica sanguínea (glucosa, calcio, albúmina... etc.), electrolitos (Na, k, Cl) o test rápidos para detección de Ig G.

El examen ecográfico y/o radiográfico así como otros procedimientos, como la punción articular o la abdominocentesis, aunque de gran utilidad, requieren equipamiento y familiariación con la técnica y su correcta interpretación.

**Actitud:** Frecuentemente los signos de enfermedad en el neonato pueden pasar desapercibidos. Muchos potros se encuentran aparentemente bien durante las primeras horas tras el nacimiento (lo que se conoce como "grace *period*"), intervalo de tiempo que a menudo va seguido de un empeoramiento de las condiciones de 12 a 24 horas después. A esto hay que añadir la rapidez con que se llevan a cabo dichos cambios en el recién nacido, (tanto a peor como a mejor), lo que nos obliga a actuar con diligencia, puesto que en la mayoría de las situaciones vamos "contra reloj".

**Consumo de calostro:** A la dependencia de consumir calostro, se añade un sistema inmune aún por desarrollar y no tan efectivo como el del adulto (incluso cuando los niveles de Ig G son óptimos). La falta de transferencia pasiva de Ig G supondrá entonces un problema importante, marcando la evolución del animal y la capacidad de defensa de éste ante los microorganismos (patógenos o no), existentes en su entorno.

Con los datos obtenidos podremos clasificarlo dentro del grupo de neonatos de alto riesgo anteriormente mencionado, al que habría que prestar especial atención. La siguiente tabla recoge las condiciones asociadas a neonatos de alto riesgo:

**Historia:**
- Anteriores potros con:
  - Isoeritrolisis neonatal
  - Síndrome de mala adaptación
  - Malformaciones congénitas
  - Prematuros
  - Nacidos más tarde
  - Muertos por asfixia
- Distocia
- Separación prematura de la placenta
- Rechazo al potro
- Exposición reciente a enfermedades infecciosas asociadas con abortos y mortinatos (herpesvirus equino, arteritis viral, *leptospira* spp.)

**Problemas reproductivos y de la glándula mamaria**
- Fibrosis endometrial severa
- Hidroalantoides
- Descarga vaginal purulenta
- Ruptura del tendón prepúbico
- Lesiones pélvicas
- Agalactia
- Calostro de mala calidad
- Lactación prematura o perdida de leche

**Problemas neonatales**
- Tinción de meconio
- Problemas de la placenta:
  - Placentitis
  - Atrofia de las vellosidades
  - Edema
- Gemelos
- huérfanos
- Retraso o falta de consumo de calostro
- Inmaduros
- Prematuros
- Exposición a enfermedades infecciosas (influenza)
- Traumatismos
- Condiciones ambientales adversas
- Retraso para levantarse y mamar
- Anormalidades congénitas
- Septicemia
- Uraco persistente

**Problemas sistémicos:**
- Fiebre
- Anemia o hipoproteinemia
- Endotoxemia
- Patologías gastrointestinales (cólico)
- Malnutrición
- Infección sistémica severa
- Laminitis
- Decúbito prolondado (problemas musculoesqueléticos o neurológicos)
- Medicación excesiva
- Transporte prolongado antes del parto

**Problemas durante la gestación o el parto**
- Parto prematuro
- Gestación prolongada
- Parto prolongado
- Inducción del parto
- Distocia
- Ruptura prematura del cordón umbilical
- Anormalidades del cordón umbilical
- Cesárea
- Separación prematura de la placenta

*Fuente: Large Animal Internal Medicine 3rd ed., Bradford P. Smith.*

**Conclusión:**

Los resultados de nuestro estudio fueron los siguientes : De una muestra de 30 potros, el 52% sobrevivieron, mientras que el resto murieron o fueron eutanasiados (48%). Se estudiaron 25 parámetros (actitud, Fc, Fr, Tª, color de las mucosas, TRC, grado de hidratación, sonidos intestinales, heces, orina, consumo de calostro, reflejo de succión, hematocrito, proteínas totales, recuento leucocitario, glucosa, calcio, sodio, potasio y cloro, albúmina, creatinina y urea, bilirrubina total, concentración de Igs en plasma). Se encontraron marcadas diferencias en 3 parámetros: actitud, consumo de calostro y presencia de reflejo de succión.

En lo referente a la actitud, el 73% de los animales que murieron, ingresaron deprimidos o débiles. Por el contrario solo el 46% de los que sobrevivieron fueron recibidos en este estado.

Consumieron calostro el 86% de los potros que sobrevivieron, y un 71 % de los pertenecientes al grupo de los fallecidos o eutanasiados lo hicieron. Más significativas, fueron las diferencias en cuanto al reflejo de succión, que presentaron el 66% de los potros que fallecieron, alcanzándose el 82% en los supervivientes.

En cuanto a la concentración de Igs en plasma, decir que entre los potros que consumieron calostro, un 16% de los supervivientes presentó un déficit de las mismas (< 400 mg/dl); porcentaje que ascendía al 25% en el grupo de los fallecidos. Ni que decir tiene, que en todos los potros que no consumieron calostro (sobrevivieran o no), también se encontraron bajos niveles de Igs.

La experiencia demuestra que aquellos potros que fueron remitidos a tiempo al Hospital y que por lo tanto llegaron en mejor estado (constantes físicas menos deterioradas) siempre tuvieron una mejor evolución, incluso en aquellos casos en los que la concentración de Igs en plasma era baja, déficit que se corrigió mediante transfusiones de plasma. Por el contrario en la mayoría de los casos que fueron atendidos tardíamente, fallecieron en pocas horas, o fueron eutanasiados.

Por esta razón y de acuerdo con los datos obtenidos, podemos deducir que el examen físico aportará información valiosa a la hora de hacer una primera valoración y decidir un tratamiento agresivo a tiempo, lo que es crucial para la supervivencia del neonato. La vigilancia intensiva y monitorización continua del animal, así como la realización de determinadas pruebas diagnósticas serán claves para el éxito del caso.

En definitiva, el futuro del animal dependerá del "tiempo" del que dispongamos, atendiendo a la siguiente fórmula:

+ rapidez con que somos requeridos por el propietario.
- la gravedad del caso.
- velocidad con que se desarrollan los acontecimientos en los neonatos.
+ diligencia a la hora de emitir un diagnóstico y/o referir al animal.
- limitaciones de la clínica de campo.

Ni que decir tiene, que además de estos factores, existen otros de índole económica, que muchas veces suponen la principal limitación a la hora de tomar una decisión con respecto a la estrategia a seguir con el animal: referirlo a un centro especializado, realizar un tratamiento general paliativo, sacrificarlo....

Finalmente, seguir un orden y realizar por escrito una buena recogida de datos, es fundamental para no dejar atrás detalles a simple vista insignificantes, que sin embargo pueden ser de muy valiosa ayuda a la hora de establecer un posible diagnóstico y tratamiento.

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# REHIDRATACIÓN ORAL COMO MEDIDA TERAPÉUTICA EN CABALLOS CON CÓLICO

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El objetivo de este estudio era evaluar la utilidad clínica de la rehidratación oral como medida terapéutica en los procesos obstructivos de intestino grueso en caballos.

Se incluyeron los animales referidos con cólico y recibidos por el Servicio de Medicina Interna Equina durante el año 2003, y se excluyeron los que requirieron tratamiento quirúrgico. El tratamiento médico recibido fue de sobrehidratación y analgésicos. La sobrehidratación consistió en fluidoterapia intravenosa a velocidad de mantenimiento y rehidratación oral con soluciones poliiónicas isotónicas por sonda nasogástrica a velocidad media de 6-8 l/h. Los animales se mantuvieron en vigilancia intensiva, con exámenes físicos, rectales y analíticas periódicas. Se consideró el proceso resuelto en base a la evolución del problema.

De los 28 animales con cólico incluidos, 15 tenían una impactación de colon mayor, 9 un desplazamiento de colon, y 4 un proceso obstructivo simple. De las impactaciones, 12/15 (80%) se curaron empleando la rehidratación oral; el resto, se solucionaron sólo con fluidoterapia intravenosa. De los 9 desplazamientos de colon, 7 (77,8%) se solucionaron con rehidratación oral, mientras que todos los procesos obstructivos simples se resolvieron con esta medida terapéutica. Los procesos se solucionaron en un periodo medio de 15,7 h ±2,7 y con un volumen de rehidratación oral medio de 79 l.

Con estos resultados, se puede considerar que la rehidratación oral es una medida terapéutica útil en caballos con cólico por un proceso obstructivo de intestino grueso. Sin embargo, hay que recalcar que se requiere un diagnóstico preciso y una monitorización intensiva.

# LA HIPERTRIGLICEMIA, HIPERLIPEMIA E HIPERLIPIDEMIA, ¿SON UNAS COMPLICACIÓNES COMUNES DE LOS ANIMALES ENFERMOS?

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## RESUMEN

La hipertriglicemia severa es una es una enfermedad más común de lo que nosotros creemos. En los animales inapetentes por cualquier enfermedad o pos- cirugía.

Es un parámetro bioquímico que deberíamos medir rutinariamente en animales anoréxicos por cualquier causa. La hiperlipemia y la hiperlipidemia son patologías menos comunes.

Hemos cogido un periodo de tiempo de un año y ha hecho una selección de 130 caballos con diferentes patología s relacionadas de los cuales 12 tenían los triglicérido por encima de los valores normales:

- 8 caballos tenían los triglicérido (TRG) por encima de 300 mg/dl de los cuales
- 6 tenía una hipertriglicemia severa con valores TRG por encima de 500 mg/dl

Todas están causadas `por un ***equilibrio metabólico negativo***, normalmente causado por:

1. Otras enfermedades tales como insuficiencia renal, colitis, babesosis...
2. Mala nutrición
3. Aumento de la demanda en lactación, gestación
4. Disminución de la ingesta obligada en cólicos quirúrgicos, problemas esofágicos, laminitis

Este balance energético negativo hace los triglicérido del tejido adiposo se rompan en glicerol y ácidos grasos. Una parte significativa de estos ácidos grasos se llevan al hígado donde se completará su oxidación produciéndose como resultado de estas cetonas y la reesterificación de los triglicérido. Los triglicérido son acumulados en el hígado o soltados al plasma en forma de lipoproteinas de baja densidad molecular.

En los caballos la producción de triglicérido supera la de cetonas por lo tanto en el ayuno prolongado es más común la hipertriglicemia que la cetosis. Un aumento prolongado de los TRG en suero está asociado con la acumulación de lípidos en hígado, riñón,miocardio, músculo esquelético y esta acumulación provoca un mal funcionamiento de estos órganos.

A continuación haremos una breve descripción de estas patologías así como su diagnóstico y tratamiento.

## HIPERLIPEMIA:

Ocurre fundamentalmente en ponies, caballos miniatura y burros obesos y esta caracterizada por **una infiltración grasa** de diferentes órganos fundamentalmente el hígado y por **un suero que este opaco** por acumulación de lípidos.

- Los triglicérido en sangre sobrepasan los **500 mg /dl**
- Normalmente está asociada con **azotemia** y esto bloquea todavía mas la captación del hígado de los triglicérido.

Está caracterizada por una **producción por el hígado anormal** de la fracción de la lipoproteína de baja densidad molecular. (VLDL1).Además se ha descubierto que en estos animales esta lipoproteína tiene un contenido en apolipoproteína B100 reducido y un incremento de la apolipoproteina B48, esta substitución parece que permite un aumento del contenido de triglicérido,

En otro estudio se veía además que las lipoproteína lipasa y lipasa enzima responsable de la metabolización de las VLDL estaban incrementadas en ponies hiperlipemicos. Está demostrado que la hiperlipidemia está causada por una sobreproducción de VLDL por el hígado.

La **resistencia relativa a la insulina en los animales obesos** hace además que la lipólisis se haga más incontrolable en un balance energético negativo

## SIGNOS CLINICOS

1. Depresión
2. Disminución del apetito
3. Taquicardia, taquipnea, hipertermia
4. Edema pendiente
5. Ictericia
6. Diarrea es común
7. Casos graves pueden haber signos clínicos de fallo hepático agudo
8. La mortalidad en la hiperlipemia reflejada en los últimos estudios es alta 60- 80 %, pero si se trata de manera adecuada y rápidamente el índice de supervivencia mejora mucho.

## PATOLOGÍA CLINICA Y EXAMENES DIAGNOSTICOS

1. Observación del plasma es un signo patognomónico, encontrándonos el plasma denso de color pálido, opaco debido a la alta concentración de lípidos
2. Los TRG en sangre están elevados por encima de 500mg/dl
3. Generalmente esta asociada a una azotemia de leve a moderada

4. La bilirrubina generalmente está elevada, (como en la mayoría de los caballos que están anoréxicos)
5. Generalmente existe una acidosis metabólica acentuada.

## HIPERLIPIDEMIA

Es una forma más suave que consiste en una elevación moderada de los TRG **que casi no sobrepasan los 500mg/dl , plasma claro** y no existe infiltración grasa de órganos. Un aumento ligero de las calorías ingeridas nos harían revertir generalmente los signos clínicos.

## HIPERTRIGLICEMIA

Es un aumento de los **TRG en sangre por encima de los valores normales** y no siempre relacionado con un problema clínico aparente.

## TRATAMIENTO GENERICO

1. Tratar la **enfermedad primera** si existiera
2. **Mejorar el balance energético:**
    - **Ofreciendo alimentos** palatables y energéticos
    - **Nutrición enteral** con o sin nutrición parenteral

      Deberemos sondarlo de 4 a 6 veces al día a razón de 1 caloría por ml debemos no aportar más energía en forma de lípidos por que exacerbaremos la hiperlipemia. Existen dos formulas estándares para ello en una se incluye pienso en pelets molido con agua y en el otro método utilizaremos alfalfa deshidratas, caseína, electrolitos y dextrosa
    - **Nutrición parenteral** exclusiva o combinada con nutrición enteral en forma de:
        - Dextrosa al 5% , (2 ml / Kg /hr). ( no sirve como única fuente de energía por que las calorías son insuficientes)
        - Soluciones para nutrición parenteral con lípidos, hidratos de carbono y aminoácido en una proporción adecuada.
3. **Heparina 40-100 UI** /Kg/12 h, se ha usado para alterar la actividad de la lipasa y inhibir la hormona lipasa sensitiva del tejido adiposo.
4. **Insulina** en forma de zinc protamina 0,1-0,3 UI /Kg / SC o IM /12 H.

Una vez definidas estas patologías podemos concentrarnos en la haremos un estudio de los casos referidos a nuestro hospital en los cuales hemos tenido unos valores de triglicérido por encima de lo normal.

Hemos cogido un periodo de tiempo de un año :

De 130 caballos hospitalizados por diferentes patologías: en 12 caballos se descubrió que tenían los TRG por encima de lo normal.(67 mg/DL). , Dé los cuales 3 tenían valores correspondientes a una hipertriglicemia moderada entre 300 -500 mg /dl , y 6 tenían una hipertriglicemia severa , por encima de 500 mg /dl.

Las patologías por lo que fueron hospitalizados fueron :

- Babesiosis 1
- Cólicos 4
- Insuficiencia renal aguda 1
- Potros 3
- Hiperlipemia 3

El tanto por ciento sobre el total de los caballos que tenían los TRG por encima de los valores normales es de un 9,2 %. La mayoría de los caballos tenían sintamos clínicos y laboratoriales de síndrome de respuesta sistémica inflamatoria. , Tales como: taquipnea, taquicardia e hipertermia. Un 36% tenía azotemia, e hipoglucemia, 50 % tenían hipoalbuminemia , un 70 % tenia la bilirrubina total por encima de 4 mg/dl y un 37,4 %tenía la GGT por encima de 56 U/L.

67 % de estos sobrevivieron, 2 fueron eutanasiados por cuestiones económicas y uno de ellos murió.

El tratamiento consistió en nutrición enteral de 4 a 6 veces al día en 2 /12 , en 5/12 glucosa al 5 % en combinación con nutrición enteral, en 2 /12 el tratamiento sólo consistió en darles comida más palatable y en los potros 3/12 se utilizó solo glucosado 5% . Se utilizó heparina en un 5 /12.

La media de días de tratamiento en los caballos con hipertriglicemia moderada (<500 mg /dl) fue de 5 días . La media de días de tratamiento en los caballos tratados con triglicérido por encima de 500 mg /dl fue de 7 días de los 2 casos que tenemos dato .

| CASO | DIA | FC | FR | T° | STG mg/dl | CRE mg/dl | GLU mg/dl | ALB g/dl | BT | GGT | WBC | RAZA | EDAD | SEXO | RESUL | TTO | PATOL | DIAS TTO |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 88 | 42 | 40° | 372,2 | 5,21 | 241 | | | | 9400 | PRE | 9 | MACHO | POS | GLU5% | IRA | 9 |
| | 2 | 60 | 20 | 39,1° | 349,3 | 3,53 | 140 | 2 | | | | | | | | SONDA | | |
| | 4 | 58 | 26 | 38° | 264,3 | 2,77 | 121 | 2,61 | | | | | | | | | | |
| | 6 | 60 | 20 | 37,1° | 375 | 1,94 | 136 | | | | | | | | | | | |
| | 10 | 52 | 16 | 37,1° | 252 | | | | | | | | | | | | | |
| 2 | 1 | 44 | 16 | 39° | 290 | 1,53 | 128,9 | 2,39 | 7,43 | 13 | 3425 | AA | 15 | MACHO | POS | SONDA | BABESI | 6 |
| | 4 | 40 | 20 | 38,4° | 242 | | 126 | 2,4 | 3,74 | 15 | 5400 | | | | | | | |
| | 6 | 40 | 20 | 38,4° | 166,6 | | 122 | 1,46 | | | 6925 | | | | | | | |
| 3 | 6 | 40 | 40 | 38,4° | 980 | 1,66 | 25,8 | 2,61 | 6,7 | | #### | CRU | 10 | MACHO | POS | GLU% SONDA | COLICOQ | 3 |
| | 7 | 44 | 50 | 37,5° | 191,9 | | 90,2 | | | | | | | | | HEPARINA | | |
| 4 | 14 | 64 | 32 | 37,8° | 189 | 1,29 | 100,6 | 2,6 | 4,48 | 33 | 3400 | CRU | 9 | MACHO | MUERTO | PIENSO | COLICOQ | 4 |
| | 19 | 68 | 40 | 38,5° | 373 | 1,7 | 90,5 | | | | | | | | | | | |
| 5 | 1 | 64 | 32 | 38,8° | 360 | 2,25 | 143,7 | | 2,71 | 57 | | PRE | 13 | MACHO | POS | GLU5% | HIPERL | 10 |
| | 2 | 56 | 28 | 38,9 | 300 | | 130 | | | | | | | | | HEPARIN | REFERID O | |
| | 4 | 44 | 20 | 37,8 | 307 | | | | | | | | | | | PIENO | | |
| | 6 | 44 | 18 | 37,7 | 127,6 | | 123,8 | | | | | | | | | SONDA | | |
| | 10 | 40 | 12 | 38,1 | 94,7 | | 122 | | | | | | | | | | | |
| 6 | 1 | ## | 76 | 40,1 | 897,1 | 7,16 | 100,6 | 1,9 | | | #### | PRE | 3 DIAS | MACHO | EUTAN | GLU5% | PREMAT U | 1 |
| 7 | 4 | 48 | 44 | 40,2 | 163,5 | 1,2 | 131 | 2,68 | 3,67 | 92 | #### | CRU | ???$$$ | HEMB.L | POS | PIENSO | COLLICQ | ¿? |
| 8 | 1 | ## | 40 | 39;1 | 670 | 3,29 | 155 | 2,29 | 4,15 | 7 | 5.300 | PRE | 3 MESES | HEMPRA | EUT | GLU5% | RESP.ST R | |
| 9 | 1 | | | | 1490 | 1,31 | 108,9 | 1,9 | 5,81 | 21 | #### | PRE | 3 MESES | HEMBRA | POS | GLU 5% | RESP.ST R | 7 |
| | 3 | ## | 44 | 40,3 | 124,3 | | 171 | 2,18 | 1,82 | 21 | | | | | | HEP | | |
| | 7 | ## | 48 | 40 | 114 | 0,65 | 198 | 2,59 | | | | | | | | | | |
| 10 | 4 | 80 | 16 | 39,2 | 172 | | 117 | | 5,77 | 79 | | CDE | 10 | MACHO | POS | PIENSO | COLICO.E | 1 |
| 11 | 1 | 60 | 14 | 39,4 | 2483 | 0,33 | 83 | | | 110 | 6.400 | CRU | 14 | MACHO | POS | PIENSO, S | HIPERLIP | 7 |
| | 4 | 64 | 16 | 38,4 | 1450 | | 140 | | | 102 | | | | | | HEP | | |
| 12 | 1 | 67 | 18 | 39,6 | 1590 | 1,61 | 105 | | 8,18 | 36 | 8435 | CRU | 13 | MACHO | POS | PIENSO,S HEPAR | HIPERLI | ?? |

## CONCLUSION

La hipertriglicemia severa ocurre más frecuentemente que lo que creemos hasta ahora en caballos y potros inapetentes y enfermos. Siendo un parámetro que deberíamos incluir cuando nos encontramos con estos signos clínicos. Además concluiremos que la mayoría de los caballos respondieron satisfactoriamente al tratamiento.

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# NOVEDADES SOBRE LAS CESTODOSIS EQUINAS

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## RESUMEN

Las cestodosis equinas son procesos parasitarios producidos por anoplocefálidos que se localizan en el intestino delgado y grueso de los équidos. Tras un estudio realizado en mataderos de diferentes zonas geográficas españolas, se ha determinado su presencia en el 39% de los animales analizados (n=437). Por primera vez en España se ha denunciado la presencia de *Anoplocephala magna* con una prevalencia del 19%. La especie más patógena, *Anoplocephala perfoliata*, se ha encontrado en un 34% de los animales. En ambos casos, las cargas parasitarias eran bajas: la mayoría de los animales albergaban menos de 30 parásitos adultos. En el 71% de los animales, los cestodos se encontraban adheridos a la válvula ileocecal: casi en la mitad de éstos las alteraciones provocadas fueron graves o muy graves (desde una enteritis pseudomembranosa hasta una enteritis necrótica focal). Tras la identificación del estado de madurez de los cestodos se ha podido establecer un patrón epidemiológico para áreas templadas secas: el periodo de riesgo de los animales abarcaría desde el verano hasta el invierno, alcanzando los máximos niveles de prevalencia durante los meses húmedos y fríos (otoño e invierno). A finales del invierno y primavera, los vermes ya grávidos eliminarían huevos al medio ambiente para ser ingeridos por los hospedadores intermediarios (ácaros oribátidos), apareciendo las fases larvarias en su interior a partir del verano. No se descarta la posibilidad de solapamiento de dos ciclos en primavera, si se dieran las condiciones medioambientales adecuadas, como parece que sucede en países cuyo clima es más frío y húmedo.

## DISEÑO EXPERIMENTAL

El estudio de las cestodosis equinas se ha realizado en tres áreas geográficas españolas en animales sacrificados en matadero. Para ello, se realizó un corte longitudinal al intestino delgado y grueso, con especial atención en las áreas de fijación predilectas por los cestodos, como es la válvula ileocecal y zonas adyacentes. Se presentan datos desde octubre de 2001 hasta septiembre de 2003 de un total de 437 équidos distribuídos de la siguiente manera: 108 procedentes del

área noreste y sacrificados en el Matadero de MercaBarna (Barcelona), 135 procedentes de áreas septentrionales en el Matadero Municipal de León y 194 del área central de la Peninsula Ibérica y sacrificados en el Matadero Municipal de Guadalajara. Alrededor del 75% de los animales tenían menos de un año y prácticamente todas las canales se destinaban a consumo humano. Sólo en los mataderos de León y Guadalajara se han podido tomar las muestras con una periodicidad semanal o quincenal y tan sólo éstas se han incluido en el estudio de prevalencia estacional.

## RESULTADOS Y DISCUSIÓN

La prevalencia global para las cestodosis equinas en España ha sido del 39%. Tanto en el área septentrional como en la zona centro se ha detectado por primera vez en España el cestodo *Anoplocephala magna* con una prevalencia del 19%. Las cargas parasitarias fueron muy bajas (9 ± 12 vermes) con un 96% de los animales con menos de 30 parásitos en los tramos anteriores del intestino delgado. No se observó ninguna lesión relacionada con su presencia.

*Anoplocephala perfoliata* es la especie de cestodo más patógena de los équidos y se ha detectado en un 34% de los animales, apareciendo de forma mixta junto a la anterior en el 13% de ellos. Las cargas parasitarias también fueron bajas (24 vermes ±49) y en un 71% de los animales los cestodos estaban fuertemente adheridos a la válvula ileocecal. En todos estos animales aparecieron lesiones relacionadas con esta fijación. Se establecieron tres grados de lesión: desde una ligera hiperemia con las características petequias por el efecto de succión de las ventosas, hasta lesiones más graves como enteritis pseudo-membranosas con o sin necrosis de los tejidos subyacentes. Estas enteritis se caracterizaban por la presencia de membranas diftéricas que englobaban los escolex de los parásitos. Casi la mitad de los animales con cestodos en la válvula ileocecal presentaban enteritis más o menos grave. Estaban presentes en todos los animales con cargas parasitarias altas, pero también en un alto porcentaje de animales con menos de 30 vermes. Por lo tanto, en las condiciones españolas la prevalencia de cestodos es alta y las cargas parasitarias muy bajas, pero la existencia de lesiones en la válvula ileocecal compatibles con manifestaciones clínicas es muy frecuente.

Los datos obtenidos a lo largo de todos los meses de muestreo han permitido establecer un patrón estacional en el que la prevalencia de los cestodos es baja en primavera (12%) y verano (9%), muy alta en otoño (47%) con meses puntuales que han superado el 75% de los animales parasitados (octubre 2002 en zona centro) y elevada en invierno (31%). Tras el montaje, identificación y establecimiento del estado de desarrollo de los cestodos recogidos de la especie *Anoplocephala perfoliata* (n=1420) se observaron diferencias en el grado de madurez de los parásitos. No se pudó realizar con *Anoplocephala magna* dado el escaso número total de parásitos recogidos. Se establecieron tres niveles de madurez: cestodos inmaduros que no presentaban los órganos reproductores evolucionados, cestodos maduros en los que se observaban con nitidez ovarios o estructuras del aparato reproductor masculino como el cirro y cestodos grávidos en los que el útero aparecía cargado de huevos. Sólo se observaron vermes inmaduros en verano, otoño e invierno, estableciendo así el periodo de riesgo para los animales, ya que la presencia de éstos marcaba la reciente ingestión de ácaros oribátidos parasitados. En otoño aparecieron los primeros vermes grávidos con anillos cargados de huevos, su número aumentó en invierno hasta alcanzar prácticamente el 100% en primavera. De esta manera puede deducirse que los cestodos eliminan huevos sobre todo al final del invierno y en primavera, periodo idóneo para el desarrollo de los hospedadores intermediarios, que tendrán de esa forma a su disposición gran cantidad de huevos en las heces. El tiempo de desarrollo de los huevos hasta la forma larvaria dentro del ácaro o cisticercoide está establecido en unos dos meses según condiciones mediaoambientales, por lo

que los ácaros infectantes estarán a disposición de los équidos en el pasto a partir del verano, lo que coincide con los resultados previos obtenidos.

Las condiciones medioambientales van a regular este ciclo anual de tal forma que pueden llegar a solaparse dos ciclos en primavera al adelantarse el periodo de riego a la primavera como parece que sucede en los países más septentrionales con un mayor nivel de humedad y mejores condiciones para el desarrollo de los hospedadores intermediarios.

## CONCLUSIONES

Desde que se ha establecido la clara relación de la presencia de cestodos en los équidos con la presentación de cuadros cólicos espasmódicos y de impactación ileal, la importancia de los cestodos ha aumentado notablemente. Como ha quedado demostrado en este trabajo, en España, a pesar de las adversas condiciones para el desarrollo de algunas de las fases de su ciclo biológico, se dan los requisitos mínimos para que la presencia de estos parásitos constituya un claro riesgo para los équidos, en especial, los mantenidos en pastoreo permanente. La posibilidad de que los hospedadores intermediarios encuentren adecuadas condiciones para su desarrollo y supervivencia en el alimento almacenado de los équidos ha sido recientemente sugerida en países nórdicos, donde la prevalencia de estos parásitos es superior al 60% durante todo el año y los animales están estabulados como mínimo seis meses.

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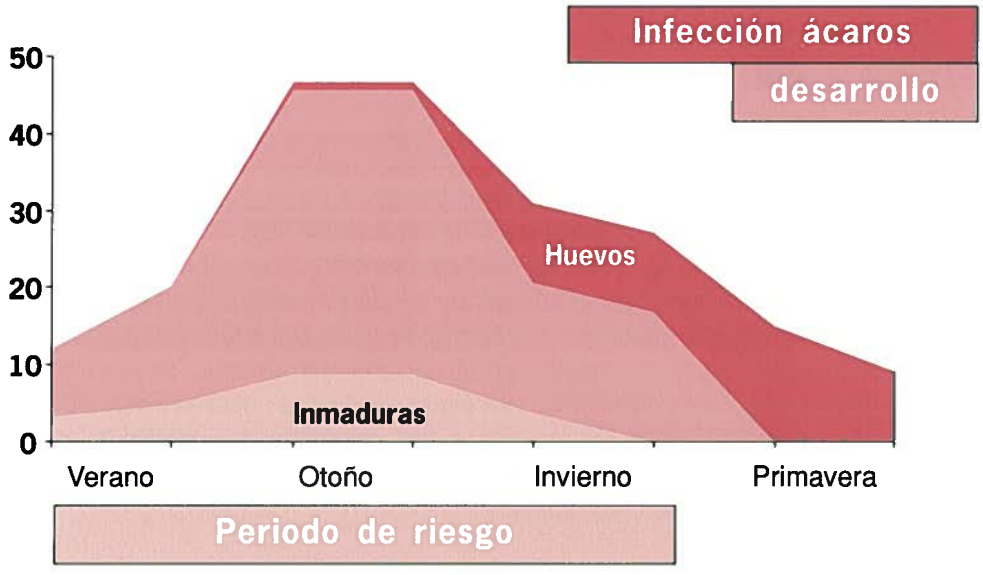
## Patrón epidemiológico
### *Anoplocephala perfoliata*



# REPARACION DE FRACTURAS CONDILARES LONGITUDINALES BAJO SEDACION IN CUATRO CABALLOS

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## Resumen

Fracturas condilares son una lesion comun en caballos de carreras.

Fracturas condilares longitudinales o tipo IV, son fracturas largas, incompletas y con configuración en espiral , que afectan al condilo medial normalmente y que se extienden hacia la diafisis.

El tratamiento de elección para este tipo de fracturas es fijación interna bajo anestesia general, sin embargo un sistema de recuparacion especial ha sido sugerido para evitar un riesgo potencial de que ocurra una fractura catastrófica durante la recuperación de estos pacientes.

Cuatro caballos con fracturas condilares longitudinales fueron referidos al hospital para su reparación. Examinacion radiológica revelo que dos caballos presentaban una fractura del condilo medial afectando a la extremidad anterior derecha, un caballo con una fractura del condilo lateral afectando a la extremidad anterior izquierda y otro caballo con una fractura del condilo medial afectando la extremidad posterior derecha.

La reparación se realizo usando tornillos corticales de 4.5 mm insertados en efecto lag con el caballo de pie usando sedacion y un bloqueo nervioso, de esta manera no tenemos que recuperar el animal y eliminamos el riesgo de que el caballo se fracture en la recuperación.

Compresión satisfactoria de la fractura fue conseguida en los cuatro casos.

Actualmente 1 de los caballos operados ha corrido 2 veces,2 estan en entrenamiento y otro esta en reposo en el establo.

Reparación de este tipo de fracturas con el caballo de pie es una técnica viable que elimina el riesgo de que una fractura castatrofica ocurra durante la recuperación de estos pacientes.

## Comunicación

### Introducción

Fracturas condilares son una lesion comun de alta velocidad en caballos de carreras.

Fracturas condilares han sido clasificadas en cuatro grandes grupos por diferentes autores (Auer 1982):

- Fracturas condilares incompletas
- Fracturas condilares completas pero sin desplazamiento
- Fracturas condilares completas y con desplazamiento
- Fracturas condilares longitudinales o especiales

Estas ultimas son fracturas largas , incompletas y con una configuración en espiral que afectan al condilo medial mas frecuentemente y que se extienden hacia la diafisis.

El tratamiento de elección de este tipo de fracturas es fijación interna bajo anestesia general mediante tornillos en efecto lag, sin embargo, un sistema de recuperación asistida especial ha sido sugerido para evitar el posible riesgo de una fractura catastrófica durante la recuperación de estos pacientes.

El objetivo de esta presentación fue evaluar una técnica quirúrgica alternativa para eliminar este riego potencial.

### Materiales y metodos

Cuatro caballos pura sangre de carreras con edades comprendidas entre cinco y seis años fueron referidos al hospital para la reparación quirúrgica de una fractura condilar del tercer metacarpo/metatarso.

El examen radiológico revelo la existencia de una fractura del condilo medial afectando a la extremidad anterior derecha en dos caballos, una fractura del condilo lateral afectando a la extramidad anterior izquierda en un caballo y otro caballo con una fractura del condilo medial afectando a la extremidad posterior derecha.Todas las fracturas eran desplazadas en la superficie articular de 1 a 3 mm por lo que la posibilidad de tratamiento conservativo estaba descartada. Examinacion radiológica tambien demostro no haber fracturas secundarias como fractura apical del sesamoideo, fractura axial del sesamoideo o chip (ratones articulares) en la superficie palmar del tercer metacarpo/metatarso.

### Tratamiento

La reparación se realizo en los cuatros casos en el box de inducción que tiene el suelo acolchado. El caballo fue sedado usando una combinación de 10 mg hidroclorato de detomidina y 10 mg butorfanol tartrade consiguiendo una sedaccion profunda. Anestesia de la extremidad se realizo mediante infiltración local de las ramas lateral y medial del nervio palmar/plantar metacarpiano y nervio palmar/plantar ( anestesia alta de los cuatro puntos) junto con una infiltración en anillo justo debajo del carpo/ tarso.

La extremidad fue preparada para cirugía.

La inserción de los tornillos fue asistida a traves de dos agujas insertadas subcutáneamente en la superficie dorsal del tercer metacarpiano paralelas a la superficie articular. Los tornillos fueron

insertados en efecto lag a traves de la cortical lateral o medial dependiendo de que condilo era el afectado a traves de pequeñas incisiones.

Después de hacer el agujero de 4.5 mm en la primera cortical una radigrafia fue tomada para asegurar de que la fractura fue alcanzada con el taladro y el agujero fue terminado con el taladro de 3.2 mm y el tornillo fue insertado.

Una vez que todos los tornillos fueron insertados, la extremidad fue levantada del suelo gracias a la ayuda de un asistente para apretar todos los tornillos y asi conseguir la máxima compresion de la fractura.

Abundante hemorragia fue evidente durante el procedimiento.

Todos los caballos toleraron muy bien toda la cirugía sin manifestar ninguna reaccion en contra del procedimiento.

Dos fracturas del condilo medial y la del condilo lateral fueron resueltas con 2 tornillos y otra fractura del condilo medial fue resuelta con 3 tornillos.

Compresión satisfactoria de la fractura en la superficie articular fue alcanzada en todos los casos.

Un bendaje compresivo fue puesto en la extremidad y el caballo fue llevado al box donde podia caminar libremente, no se empleo ningun sistema de sujeción.

**Post-operatorio**

Todos los caballos recibieron 3 mg marbofloxacina (Marbocil 10%, Vetoquinol) durante 5 dias y 2g fenilbutazona ( Pro-Dinan ) una vez al dia.

Todos los caballos incrementaron el grado de cojera cuando el efecto de la anestesia local desaparecio, en dos casos tuvimos que poner el caballo en fluidos intravenosos debido a la anorexia durante un dia y en otro caso tuvimos que usar butorfanol para controlar el dolor.

Al cuarto dia post-cirugia los caballos empezaron a mejorar en el grado de confort.

Todos los caballos fueron dados de alta del hospital una semana post-cirugia.

**Resultados**

Todos los caballos fueron confinados en el box durante 4 meses con periodicos cambio de vendaje, seguido de un mes de paso en la mano y finalmente se dejaron libres en un pequeño prado.

Todos los caballos empezaron a entrenar 7 meses post-lesion.

Actualmente un caballo corrio dos carreras pero fue retirado por otros motivos , 2 caballos estan en entrenamiento y otro esta en el box en reposo.

**Discusion**

Fracturas condilares son muy comunes en nuestra clinica debido la la gran población de caballos de carrera por la que estamos rodeados.

Las mas comunes son fracturas del condilo lateral incompletas o completas sin desplazamiento o minimamente desplazadas, estas son reparadas con tornillos de 4.5 mm bajo anestesia general y el caballo es recuperado con un bendaje compresivo sin problemas.

Fracturas del condilo medial son raras, fracturas sin desplazamiento pueden ser tratadas con reposo o con fijacion interna y tener buen pronostico, sin embargo, hay ciertas ventajas de la fijación interna sobre tratamiento conservativo( Richardson,1984). Los tornillos en efecto lag van a incrementar el confort del caballo, habra mejor compresión en la superficie articular por lo que va a

disminuir la enfermedad degenerativa articular ( artrosis del menudillo) y va a evitar la propagacion y extensión de la fractura.

Los inconvenientes son el coste, infección y dolor asociado con los implantes a largo plazo.

Fracturas del condilo medial o lateral con configuración espiral y con desplazamiento en la superficie articular deben ser tratadas quirúrgicamente si queremos mantener la funcion atletica de nuestro caballo.

Estas fracturas pueden ser tratadas de diferente manera:

Bajo anestesia general, usando multiples tornillos en efecto lag, usando una placa de compresion dinamica combinada con tornillos en efecto lag o usando dos placas de compresión dinamica

.El mayor riesgo de reparar esta fractura bajo anestesia general es el posible fallo del implante durante la recuperación de la anestesia y que ocurra una fractura catastrófica .

Un sistema de recuperación especial ha sido sugerido (piscina de recuparacion) para evitar este riesgo.

Este tipo de fracturas pueden ser reparadas con el caballo de pie, asi pues, eliminamos los riesgos asociados a la recuperación anestesica .

Las desventajas de la técnica van a ser el movimiento del caballo, mantener el campo quirúrgico esteril y una pobre visibilidad debido a la inadecuada retracción del tejido y a la dificultad para controlar la hemorragia.

Las fracturas en los cuatro casos eran incompletas pero con desplazamiento abaxial en la superficie articular, con lo que la fractura fue facil reducirla con la inserción del tornillo.

Los tornillos fueron insertados bajo control radiográfico y sin dificultad ya que el tercer metacarpo esta perpendicular al suelo lo que facilita la correcta posición del tornillo.

Un máximo de tres tornillos fueron insertados perpendiculares a la linea de fractura siendo suficientes para conseguir una compresión satisfactoria de la fractura.

La fractura fue reparada en todos los casos en el box de inducción, de esta forma las vibraciones creadas cuando el hueso es taladrado son absorbidas por la goma del suelo y el caballo no tiende a moverse.

Sedacion profuda junto con anestesia local fue suficiente para realizar la cirugía ya que el caballo paso de una cojera de no apoyo a apoyar normalmente después del bloqueo nervioso, asi que el caballo no debe ser movido para evitar la propagación de la fractura o aumentar el desplazamiento en la superficie articular.

Buena calidad de las radiografias es fundamental para evaluar la propagación de la fractura asi como la identificación de fracturas secundarias.

En conclusión es de nuestra opinión que reparacion de fracturas longitudinales bajo sedacion y anestesia local es una técnica viable que elimina el riesgo asociado con la recuperación de estos pacientes de la anestesia general.

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# UTILIDAD DE LA GAMMAGRAFÍA EN EL DIAGNÓSTICO DE COJERAS EN ÉQUIDOS. 4 CASOS CLÍNICOS DE FRACTURAS PRODUCIDAS POR ESTRÉS.

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## INTRODUCCIÓN

El uso de la medicina nuclear como técnica diagnóstica de cojeras en los équidos es relativamente reciente, siendo cada vez mas frecuente su utilización entre los caballos de deporte.

Dicha técnica nos ayuda a diagnosticar diversos casos de cojeras cuya procedencia hasta el momento era difícil discernir. La gammagrafía es utilizada en la valoración de determinadas alteraciones radiológicas, en el diagnóstico de cojeras en animales que no presentan evidencias radiográficas, en la evaluación de cojeras de origen desconocido, en la evaluación de zonas de difícil acceso a otras técnicas diagnósticas, como es el caso de la pelvis; así como en la evaluación de animales que presentan un rendimiento poco satisfactorio.

## MATERIAL Y MÉTODOS

En la presente Comunicación se ha utilizado una gammacamara. Como radioisótopo se ha empleado tecnecio 99 meta-estable, cuya vida media es de seis horas. Dicho radioisótopo se une a un grupo polifosfonato (HDP) obteniéndose así un radio-fármaco que presenta gran afinidad por los tejidos óseos activos y que emite radiación en forma de rayos gamma.

La técnica es relativamente sencilla y consta de dos pasos:

- Inyección del radio fármaco en el animal.
- Detección del radio fármaco y análisis de su distribución.

Se describen tres fases a medida que el tecnecio se distribuye a traves del organismo:

Fase 1: (fase inicial o fase vascular): que tiene lugar inmediatamente después de la inyección del tecnecio.

Fase 2: segunda fase que afecta a los tejidos blandos y que se desarrolla entre los 2 y 10 minutos postinyección. En ella el radio fármaco se distribuye a través del líquido extracelular.

Fase 3: (fase ósea o fase retardada): en la que el radio fármaco es captado por el tejido óseo, mostrando una mayor predilección por las zonas de mayor actividad metabólica. Tiene lugar 1,30 - 2 horas postinyección.

## RESULTADOS

Los casos que nos disponemos a presentar pertenecen todos ellos a la Fase 3 o fase ósea.

### Caso clínico Nº 1

Anamnesis: Yegua de cuatro años PSI en entrenamiento para carreras.

La yegua presentaba una cojera de grado moderado que afectaba al miembro posterior derecho, valorándose en 3/5 al trote en línea recta. No se evidenciaron signos de atrofia muscular en la pelvis ni alteraciones a la palpación del miembro afectado.

Hallazgos de gammagrafía: Se procedió a la realización de un escáner óseo de la pelvis y miembros posteriores, en el que se observó un intenso incremento de la actividad ósea a nivel de tercio distal de la tibia de la extremidad posterior derecha, así como de la región correspondiente a la zona media del peroné del miembro izquierdo. De igual modo, la actividad ósea se encontraba moderadamente incrementada en el cóndilo medial del segundo metatarsiano de la extremidad posterior izquierda.

Posteriormente se procedió a la realización de un examen radiológico de las tres zonas afectadas en el que se encontraron los siguientes hallazgos: la existencia de una fractura crónica en el tercio medio del peroné izquierdo con un callo óseo evidente, no observándose ninguna alteración en las otras dos regiones.

Conclusión:

-Fractura por stress sin desplazamiento de la zona distal de la tibia derecha.

-Fractura antigua del peroné izquierdo.

-Incremento del proceso de remodelación ósea del cóndilo medial del tercer metatarsiano izquierdo.

Resolución: Como ha sido descrito previamente en diversos estudios, en este caso se realizo un tratamiento conservativo. Normalmente es aconsejable para el animal un periodo de reposo en el box que permita la curación del hueso antes de comenzar un programa de ejercicio controlado que incorpore los aires de paso y trote.

En este caso se recomendaron al propietario tres o cuatro meses de reposo, periodo de tiempo que se estimó necesario para corregir la fractura de la tibia así como los problemas que afectan al peroné y al tercer metatarsiano. Es aconsejable que la yegua permanezca en la cuadra durante cuatro semanas y después pasar a un pequeño prado durante tres meses antes de que comience el entrenamiento de nuevo.

**Caso clínico Nº 2**

Anamnesis: Este segundo caso se trata de un macho castrado de seis años P.S.I. en entrenamiento para carreras.

El caballo presentaba una leve cojera que afectaba al miembro posterior izquierdo, valorándose en 1/5 al trote en línea recta y siendo ligeramente positivo a los test de flexión del menudillo y de la babilla. El animal Manifestaba un dolor evidente a la palpación en la zona derecha de la pelvis así como en la zona sacroileaca. La pelvis se presentaba simétrica y no se evidenciaron signos de atrofia muscular en ella.

En el examen rectal de la pelvis no se pudo evidenciar ninguna alteración de la zona lo que se pudo confirmar mediante examen ecográfico.

Hallazgos de gammagrafía: Se procedió a la realización de una gamma grafía de la pelvis y extremidades posteriores en la que se observó un marcado incremento de la actividad ósea en la zona proximal del tercer metacarpiano izquierdo, no observándose ningún cambio significativo que afectara a la pelvis.

Posteriormente se procedió a la realización de un examen radiológico de la zona afectada evidenciándose la presencia de una fractura de unos dos centímetros de longitud con zonas de esclerosis alrededor. En este area el patron trabecular se encontraba alterado como consecuencia de la esclerosis presente en el cortex.

Conclusión: -Presencia de una fractura por estres en la zona proximal del tercer metatarsiano.

Resolución: La mayoría de las fracturas producidas por estrés del metatarso suelen llegar a su completa curación mediante la utilización de un tratamiento conservativo consistiendo este en reposo, aunque los procedimientos quirúrgicos reducen el tiempo necesario para la curación completa del animal. En este tipo de patologías se suelen utilizar tratamientos de hidroterapia y se recomienda la administración de antiinflamatorios para reducir el dolor que presenta el animal en los primeros momentos. Se aconseja la realización de posteriores exámenes bien radiográficos o mediante escáner óseo tras un mínimo de ocho semanas de reposo.

En el presente caso, que nos ocupa tras un periodo de reposo en el box de seis meses, el animal vuelve a la clínica sin signos de cojera al trote ni dolor en la zona de la pelvis. En el escáner óseo se confirmó una reducción de un 95% de la actividad ósea presente en la "cana". Se aconseja que el animal comience a hacer ejercicio al paso durante un periodo de seis semanas pudiendo volver gradualmente al entrenamiento.

**Caso clínico Nº 3.**

Anamnesis: Semental de 13 años de edad P.S.I.

Presenta una cojera que no se evidencia al paso pero si al trote de 1/5 en la extremidad anterior derecha, no detectándose ninguna anomalía a la palpación de la misma.

Hallazgos de gammagrafía: Se decide la realización de gamma grafía de ambas extremidades anteriores en la que se pudo observar un incremento de la actividad ósea en el tercio medio del radio derecho.

Una vez localizada la lesión se realizaron placas radiológicas de la zona afectada no observándose ninguna alteración a excepción de un ligero patrón trabecular inusual del cortex del radio derecho al compararlo con el de la otra extremidad.

Conclusión: Fractura por estrés en la zona proximal del radio derecho.

Resolución: Este tipo de fracturas se han tratado únicamente mediante reposo en box. Normalmente con un periodo de reposo de un mes suele ser tiempo suficiente para el establecimiento de la normalidad en el animal, pero normalmente se suelen aconsejar la realización de posteriores reexámenes para tener una completa seguridad.

En el caso que nos ocupa se aconsejó la realización de ejercicio controlado del animal, comenzando con paseo durante 10 minutos e incrementando el tiempo poco a poco, siempre que el animal no desarrollara una cojera marcada. Tras 7 días se aconsejó comenzar con 5 minutos de trote y en un periodo de tres semanas repetir el escáner óseo para ver si presentaba mejoría. Ésta fue confirmada pues se observó una reducción del 60-70% de la actividad ósea.

**Caso clínico Nº 4.**

Anamnesis: Macho de tres años de edad P.S.I.

El animal presentaba una historia de una cojera gradual con patentes signos de dolor en la ultima semana. El caballo manifestó una cojera de 2/5 en la extremidad posterior izquierda y a la palpación se detecto una ligera atrofia muscular de la cadera izquierda. También se pudo observar una zona de ligera efusión de líquido en la zona distal de la cana del miembro posterior izquierdo. En los test de flexión los resultados fueron negativos para la flexión de la babilla y ligeramente positivos a la flexión del menudillo.

Hallazgos de gammagrafia: Se procedió a la realización de un escáner óseo de la pelvis y extremidades posteriores en el que se observó un claro e intenso aumento de la actividad ósea en la zona correspondiente a las alas del ileum izquierdo.

Conclusión: Fractura por estres del ala del ileum izquierdo.

Resolución: Como ha sido descrito en numerosos estudios el tratamiento de elección es reposo durante un periodo de diez a dieciséis semanas, aunque el tiempo requerido por cada animal se debe establecer en base a los exámenes seriados que se realicen del animal. Una vez la superficie ósea es continua y de apariencia normal mediante ultrasonografía, se puede restablecer la práctica de ejercicio, lo que suele observarse aproximadamente tras doce semanas de reposo aproximadamente, aunque esto depende del grado de la lesión inicial así como de la edad del animal.

En este caballo se recomendó su estabulación durante dos meses para pasar, posteriormente, a pastar en un prado pequeño. Se debe tener en cuenta que el tiempo necesario para la resolución de este tipo de fracturas a veces puede llegar a ser superior a un año.

## CONCLUSIONES.

Con la presentación de estos ejemplos de fracturas por estrés se ha pretendido mostrar la gran utilidad de esta técnica de diagnóstico por imagen, siendo a veces el único método que se puede utilizar para llegar a emitir un diagnóstico correcto.

Las fracturas por estrés que afectan a la tibia son una de las causas relativamente comunes de cojeras en PSI y pueden llegar a ser una de las mas difíciles de diagnosticar debido a que no se encuentran afectados por los bloqueos anestésicos regionales o intraarticulares. Por ello por lo que esta técnica es de tanta utilidad en estos casos. Por otro lado, periodos de confinamiento del animal extremadamente prolongados se encuentran contraindicados debido a la inactividad que se produce de todo el esqueleto, de forma que cuando el animal vuelve al ejercicio puede llegar a desarrollar otros problemas ortopédicos como dolor en la zona de la tibia, desmitis del ligamento suspensor y fracturas por estrés en otras zonas del organismo.

La gamma grafía es una técnica de gran utilidad a la hora de detectar fracturas que afectan al tercer metatarsiano debido al obvio incremento de la actividad ósea, superior a un 600%, que puede ser detectada. Con frecuencia se han requerido la realización de múltiples radiografías para demostrar la presencia de una fractura en esta zona, siendo a veces imposible confirmar la existencia de fracturas mediante la radiología. Es en estos casos cuando la gamma grafía es considerada una técnica de gran utilidad.

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## RESUMEN

El uso de la medicina nuclear como técnica diagnóstica de cojeras en los équidos es relativamente reciente, siendo cada vez mas frecuente su utilización entre los caballos de deporte.

El escáner óseo posee una sensibilidad muy superior a la de la radiología, pudiendo detectar la presencia de alteraciones antes de que se desarrollen los cambios morfológicos visibles mediante ultrasonografía o placas radiográficas, esto es debido tanto al estudio metabólico como dinámico que se realiza del tejido.

En la presente Comunicación se ha utilizado una gammacamara. Como radioisótopo se ha empleado tecnecio 99 meta-estable, que se une a un grupo polifosfonato (HDP).

La técnica es relativamente sencilla y consta de dos pasos, inyección del radio fármaco en el animal y detección del radio fármaco con el consiguiente análisis de su distribución.

La fracturas por estrés son muy comunes en caballos PSI; debido a la alta incidencia de este tipo de patologia pensamos que seria interesante la presentacion de cuatro casos clinicos de fracturas por estrés afectando a distintas regiones anatomicas, como son: tibia, metatarso, radio y pelvis. Y observar su posterior recuperacion.

Con la presentación de estos ejemplos de fracturas por estrés se ha pretendido mostrar la gran utilidad de esta técnica de diagnóstico por imagen, siendo a veces el único método que se puede utilizar para llegar a emitir un diagnóstico correcto.

# UTILIDAD DE LA ECOGRAFIA EN EL DIAGNOSTICO Y TRATAMIENTO DE PROBLEMAS DE DORSO EN EL CABALLO (I): (ABORDAJE DORSAL DE LA REGION TORACOLUMBAR)

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## Resumen

En este estudio se describe la técnica de ecografía (abordaje dorsal) de la región toracolumbar del caballo, así como la utilidad diagnóstica y terapéutica que presenta.

La complejidad anatómica de la región del dorso, así como la enorme funcionalidad a la que ésta se ve sometida en el caballo de deporte hacen necesaria la utilización de sofisticadas técnicas de diagnóstico capaces de detectar de forma precisa la existencia de patologías que afecten a dicha región. En este sentido, la ecografía constituye un método ideal para el diagnóstico de numerosos trastornos de dorso, al mismo tiempo que se convierte en herramienta imprescindible para la realización de determinadas maniobras terapéuticas (infiltraciones) de forma correcta.

De acuerdo con numerosos autores, son relativamente frecuentes las lesiones del dorso del caballo que afectan a la parte caudal de la región torácica y a la región lumbar. En el presente trabajo describiremos brevemente la técnica de diagnóstico ecográfico de la región toracolumbar en su abordaje dorsal, así como la maniobra de infiltración terapéutica.

En primer lugar, y como paso previo al **estudio ecográfico** del dorso del caballo, debemos considerar la realización de una limpieza exhaustiva de la superficie de escaneo, con la finalidad de evitar la interposición de cualquier resto de grasa y suciedad al paso de los ultrasonidos que pueda dar lugar a la formación de artefactos, para lo que también se recomienda el rasurado de dicha superficie.

Una vez limpia el área de escaneo, el siguiente paso es favorecer la transmisión de ultrasonidos, para lo que aplicaremos gel de ultrasonidos o bien bañaremos constantemente la superficie de contacto con la sonda ecográfica con alcohol.

Otro aspecto importante a considerar, previamente a la realización del examen ecográfico propiamente dicho, es la correcta identificación de las estructuras a valorar o tratar. Para ello, procederemos al marcaje de los extremos craneales de las últimas apófisis espinosas torácicas y lumbares, comenzando en la 6ª vértebra lumbar y continuando en sentido craneal. Todo ello sirve por un lado para establecer los distintos niveles topográficos del ligamento supraespinoso, y por otro para situar con exactitud las articulaciones entre apófisis articulares, siendo ambas estructuras las más frecuentemente afectadas en los procesos patológicos de la región.

El estudio ecográfico debe realizarse en un ambiente tranquilo y cómodo para el caballo, siguiendo un patrón de escaneo en el que de forma metódica ecografiemos todos y cada uno de los segmentos vertebrales objeto de estudio.

Ocupándonos del examen ecográfico en sí, éste debe comenzarse utilizando sondas de ultrasonidos de frecuencias que oscilen entre los 6.0 y 7.5 MHz, pudiendo acceder así hasta profundidades de 6-10 cm. No obstante, el estudio de estructuras localizadas a mayor profundidad requiere sondas de menor frecuencia (5.0, 4.0, etc MHz) que nos permitan acceder hasta profundidades de 15-20 cm para su diagnóstico.

En aquellos casos en los que existe indicación terapéutica, se procede a la **infiltración** de la medicación seleccionada. Para ello, una vez conseguido un adecuado grado de relajación y analgesia en el caballo, y preparada asépticamente la zona de trabajo, se introduce una aguja de longitud y calibre apropiados, siguiendo paso a paso su camino a través de los planos anatómicos identificados hasta alcanzar la zona lesionada.

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# UTILIDAD DE LA ECOGRAFIA EN EL DIAGNOSTICO Y TRATAMIENTO DE PROBLEMAS DE DORSO EN EL CABALLO (II) : ABORDAJE DORSAL DE LA REGIÓN SACROILIACA

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## Resumen

En el presente trabajo se hace un breve recordatorio de la anatomía ecográfica de la región sacroilíaca del caballo, así como también se consideran los aspectos más relevantes de la técnica de ecografía (abordaje dorsal) de esta zona y la utilidad diagnóstica y terapéutica que nos ofrece.

El conocimiento exhaustivo de las estructuras anatómicas de la región sacroilíaca capacita al examen ecográfico en su abordaje dorsal como método diagnóstico de primer orden en la detección de lesiones de dorso que afectan a la citada región en sus porciones más superficiales. Por otro lado, en la última década, son cada vez más frecuentes los diagnósticos de lesiones de articulación sacroilíaca en los que la ecografía de superficie es un medio auxiliar de enorme interés para la realización de infiltraciones terapéuticas.

El estudio ecográfico debe realizarse en un ambiente tranquilo y cómodo para el caballo, logrando un adecuado nivel de relajación y analgesia. Una vez preparada asépticamente el área de escaneo, el siguiente paso es favorecer la transmisión de ultrasonidos, para lo que aplicaremos gel de ultrasonidos o bien bañaremos constantemente la superficie de contacto con la sonda ecográfica con alcohol. Ocupándonos del examen ecográfico en sí, éste debe comenzarse utilizando sondas de ultrasonidos de frecuencias que oscilen entre los 6.0 y 7.5 MHz. No obstante, el estudio de estructuras localizadas a mayor profundidad requiere sondas de menor frecuencia (5.0, 4.0 MHz, etc.).

Mediante este examen se obtienen imágenes transversales, sagitales y parasagitales del extremo dorsal de las tuberosidades sacras de ambos coxales, de las apófisis espinosas sacras, de la porción funicular del ligamento sacroilíaco dorsal y de la musculatura de la zona entre la que

destaca la porción caudal del músculo longísimo del dorso. La ecografía permite así el diagnóstico lesional de estas estructuras y, si es preciso, la infiltración terapéutica guiada. Otra aplicación de la ecografía de esta región es la de localizar el sitio de punción adecuado y el seguimiento del primer trayecto del recorrido que sigue la aguja en la infiltración de la articulación sacroilíaca. En el presente trabajo se realiza un breve recordatorio de las estructuras anatómicas de interés para dicha infiltración, así como una descripción de la técnica de abordaje que utiliza el borde craneal de la tuberosidad sacra del lado opuesto y el trocánter mayor del mismo lado a la articulación afectada como referencias.

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# COJERAS DE EXTREMIDADES ANTERIORES MÁS MANIFIESTAS EN EL CÍRCULO EXTERIOR

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Uno de los problemas del estudio de las cojeras en el caballo es que la gran mayoría de los alteraciones locomotoras producen modificaciones del paso comunes e inespecíficas, lo que dificulta la localización de la fuente del dolor y hace que la lista de diagnósticos diferenciales sea muy amplia. Sin embargo, el estudio detallado de las características de la cojera, en los movimientos del caballo cuando trabaja en círculo, puede mostrar algunos datos de interés que nos ayuden a reducir la lista de enfermedades posibles y a concretar mucho más los diagnósticos diferenciales

Si bien la gran mayoría de las cojeras de las extremidades anteriores tienden a producir una cojera más marcada cuando el animal se mueve en círculo, con la mano afectada por dentro, existen una serie de cojeras que tienden a manifestar lo contrario, es decir, mayor grado de cojera cuando la extremidad afectada se mueve por el círculo exterior. En ocasiones son movimientos sutiles manifestados en forma de acortamiento del paso cuando la mano afectada va por fuera y movimientos a paso inconstante, sin ritmo, cerrándose en las vueltas (volcándose) o con dificultad para mantener el aire de trabajo, cuando la mano afectada se mueve por dentro.

En base a estas observaciones, en el presente trabajo pretendemos mostrar una serie de casos clínicos, observados en el último año en el Hospital Clínico Veterinario de la Universidad de Córdoba, caracterizados por producir cojeras con los signos clínicos descritos, u otros similares. Entre las patologías capaces de producir cojeras de este tipo se citan hemos observado las siguientes:

**Lesiones de la cara dorsomedial del carpo:** En casos de osteoartritis suele aparecer inflamación de la cara dorsomedial del hueso carporadial y hueso tercer carpiano dando este tipo de cojera y acompañandose de una abducción del miembro.

**Lesiones de la cara palmar del carpo:** En las que el caballo manifiesta la cojera cuando alarga el miembro, de ahí que manifieste más la cojera al moverse la extremidad por fuera.

**Desmitis del origen, o de la rama medial del ligamento suspensor:** Estas, además tienden a ser más acusadas cuando el animal trota en suelo blando especialmente las del cuerpo. En ocasiones son tan sutiles que son solamente apreciados por jinetes expertos.

**Lesiones de la cara medial del menudillo:** Las osteoartritis en fase inicial con mayor afectación de la cara medial del menudillo, especialmente si van acompañadas de entesitis de los ligamentos colaterales mediales.

**Sobrehuesos en fase inicial en la tróclea medial de la primera falange:** Tienden a manifestarse especialmente cuando el animal trota en suelo duro.

**Fracturas sagitales de la cara medial de la tercera falange**. En algún caso resulta difícil reconocer la mano afectada por el movimiento irregular del animal al trote.

**Enfermedad del navicular** con lesión de la superficie flexora y afectación del tendón flexor digital profundo (forma tendinosa). Si bien la enfermedad del navicular produce casi siempre una cojera mas acusada por el círculo interno las que se manifiestan de esta forma suelen ser más pronunciadas en el círculo exterior.

El trabajo concluye que el reconocimiento de este tipo de movimientos anormales es muy interesante, sobretodo en la porción más distal del miembro, ya que permite realizar anestesias solamente de los ramos mediales y llegar a una localización mucho más exacta de la zona de lesión.

# VALOR PRONÓSTICO DE ALGUNOS SIGNOS RADIOGRÁFICOS DEL TARSO EN EL EXAMEN PRECOMPRA DEL CABALLO

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En el presente trabajo pretendemos mostrar los posibles hallazgos radiográficos presentes en el tarso durante un examen de precompra, y el valor de cada uno de ellos a la hora de enjuiciarlo en el marco de un examen de precompra. De igual forma pretendemos mostrar nuestra experiencia sobre las mejores proyecciones radiográficas, en función de los condicionantes económicos del estudio de precompra.

En estudio radiográfico del tarso se deben de realizar al menos dos proyecciones radiográficas, con una diferencia de angulación de 90º entre ambas. Para ello nosotros recomendamos el uso de las proyecciones oblicuas: dorsolateral-plantaromedial y dorsomedial-plantarolateral. Con la inclusión de una tercera proyección lateromedial se obtiene una información adicional muy interesante en algunos casos. Posiblemente la proyección que aporte menos información sea la dorsoplantar si bien es de muy recomendada para algunos casos específicos donde tengamos duda sobre algún signo radiográfico de las proyecciones anteriores.

***Signos radiográficos a valorar especialmente en un examen de precompra:***

***En la proyección lateromedial.***

- osteofitosis borde dorsoproximal hueso metatarsiano III: favorable.
- pérdida del espacio articular de articulaciones distales del tarso: reservado.
- osteocondrosis del labio lateral del astrágalo: según lesión de favorable a desfavorable.
- osteocondrosis del labio medial del astrágalo: normalmente favorable si se trata de ligeras depresiones del labio.

***En la proyección Dorsoplantar***.

- osteoartritis de las articulaciones distales del tarso: según el signo radiográfico detectado.
- osteocondrosis del maleolo lateral de la tibia: desfavorable.
- osteopatías de inserción: mineralizaciones distróficas y entesiofitos: reservado.

**En la proyección Dorsolateral-plantaromedial oblicua (oblicua externa).**

- osteoartritis de las articulaciones distales del tarso: según signo radiográfico.

**Dorsomedial-plantarolateral oblicua (oblicua interna).**

- osteoartritis de las articulaciones distales del tarso: según signo radiográfico.
- osteocondrosis de la eminencia intermedia de la cóclea tibial en función de la edad del animal y su dedicación. Favorable como caballo de alta competición desfavorable como reproductor.
- osteocondrosis del labio lateral del astrágalo: según el grado lesión, de favorable a desfavorable.

# CASO CLINICO: FRACTURA ATLANTO-AXIAL EN UN POTRO Y SU EVOLUCION NEUROLÓGICA

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## INTRODUCCION

Se presenta un caso clínico de un potro al que se le diagnostica una fractura vertebral e inestabilidad cervical asociada. La clínica neurológica que muestra es progresiva y propia de una lesión cervical craneal.

## HISTORIA

Es referido a la Unidad Equina del HCV de la UAB un potro PRE de 6 meses de edad con historia de traumatismo con herida en la zona cervical craneal derecha de 10 días de duración.

## EXAMEN CLINICO Y PRUEBAS COMPLEMENTARIAS

El examen neurológico no reveló anormalidades. A la inspección presentaba un herida de 2 cm de diámetro con secrección hemorrágico-purulenta ventralmente al área de proyección del ala derecha del atlas. En la radiografía cervical la imagen obtenida era compatible con fragmentación del ala derecha del atlas.

En los días posteriores a su ingreso el potro comenzó a mostrar una dificultad progresiva en la locomoción, comenzando por ataxia de las extremidades anteriores y continuando por el tercio posterior.

El análisis del líquido cefalorraquídeo no reveló anormalidades. En la mielografía se observó marcada compresión de la columna de contraste dorsal sobre C1-C2.

El diagnóstico fue compresión medular a nivel de la articulación atlanto-axial debido a fractura vertebral e inflamación secundaria.

En la necropsia se halló fractura del ala derecha del atlas y de la apófisis articular anterior derecha del axis.

## CONCLUSION

El caso presentado resulta interesante desde el punto de vista de la clínica neurológica, de presentación tardía y progresiva, asociada a la inestabilidad cervical provocada por la fractura atlanto-axial y la consecuente subluxación atlanto-axial.

# ILEO POSTOPERATORIO: UN CASO CLÍNICO

## Introducción

El Íleo postoperatorio, la alteración funcional de la motilidad del tramo intestinal proximal, es una de las complicaciones postoperatoria más común en caballos operados de cólico. Isquemia intestinal, distensión, desequilibrio electrolítico, peritonitis, endotoxemia, manipulación traumática (intensa) del intestino, resección y anastomosis y anestesia son los principales factores predisponentes. A pesar de la evolución en los tratamientos, al día de hoy el íleo postoperatorio representa el 40% de la mortalidad de los cólicos quirúrgicos en el postoperatorio.

## Caso clínico

### Historia

Un caballo PRE, de 7 años y castrado, fue ingresado en el Hospital Veterinario Sierra de Madrid con signo de cólico durante las ultimas 12 horas. En el periodo previo, había estado tratado con dos dosis de 500 mg de Flunixin Meglumine por el veterinario remitente. A la palpación rectal se detectaba la presencia de asas de intestino delgado distendido, por lo que se decidió remitir el animal al hospital.

### Examen clínico

A su llegada al hospital, el caballo se encontraba deprimido y sin signos de dolor, con mucosas congestivas, frecuencia cardiaca de 84 ppm y respiratoria de 36 rpm, temperatura rectal de 38,7 °C y se obtuvieron 8 litros de reflujo nasogastrico. El hemograma y la bioquímica sanguínea se encontraban dentro de los limites normales excepto el valor hematocrito de 57% y las proteínas plasmáticas totales de 8,4 g/dl. El liquido abdominal, con aspecto macroscópico y recuento celular normal, presentaba PT de 2,8 g/dl. A la palpación rectal se hallaron asas de intestino delgado distendidas confirmadas posteriormente mediante ecografía.

### Diagnostico

En base a estos hallazgos el posible diagnostico diferencial era entre problemas de naturaleza obstructiva no estrangulantes, o de naturaleza inflamatoria como la enteritis anterior.

### Tratamiento y Evolución

Se instauró un tratamiento medico con antibióticos de amplio espectro (Ceftiofur 4 mg/kg i.v. cada 6 horas), antiinflamatorios no esteriodeos (Flunixin Meglumine 250 mg i.v. tid), fluidoterapia con

solución isotónica poliionica (Ringer Lactato) suplementada con gluconato de calcio y sondaje nasogastrico cada 2 horas.

En las primeras 24 horas de tratamiento el animal experimentó una ligera mejoría clínica, volviendo el hematocrito y las proteínas totales a valores normales. La frecuencia cardiaca y respiratoria disminuyeron a 60 ppm y 24 rpm. El caballo seguía sin dolor, pasando heces y el reflujo gástrico bajó desde 8 litros cada 2 horas a 2 litros cada 3 horas. El examen rectal seguía igual con asas de intestino delgado ligeramente distendidas.

En las siguientes 48 horas el paciente empeoró gradualmente y a las 72 horas de su ingreso al centro presentaba ligera hipertermia (38,9), 52 ppm y 10 litros de reflujo cada 3 horas. La abdominocentesis reveló un recuento celular de 6,100 y proteínas totales de 3g/ml. Dada la evolución el caballo se optó por una laparotomía exploratoria.

## Cirugía

Bajo anestesia general inhalatoria se practicó una laparotomía exploratoria en la que se evidenció una impactación de íleon. Una vez valorada la válvula ileocecal, y la dificultad en el transito de contenido alimenticio a través de dicha válvula, se optó por la realización de un "by pass" ileociecal parcial.

## Evolución Postoperatoria

Tras la cirugía, se mantuvo la terapia iniciada anteriormente con la adjunta de metronidazol (20 mg/kg p.r. TID) y lidocaina como estimulante de la motilidad intestinal (1,3 mg/kg en bolo, 0.05 mg/kg/min en infusión intravenosa.

En las primera 24 horas la evolución del paciente fue favorable: se suspendió la administración de lidocaina y se retiró la sonda nasogastrica. El segundo día se le ofreció pequeñas cantidades de agua de bebida cada 2 horas y a las 48 horas 0,25 gramos de pienso compuesto.

A las 72 horas tras la intervención, el caballo presentaba ligero dolor, borborigmos intestinales disminuidos, frecuencia cardiaca aumentada, 12 litros de reflujo gástrico. El hematocrito subió a 45% y las proteínas totales a 7 g/dl. La palpación rectal y la ecografía intestinal evidenciaron asas intestinales distendidas y sin motilidad. Se le administró metoclopramida en infusión continua en dosis de 0,04 mg/kg/hr. A partir del sexto día tras la cirugía disminuyó notablemente el reflujo gástrico, se normalizaron las constantes vitales del caballo, el hemograma y la bioquímica sanguínea.

## Discusión

Aunque el íleo adinámico probablemente ocurra de forma temporál en todos los caballos tras una laparotomía exploratoria, se habla de íleo postoperatorio solo en aquellos casos en los que se manifiestan signos clínicos asociados a una alteración de la motilidad gastrointestinal prolongada en el tiempo. Los síntomas clínicos del íleo son debidos al acumulo progresivo de gas y fluido en el tracto intestinal producido por la perdida de la motilidad propulsiva. En 12 a 36 horas, por flujo retrogrado, se produce distensión gástrica, manifestada por el animal con depresión y dolor cólico. Los borborigmos intestinales normalmente son disminuidos o ausentes. El rápido acumulo de fluidos en intestino produce una hipovolemia con compromisión del sistema cardiovascular: aumenta la frecuencia cardiaca, el tiempo de relleno capilar, el valor hematocrito y la concentración de proteinas totales. La concentración del cloro y del potasio en el plasma disminuyen. El reflujo es siempre constante y la descompresión del estomago mediante sondaje nasogastrico suele producir una mejoría de los signos clínicos.

La progresión del alimento en trato intestinal se realiza mediante contracciones perfectamente coordinadas tanto en el espacio como en el tiempo de la musculatura lisa de la pared del intestino. Tal actividad es regulada por el sistema nervioso vegetativo y en particular el sistema nervioso simpático es principalmente inhibidor de la motilidad, mientras el parasimpático es activador. Una hiperestimulación del simpático y la hipoactividad del parasimpático, así como un daño directo a las células musculares de la pared intestinal pueden producir el íleo postoperatorio.

El tratamiento del íleo postoperatorio se realiza mediante prevención de los factores predisponentes, fluidoterapia, constante monitorización de los electrolitos, antibioterapia, AINES, y el empleo de sustancias farmacológicas que actúan sobre la motilidad intestinal.

En termino general los agonistas a-adrenergicos disminuyen la motilidad intestinal, mientras los antagonistas a-adrenergicos y los cholinomimeticos aumentan la motilidad.

Los medicamentos más utilizados como estimulantes de la motilidad intestinal son:

Eritromicina: es un antibiótico macrolide que en dosis inferior de la requerida por el efecto antimicrobiano promueve la motilidad gástrica y la coordinación intestinal. Actúa como agonista de la motilina, una hormona que incrementa la actividad contráctil del estomago y del intestino delgado y aumenta la liberación de acetilcolina. Se utiliza en dosis de 2,2 mg/kg en 1 litro de suero administrado en 60 minutos cada 6 horas. El efecto disminuye con tratamientos repetidos y puede causar dolor abdominal.

Metoclopramida: es una benzamida que actúa mediante varios mecanismos: es un antagonista de la dopamina, aumenta la liberación de la acetilcholina y es un bloqueante adrenergico. Se administra, preferiblemente en dosis de 0,04 mg/kg/hr en infusión continua. Puede causar efectos colaterales extrapiramidales como excitación y sudoración.

Cisapride: es una benzamida que promueve la liberación de acetilcolina. En caballos sanos aumenta la amplitud de la contracción gástrica y estimula la motilidad del intestino delgado, colon y porción ileociegal y ciecocolica. La mayor limitación en su empleo es que es disponible solo en preparaciones comerciales destinadas a la vía oral.

Lidocaina: actúa reduciendo la concentración de las catecolaminas circulantes, activando directamente las células musculares y disminuyendo la inflamación de la pared intestinal mediante la inhibición de la liberación de prostaglandinas y de la migración de los neutrofilos. Se utiliza mediante un bolo inicial de 1,3 mg/kg i.v. administrado en 5 minutos, seguido por 0.05 mg/kg/hr en solución salina en 24 horas.

No se aconseja su utilización en caso de posible contaminación durante la cirugía.

Neostigmina: es un inhibidor de la cholinesterasa que prolonga la actividad de la acetilcolina. En caballos sanos aumenta el tiempo de vaciamiento gástrico y disminuye la motilidad intestinal a nivel del intestino delgado, mientras la incrementa a nivel de flexura pélvica. La dosis es de 0,0044 mg/kg s.c.o i.v. cada 30-60 minutos. Puede producir dolor abdominal.

Yohimbina y Acepromacina: son antagonistas a-adrenergicos. La Acepromacina se administra en dosis de 0,01 mg/kg i.m. cada 4 horas. La principal contraindicación a su empleo es la hipoten-

sión que provoca. La yohimbina se ha utilizado en dosis de 0,15 mg/kg i.v. repetida a las horas 1,4,7 y 10 de la cirugía en modelos experimentales.

Bethanecol: es un agonista cholinergico muscarinico que estimula la actividad de las células musculares intestinales. En un modelo experimental en dosis es de 2,5 mg s.c. administrada a las 2 y 5 horas de la cirugía aumenta el transito intestinal. Los efectos colaterales son espasmos intestinales, diarrea y hipersalivación.

Dexpanthenol: es un precursor del acido pantotenico y actúa incrementando la producción de acetilcolina. Se emplea en dosis de 2,5 gr/500 kg i.v.o i.m. cada 6 horas. Puede producir los mismos efectos colaterales del Bethanecol._

Los procineticos más utilizado en la practica veterinaria, en caso de íleo postoperatorio son la neostigmina, la eritromicina, la lidocaina y la metoclopramida.

## Conclusión

A pesar de la elección del procinetico, una técnica quirúrgica correcta y el cuidado postoperatorio son fundamentales por conseguir un buen éxito en estos casos. Una correcta fluidoterapia para compensar las enormes perdidas de fluidos, corregir el desequilibrio electrolítico, una terapia antibiótica de amplio espectro, el empleo de antiilfamatorios no esteriodeos y el sondaje nasogastrico son los puntos críticos del tratamiento de estos caballos. El cuidado intensivo que requieren estos pacientes genera un gasto económico importante que, a veces, es el factor limitante en el tratamiento.

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# MODIFICACIONES DE LA BIOQUÍMICA PLASMÁTICA EN EQUINOS DURANTE LAS COMPETICIONES DE RAID

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## INTRODUCCIÓN

La competición de raid es una de las mayores exigencias a la que es sometido el caballo deportivo. Consecuentemente se presentan con frecuencia alteraciones del medio interno que comprometen en menor o mayor medida la vida del animal.

La bioquímica sanguínea es un método complementario ampliamente utilizado para el diagnóstico y el pronóstico de las alteraciones del medio interno. Sin embargo los valores considerados basales para un animal en reposo no coinciden con los obtenidos en un individuo sano luego de haber culminado un raid.

Las alteraciones en carreras de raid fueron estudiadas por varios autores (3), sin embargo, no se encuentran datos ajustados a la reglamentación actualmente vigente, como así tampoco estudios realizados en España, con las hostiles condiciones climáticas y geográficas imperantes. La mayoría de estos estudios fueron enfocados para comprender fisiología del esfuerzo prolongado más que para generar unos datos de referencia. Los objetivos del trabajo son entonces verificar modificaciones de diversos parámetros plasmáticos en equinos que son sometidos a un esfuerzo prolongado; y obtener datos que puedan ser utilizados como referencia normal en la interpretación de resultados bioquímicos en la clínica de este deporte.

## MATERIALES Y MÉTODOS

A fin de generar esta información básica faltante, se efectuó un relevamiento en un raid de 114 km realizado en Tuéjar (Campeonato de España Junior y JJ, 3-may-03) bajo la reglamentación de la Federación Ecuestre Internacional.

Se analizaron un total de 14 animales, de los cuales sólo 10 (6 hembras y 4 machos) concluyeron satisfactoriamente la prueba y no mostraron alteración alguna en los días subsiguientes por lo que sólo estos últimos fueron incluidos en el ensayo. Sus edades estaban comprendidas entre los 8 y 13 años, y eran de raza árabe, Anglo-árabe y Anglo-hispano-árabe. Las velocidades promedio a la que realizaron el recorrido están comprendidas entre 13 y 17 km/h.

Se tomaron muestras pre y pos ejercicio por punción yugular, que fueron heparinizadas y seguidamente centrifugadas para la extracción de plasma, el cual fue inmediatamente refrigerado para su posterior análisis dentro de las 48 hs siguientes. Las muestras pre ejercicio se obtuvieron el

día previo mientras que las pos ejercicio entre 5 y 15 minutos luego de superar el último control veterinario.

Se realizaron determinaciones de creatina quinasa (CPK/CK), aspartato aminotransferasa (GOT/AST), lactato deshidrogenasa (LDH), fosfatasa alcalina (FA), ácido úrico, creatinina, urea. Todas se efectuaron por espectrofotometría utilizando reactivos BioSystems designados para cada determinación.

Las temperaturas registradas durante el evento oscilaron entre 22 y 28º, y la humedad relativa fue de 49%.

El método estadístico empleado fue el análisis de la varianza simple.

## RESULTADOS Y DISCUSIÓN

Los resultados se exponen en la tabla 1:

| n = 10 | | | Pre ejercicio | | POS ejercicio | |
|---|---|---|---|---|---|---|
| | Unidad | Valores de referencia | Promedio | Desv Sta | Promedio | Desv Sta |
| CPK | U/l | 150 – 420 | 211,89 | 128,51 | 940,41* | 474,84 |
| GOT | U/l | 60 – 350 | 251,99 | 89,01 | 287,22 | 97,39 |
| LDH | U/l | Menor a 450 | 321,93 | 118,47 | 563,72 | 168,41 |
| FAL | U/l | 140 –400 | 258,88 | 96,09 | 398,18 | 118,88 |
| Creatinina | mg/dl | 1 a 2 | 1,03 | 0,17 | 1,67 | 0,62 |
| Urea | mg/dl | 10 a 30 | 25,27 | 6,69 | 42,02* | 8,19 |
| Ácido Úrico | mg/dl | 0.7 – 1.4 | 1,48 | 0,57 | 4,43* | 2,61 |

**Tabla 1**

*Diferencias con pre ejercicio ($p < 0.05$)

Aunque los valores elevados de enzimas plasmáticas se interpretan generalmente como prueba de necrosis celular, el ejercicio intenso realizado en forma prolongada también da por resultado la liberación de cantidades importantes de enzimas musculares. La difusión de las mismas al torrente circulatorio no necesariamente es consecuencia de una lisis celular, pudiendo producirse también por una producción excesiva, alteraciones en la inactivación y remoción, y mas comúnmente, por aumento de la permeabilidad de membrana. En nuestro trabajo encontramos una elevación de la concentración de las enzimas muestreadas, siendo este aumento significativo sólo para el caso de la CK. Probablemente la causa de elevación enzimática con el ejercicio se deba tanto a un aumento de la permeabilidad de membrana favorecido fundamentalmente por la temperatura y déficit energético, como así también a una apoptosis inducida por el ejercicio (fundamentalmente en las enzimas musculares). La velocidad de difusión depende de la concentración de la enzima, la localización dentro de la célula y del peso molecular de la enzima (5). De esta forma observamos que una enzima de relativamente pequeña, citoplasmática, y sumamente abundante, como la CK, alcanza valores significativamente superiores a los registrados antes de la competencia. Sin embargo los aumentos son menores cuando el peso molecular aumenta (LDH), o bien aumenta el peso molecular y su concentración celular es menor (FA), o en última instancia, cuando su ubicación es citosólica-mitocondrial (AST).

Los valores de urea y creatinina resultan de el equilibrio entre su producción y su eliminación renal. Contrariamente a la urea, la producción de creatinina es marcadamente mayor durante el ejercicio. Sin embargo, el aumento de la concentración plasmática de urea en este ensayo es proporcionalmente mayor al de la creatinina, llegando a tener significación estadística. Esto puede ser explicado porque al aumentar al ADH, la urea se reabsorbe en los túbulos colectores medulares, aumentando la hiperosmolaridad medular, y contribuyendo a la concentración urinaria. En definitiva la eliminación renal de urea se ve disminuída (1). Por lo tanto el valor de la urea plasmática como indicador de la función renal en un individuo deshidratado es limitado.

Encontramos en nuestro trabajo un incremento significativo de ácido úrico como consecuencia del ejercicio. El ácido úrico es un metabolito intermedio de la degradación de las purinas, siendo con gran diferencia el que sufre mayor aumento cuando se sobrecarga esta vía, al incrementarse la relación ADP-ATP. Paralelamente esto promueve la glucólisis anaeróbica, lo que justificaría la correlación entre lactato y ácido úrico descripta por varios autores (2). Se han publicado numerosos trabajos donde se evalúa la función antioxidante del ácido úrico durante el esfuerzo (4), incluso algunos investigadores correlacionan los niveles de ácido úrico la exigencia metabólica sufrida por el animal (2).

Las concentraciones de AST, LDH, FAL y creatinina, si bien mostraron aumentos, estos no llegaron a tener significación estadística, probablemente por el bajo número de muestras. Sería conveniente la realización de futuros ensayos con un número mayor de animales a fin de generar valores que puedan utilizarse como referencia en la clínica deportiva.

## CONCLUSIÓN

Las concentraciones plasmáticas de CK, urea y ácido úrico, se elevan como respuesta normal a un esfuerzo prolongado, lo que debe ser tomado en cuenta al interpretar resultados de individuos sometidos a un esfuerzo semejante.

## RESUMEN

Se estudiaron los valores plasmáticos de 10 caballos antes y después de una prueba de 114 km realizada en España bajo reglamentación de la FEI. Todos los animales finalizaron satisfactoriamente la prueba, y no mostraron alteraciones en los días siguientes. Se han determinado los valores de CK, AST, LDH, FAL, urea, creatinina y ácido úrico.

Después del esfuerzo se han registrado incrementos en todos los parámetros estudiados, siendo estos significativos solo en el caso de CK, urea y ácido úrico. Se discuten las posibles causas fisiológicas de estos incrementos.

Sería útil la realización de futuros ensayos con un número mayor de animales a fin de obtener valores que puedan utilizarse como referencia.

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# LA ECOGRAFÍA COMO TÉCNICA DIAGNÓSTICA DE PROBLEMAS PULMONARES EN EL CABALLO

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## Resumen

En el presente trabajo se hace una breve exposición de la técnica de ecografía pulmonar en el caballo, al mismo tiempo que se describe el aspecto ecográfico de las estructuras observadas durante el estudio ultrasonográfico del pulmón del caballo sano y los hallazgos ecográficos más característicos de los procesos patológicos que más comúnmente afectan a la cavidad pleural y a los pulmones en esta especie.

## Introducción

La elevada frecuencia de presentación de problemas pulmonares en la especie equina así como el potencial riesgo que suponen para la vida del animal, hacen necesaria la utilización de procedimientos diagnósticos capaces de detectar de forma precoz la existencia de tales procesos. En este sentido, la ecografía constituye un método diagnóstico ideal para el reconocimiento de numerosas patologías pulmonares comprometedoras de la vida del caballo.

A continuación, describiremos brevemente la técnica de ecografía pulmonar, así como los hallazgos ecográficos más relevantes de las patologías pulmonares que más comúnmente afectan al caballo.

## Técnica de ecografía pulmonar

En primer lugar, y como paso previo al estudio ecográfico de la cavidad pleural y de los pulmones del caballo, debemos considerar la realización de una limpieza exhaustiva de la superficie de escaneo, con la finalidad de evitar la interposición de cualquier resto de grasa y suciedad al paso de los ultrasonidos que pueda dar lugar a la formación de artefactos, para lo que también se recomienda el rasurado de dicha superficie.

Una vez limpia el área de escaneo, el siguiente paso es favorecer la transmisión de ultrasonidos, para lo que aplicaremos gel de ultrasonidos o bien bañaremos constantemente la superficie de contacto con la sonda ecográfica con alcohol

Ocupándonos del examen ecográfico propiamente dicho, éste debe comenzarse utilizando sondas de ultrasonidos de frecuencias que oscilen entre los 6.0 y 7.5 MHz, pudiendo acceder así hasta profundidades de 6-10 cm. No obstante, existen procesos en los que se requieren sondas de menor frecuencia (5.0, 3.5 o incluso 2.5 MHz) que nos permitan acceder hasta profundidades de 25-30 cm para su diagnóstico.

El estudio ecográfico debe realizarse en un ambiente tranquilo y cómodo para el caballo, siguiendo un patrón de escaneo en el que de forma metódica ecografiemos todos y cada uno de los espacios intercostales objeto de estudio a lo largo del ciclo respiratorio.

## Estructuras normales

El examen ecográfico de la cavidad pleural y de los pulmones del caballo sano pone de manifiesto, como hallazgos más relevantes, los siguientes:

- Línea hiperecogénica, correspondiente a la pleura visceral, que delimitará una zona más profunda ocupada por líneas equidistantes de artefactos de reverberación, indicativa de zona periférica de pulmón correctamente aireada. Durante la exploración ecográfica del ciclo respiratorio es fácil apreciar cómo esta línea hiperecogénica se aproxima ventralmente hacia el diafragma en la fase de inspiración, para alejarse dorsalmente en la fase de espiración.

- Ausencia de líquido en espacio pleural. En la mayoría de los caballos sanos no se aprecia la existencia de líquido durante el trascurso de la exploración ecográfica de la cavidad pleural, si bien existe un pequeño porcentaje de ellos en los que aparece escasa cantidad de líquido de características anecogénicas en las porciones más ventrales del tórax.

- Diafragma. El diafragma se presenta como una estructura curvilínea de considerable espesor y estructura muscular en las porciones más ventrales del tórax, y delgado y tendinoso en sus porciones dorsales

- Timo. Durante la exploración ecográfica pulmonar de animales jóvenes, es frecuente encontrar el timo alojado en el mediastino craneal, en porciones ventromediales del lóbulo apical del pulmón derecho.

- Finalmente, también cabe destacar la existencia de acumulaciones de grasa en ponies y caballos obesos a modo de estructuras heterogéneas y ecogénicas alojadas alrededor del corazón.

## Hallazgos más relevantes de los procesos que más comúnmente afectan a la cavidad pleural y a los pulmones del caballo

### Derrame pleural

El hallazgo ecográfico más significativo encontrado en los caballos afectados de derrame pleural es la existencia de un espacio anecogénico interpuesto entre la pared costal, el pulmón, el diafragma y el corazón.

Otro detalle característico es la visualización del ligamento frenopericárdico a modo de membrana flotante en el líquido pleural.

Además, también es posible apreciar el septo mediastínico al ecografiar las porciones craneales del tórax.

Una vez constatada la existencia de líquido en el espacio pleural, debemos prestar especial atención a las características del mismo. Así pues, intentaremos valorar la cantidad de líquido acumulado, al mismo tiempo que también atenderemos a propiedades como la ecogenicidad (los aumentos de ecogenicidad son compatibles con aumentos en la celularidad y concentración proteica) y la homogeneidad que presenta (es posible encontrar coágulos, restos de tejido, bandas de fibrina, acumulaciones de gas, presencia de capas, etc.).

### Consolidación pulmonar

El hallazgo más precoz de la existencia de zonas de consolidación pulmonar viene definido por la existencia de irregularidades en la superficie de la pleura visceral, a partir de las cuales se originan "colas de cometa".

Las zonas de consolidación pulmonar aparecen generalmente rodeadas por áreas con aireación normal y separadas de éstas por una línea irregular. El interior del área de consolidación suele presentar un patrón hipoecogénico en el que pueden destacarse trayectos bronquiales ocupados por aire o líquido, vasos pulmonares, etc.

### Abscesos pulmonares

La imagen ecográfica más típica de los abscesos pulmonares consiste en estructuras cavitarias de ecogenicidad variable, generalmente no encapsuladas. A veces, cabe encontrar una serie de capas en su interior, indicativas de la existencia de material de densidad variable, que puede llegar incluso a originar compartimentos dentro del absceso.

### Atelectasia

Los fenómenos de atelectasia vienen provocados por la compresión del parénquima pulmonar originada por acumulación de gas o líquido en el espacio pleural o por paso de vísceras desde la cavidad abdominal hacia la cavidad torácica.

Ecográficamente observaremos zonas de pulmón retraído, no aireadas y de ecogenicidad disminuida.

### Neumotórax

El diagnóstico ecográfico de neumotórax resulta especialmente característico en los casos en los que simultáneamente aparece derrame pleural (hidroneumotórax) donde encontraremos una interfase gas-líquido, situada en el espacio pleural, que se desplazará continuamente hacia porciones dorsales y ventrales, dependiendo de los movimientos respiratorios del caballo.

En aquellos casos de neumotórax simple el diagnóstico resulta más complejo dada la existencia de artefactos de reverberación

### Fístula broncopleural

El diagnóstico ecográfico de fístulas broncopleurales se caracteriza por la pérdida de la continuidad de la superficie de la pleura visceral, dando lugar a cavidades excavadas en la superficie pulmonar, pudiendo apreciar la salida de aire al espacio pleural cuando realizaremos el estudio ecográfico en tiempo real.

Como resultado de la existencia de este tipo de lesiones aparecerá neumotórax

### Pleuritis seca

El diagnóstico de pleuritis seca mediante ecografía resulta un tanto difícil dada la ausencia de líquido, alojado en espacio pleural, que nos permita contrastar fácilmente la pleura visceral y la pleura parietal.

Como hallazgos ecográficos más típicos de los procesos de pleuritis seca podemos mencionar la existencia de adherencias entre ambas pleuras, así como la falta de deslizamiento entre ellas durante los movimientos del ciclo respiratorio, si bien esta última característica se aprecia en numerosos caballos sanos en los que tienen lugar movimientos respiratorios poco profundos.

### Neoplasia pulmonar/Enfermedad granulomatosa/ Fibrosis pulmonar

El aspecto ultrasonográfico mas característico de este tipo de alteraciones viene representado por la existencia de numerosas masas de ecogenicidad variable distribuidas aleatoriamente por el pulmón.

### Hernia diafragmática

El diagnóstico ecográfico de hernia diafragmática lo basaremos en la observación de vísceras abdominales en porciones caudales de la cavidad torácica en contacto con el pulmón o flotando en el líquido pleural, no apreciándose el diafragma como septo de separación entre cavidad torácica y abdominal.

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# CASO CLÍNICO DE SEPTICEMIA NEONATAL GRAVE Y LA IMPORTANCIA EN LA ELECCIÓN DE ANTIBIÓTICOS

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El objetivo de este caso clínico es reflejar que la elección adecuada de un antibiótico en un neonato con septicemia de pronóstico muy grave puede ser esencial en su evolución.

Un potro PRE de 14 h de edad prematuro de 15 días fue admitido en la UE con signos de septicemia y cuya madre presentaba graves problemas metabólicos (enteropatía y endotoxemia intensas, junto con hiperlipidemia y babesiosis). El potro no había tomado calostro. De las analíticas realizadas, destacó una fuerte leucopenia con neutropenia y un fallo de transferencia de inmunidad pasiva. El tratamiento que se instauró fue fluidoterapia, antibioterapia (ceftiofur), transfusión de plasma y antiulcerígenos.

Doce horas post-ingreso presentó diarrea profusa, con ligera acidosis metabólica e hiponatremia. Se instauró un tratamiento con fluidoterapia compensadora y flunixin, que evolucionó de forma favorable. Como complicación del cuadro séptico también apareció un uraco persistente que se trató con nitrato de plata.

En el hemocultivo se aisló una *Escherichia coli* resistente a ceftiofur y sensible a gentamicina, amikacina, trimetroprim-sulfa y eritromicina, por lo que se cambió a penicilina/gentamicina.

A los 10 días después del ingreso, el potro presentó una artritis séptica de la articulación tarsocrural derecha. Se trató con lavados articulares y amicacina intraarticular. Dada la no mejoría de la artritis, se realizó una artrotomía y se realizó un cultivo, donde se aisló *Klebsiella pneumoniae* spp *pneumoniae* sólo con sensibilidad intermedia a amoxicilina-ácido clavulámico. Se cambió el antibiótico a amoxicilina, y cuatros días después a amoxicilina-ácido clavulámico oral. A partir de entonces, la evolución de la artritis así como del estado general del potro fue progresivamente muy favorable.

## CONCLUSIÓN

Este potro era de alto riesgo de septicemia porque era prematuro, su madre padecía graves problemas metabólicos periparto y había un fallo en la transferencia de inmunidad pasiva.

Intervinieron varios agentes bacterianos en la septicemia que se aislaron gracias a los cultivos seriados y se combatieron según el antibiograma, lo que aseguró su evolución favorable.

# INFLUENCIA DE LA OSCILACIÓN EN LA TEMPERATURA AMBIENTAL EN EL DESARROLLO DE LAMINITIS COMO COMPLICACIÓN DEL CÓLICO EQUINO.

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## RESUMEN:

El aumento en la temperatura ambiental podría favorecer un mayor edema a nivel del lecho vascular en las laminas del casco, y como consecuencia aumentar el riesgo de laminitis en animales con mayor predisposición (inflamación del tracto gastrointestinal, sobrecarga de carbohidratos, pastoreo en determinadas épocas del año, retención de placenta o endometritis, pleuroneumonía, endotoxemia, sepsis, enfermedad de Chushing, sobrecarga de una extremidad, etc.) (Stashak, 2002). La revisión de los casos de cólico, tanto médicos como quirúrgicos, referidos a lo largo del último año al Hospital Veterinario de Aznalcollar (Sevilla CP 41870, España) y que tienen como complicación posterior laminitis, revela que del total de cólicos que se complicaron con laminitis a lo largo del año, un 76,92% lo hicieron durante el verano.

## DESARROLLO:

La laminitis es un proceso que normalmente aparece de forma secundaria a cuadros inflamatorios, isquémicos o tóxicos. Independientemente de la causa que la inicia, parece bastante claro que concurren fenómenos vasculares e inflamatorios que derivan en edema y necrosis de las láminas (Stashack, 2002) (Moore, 1996) (Pollit, 1998).

A diferencia de lo que pudiera pensarse, el lecho capilar del pie del caballo es altamente permeable a fluidos y macromoléculas, incluso más permeable de lo que pudieran ser los lechos capilares en las almohadillas de perros y ratas (Eades et. Al, 2002) (Stashack, 2002). Esto conlleva una mayor concentración intersticial de proteinas, favoreciendo la formación de edema. Algunos estudios concluyen que durante el establecimiento de la infosura aguda, se produce también un aumento en el número de capilares perfundidos y un aumento de la presión hidrostática sobre los mismos, y esto podría conllevar un aumento en el movimiento de fluidos hacia el tejido intersticial (MacAllister et al., 1993) (Moore et al., 1989). Otros estudios han concluido como primera respuesta hemodinámica durante en establecimiento de la laminitis aguda, tanto en casos de sobrecarga por carbohidratos como en casos de administración de estractos de nuez negra , la venoconstricción digital. L a constricción de las venas digitales conllevaría un aumento de la presión capilar en la microvasculatura podal, forzando un transporte de líquidos hacia el intersticio, que aumentaría la presión intrapodal produciendo lesiones debidas al estrés compartimental (Moore et al., 1996).

Algunos estudios parecen indicar que la vasoconstricción en los estadios iniciales de la infosura tienen un efecto protector frente al desarrollo de la misma (Stashack, 2002) (Pollit, 2003). La vasoconstricción inducida por frio (crioterapia distal del miembro) en las fases de establecimiento de la infosura podría limitar la exposición a factores desencadenantes y reducir el impacto sobre la anatomía laminar (Pollit & Davies, 1998). La crioterapia podría actuar también reduciendo la actividad cinética de las encimas envueltas en el desarrollo de la infosura, tales como la MMP-2 y la MMP-9 (Pollit, 2002), la ET-1 (Eades et al., 2002), etc. Experimentos iniciales, apoyan la efectividad de la crioterapia a la hora de interrumpir la instauración de la laminitis (van Eps & Pollit, 2002).

Por el contrario, la terapia vasodilatadora y los baños calientes de las extremidades están contraindicados en la fase inicial de la laminitis (Pollit, 2003).

El desarrollo de un ejercicio intenso o la anestesia local de los nervios plantares y palmares conllevan un aumento de la temperatura del casco, induciendo un efecto vasodilatador sobre los capilares de la zona (Eades et. Al, 2002), que favorece el desarrollo del edema, y en ocasiones laminitis.

Del mismo modo, una temperatura ambiental excesiva, podría estimular los mismos mecanismos homeostáticos que derivaran en vasodilatación periférica; y dada la predisposición existente en el lecho capilar podal del caballo a sufrir edemas, se podría pensar que un aumento significativo en la temperatura ambiental podría favorecer el desarrollo de edemas podales que deriven en laminitis.

Diversos estudios coinciden en destacar un mayor número de casos de laminitis durante las épocas más cálidas del año. Un estudio realizado por los servicios veterinarios del Departamento de Agricultura de los Estados Unidos de América y publicado en Abril del 2000 por el "National Animal Health Monitoring System" destacó que en general un mayor número de caballos se veían afectados por cojeras y laminitis en primavera y verano, comparándolo con los casos que se daban en invierno. No obstante la mayoría de estos estudios, relacionan las mayores prevalencias en estas épocas del año con modificaciones en la composición del pasto. El consumo de pastos exuberantes es una causa común de laminitis (Stashak 1987). Estos resultados se deben principalmente a las modificaciones que sufren los pastos en su composición a medida que se suceden las estaciones.

Dorn (1975) observó un pico en el número de casos de laminitis durante la primavera y el verano. En un estudio retrospectivo realizado en el Reino Unido se observó que los casos de laminitis se daban con mayor frecuencia durante los meses de verano (Katz et. al., BEVA 2000). Un año después, el mismo grupo, en un nuevo estudio retrospectivo, obtuvo una relación significativa positiva entre las horas de luz y la incidencia y la prevalencia de laminitis (Katz et al., BEVA 2001). No obstante, otros estudios no consiguieron establecer ninguna relación significativa entre la estación del año y el riesgo de sufrir laminitis (Slater 1995, Polzer 1996).

En el Hospital Veterinario de Aznalcollar (Sevilla CP 41870, España) también se ha venido observando una mayor incidencia de laminitis como complicación de procesos de cólico, tanto quirurgicos como médicos, durante los meses estivales. Atendiendo a las observaciones que se estaban realizando en el centro y a la bibliografía existente al respecto, se decidió realizar una revisión de todos los cólicos ingresados durante el último año para estudiar la incidencia de laminitis en dichos caballos en relación a las distintas estaciones.

A la hora de realizar el estudio se tuvieron en cuenta las siguientes premisas:

1. El estudio recoge todos los cólicos referidos al centro los meses comprendidos entre Noviembre de 2002 y Octubre de 2003, ambos inclusive.
2. Se excluyeron aquellos animales menores de un año.
3. Se excluyeron aquellos animales eutanasiados o muertos en las primeras horas de estancia en el hospital.

4. Todos los animales habian mantenido la dieta habitual durante al menos un mes antes de la aparición de la patología digestiva.
5. Todos los caballos estudiados provenían de la Comunidad Autonóma de Andalucía.

En base a los datos obtenidos del Ministerio de Medio Ambiente, los Valores Climatológicos Normales, atendiendo al periodo 1971-2000 (último periodo a estudio publicado), indican la existencia de mayores temperaturas durante los meses de Julio y Agosto, obteniéndose las temperaturas máximas de la Comunidad Autónoma en las provincias de Sevilla y Córdoba (INM, 2003).

El grupo final, objetivo del estudio estaba formado por 50 animales, de los cuales 13 se complicaron con laminitis y 37 no lo hicieron.

Los caballos que se complicaron con laminitis en verano fueron el 76,92% del total de caballos que se complicaron con laminitis a lo largo del año. El resto se repartieron de la siguiente forma. Un 7,69% en otoño, un 7,69% en invierno y el restante 7,69% en primavera, lo que arroja un reparto bastante uniforme en estos periodos del año.

Atendiendo a la aparición de la laminitis en cada estación, durante el verano se complicaron con laminitis el 40% del total de cólicos remitidos en dicho periodo; en otoño el 8, 33%, en invierno el 25% y en primavera el 11,11%.

Aunque los porcentajes parecen indicar con claridad que las diferencias son significativas, se cree conveniente la necesidad de aumentar el tamaño de la muestra para obtener datos estadísticamente significativos con un alto nivel de confianza.

Es por ello que en un futuro se espera poder disponer de una muestra de mayor tamaño, que sea, por tanto, más representativa y permita esclarecer una posible significancia entre las oscilaciones en la temperatura ambiental y la incidencia de la laminitis como complicación en un cuadro cólico; así como poder incluir y comparar otros parametros como la humedad relativa, la ubicación geográfica o el patrón racial.

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# DESCRIPCIÓN DE TRES CASOS CLÍNICOS DE BRONCONEUMONÍA EN POTROS DE PURA RAZA ESPAÑOLA (PRE) ASOCIADOS A INFECCIONES POR *RHODOCOCCUS EQUI.*

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## Resumen

En este trabajo describimos las manifestaciones clínicas y hallazgos postmortem de tres brotes de bronconeumonía en potros de Pura Raza Española (PRE), procedentes de distintas explotaciones de Andalucía. El aislamiento y posterior caracterización bioquímica de Rhodococcus equi se realizó según métodos convencionales en el Servicio de Diagnóstico de la Facultad de Veterinaria de la Universidad de Córdoba.

En dos de los casos clínicos observamos un patrón patológico "mixto", detectando abscesos pulmonares y digestivos, localizados estos últimos en mesenterio y mesocolon; además de afecciones articulares severas en ambos casos (poliartritis). En el tercer caso clínico describimos la forma exclusivamente "neumónica" de la enfermedad.

En el primero de los casos, la inoculación experimental de material patológico vía oral a ratones (línea Swiss) reprodujo las lesiones digestivas (abscesos abdominales) tras cuatro días postinfección.

Las pruebas "in vitro" mostraron alta sensibilidad de las cepas aisladas frente a eritromicina y rifampicina, además de aminoglucósidos (gentamicina y neomicina) y quinolonas. Finalmente, realizamos preparados inactivados (formolizados) a partir de las cepas aisladas, que fueron administradas a las yeguas y potros de cada explotación para el control de la infección.

# TENDINITIS SÉPTICAS EN LAS EXTREMIDADES POSTERIORES

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## Objetivos del trabajo.

Durante este se hace referencia a varios casos de tendinitis de origen séptico detectadas en los tendones de los músculos flexores en las extremidades posteriores en caballos de razas y actividades físicas diferentes.

El origen de estos procesos suele ser traumático con la instauración de una herida profunda que afecta a los tendones de los músculos flexores. Estos caballos se caracterizan por la presencia de signos clínicos intensos en el área afectada, con trayectos fistulosos o heridas, inflamaciones, y cojera de moderada a intensa.

La ecografía es la técnica complementaria de elección en estos casos, pues permite una correcta valoración del trayecto fistuloso y la relación con los tejidos blandos dispuestos en la zona.

En el transcurso del trabajo se muestran lesiones ecográficas de tipo séptico tanto en el tendón flexor digital superficial como en el tendón flexor digital profundo, pero a diferentes niveles.

El pronóstico para estas lesiones es reservado a desfavorable para el inicio o vuelta a la competición, por lo que el diagnóstico precoz de la enfermedad resulta primordial para la instauración de un tratamiento adecuado y agresivo, con objeto de mejorar las probabilidades de recuperación de la lesión.

Igualmente, se hace referencia a los cambios inducidos por estas lesiones en las formaciones adyacentes.

# LAS HERIDAS EN LA SUELA, ¿SON LO QUE PARECEN?

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**Resumen:**

Los dos casos clínicos que se van a exponer a continuación hablan de dos lesiones en el casco las cuales tienen los mismos signos y síntomas. Cursan como un absceso recidivante que causa cojera. Pero están causadas por dos patologías completamente diferentes, que sólo se pudieron distinguir con un diagnóstico más en profundidad.

Con esto queremos resaltar que las lesiones en el casco no siempre son lo que parece y que hay que prestarles la atención que se merecen.

El casco es una de las regiones corporales del caballo que sufren con más frecuencia lesiones y patologías, dentro de estas lesiones nos encontramos unas muy comunes como:

- Alteraciones morfológicas
- Abscesos
- Hematomas
- Hormiguillo
- Infosura
- Cuartos...

Y otras menos:

- Gabarro cartilaginoso
- Fracturas de la 3ª falange
- Osteitis podal: séptica o no
- Calcificación de los cartílagos alares de la 3ª falange
- Queratomas
- Pododermatitis crónica verrugosa...

A continuación expondremos dos casos clínicos, relacionados con estas patologías y que han sido seleccionados de entre los casos de ortopedia que implican el casco y que han sido recibidos en los últimos 6 meses en el Servicio de Cirugía de Grandes Animales del Hospital Veterinario de Aznalcóllar.

## CASO Nº1:

### Historia:

Potro PRE de dos años y medio que tras estudio radiológico del casco realizado por su veterinario, viene referido al hospital para su tratamiento.

El potro presentaba una cojera de 3/5 al trote en línea recta de su miembro anterior izquierdo desde hace unos veinte días. Los propietarios habían estado tratándolo con povidona yodada en el casco ya que le habían visto supurar pus por la suela. Al ver que no mejoraba avisaron al veterinario, que lo remitió de inmediato.

### Diagnóstico:

En la exploración del casco se encontró un orificio de salida dos dedos craneal al vértice de la ranilla, por el cual supuraba pus. Con estos signos los posibles diagnósticos diferenciales incluían:

- Traumatismo
- Queratoma
- Hematoma
- Carcinoma
- Hemangioma
- Osteomielitis
- Infosura crónica

En el examen radiológico posterior se descubrió un trayecto que desembocaba en el orificio indicado y que ascendentemente llegaba hasta la 3ª falange. En ella existían signos radiológicos de remodelación ósea, compatibles con osteomielitis.

### Tratamiento:

Se descartó un tratamiento conservador debido a la presencia de signos radiológicos en la 3ª falange y a la duración del proceso, por lo que se optó por el tratamiento quirúrgico.

Previo a la anestesia general el herrador legró el casco. En la cirugía se preparó el campo asépticamente. Se le colocó un torniquete con una venda de Smarch. Con el Dremel y una cucharilla se extirpó gran parte del tejido corneo de la palma llegando hasta la zona necrótica de la 3ª falange. Se reconocía por su aspecto avascular, descolorida y seca, la cual se desbridó hasta llegar a tejido sano.

Para mantener la zona limpia se le colocaron unas gasas con povidona yodada cubiertas por un vendaje a base de algodón y venda adhesiva; y esta se recubrió con yeso para proteger la zona.

El tratamiento se completó con antibioterapia oral a base de enrofloxacina (7,5mg/kg/24h) y metronidazol (7,5mg/kg/6h); fenilbutazona (2mg/kg/12h) y omeprazol (4mg/kg/24h).

Cada 7 días se le realizan lavados (water-pick) con suero a presión y cambio de vendaje.

## CASO Nº2:

### Historia:

Caballo anglo-árabe de 5 años que llega al Hospital para un examen de cojera, con la siguiente historia:

El caballo presenta una cojera 2/5 al trote en el miembro anterior izquierdo de carácter recidivante, que fue examinada meses atrás por su veterinario y correspondió con un absceso subsolar. Le reseccionó la parte de la muralla afectada, que fue rellenada con acrílico y tratado con antibióticos. No se observó mejora por lo que se remite para una segunda opinión.

Presentaba la misma cojera 2/5 al trote de la extremidad anterior izquierda. La respuesta a las pinzas de casco era muy leve, lo que hizo pensar que además de tener alguna secuela por el absceso, podía presentar otra lesión. Se decidió proseguir el diagnóstico con bloqueos anestésicos para localizar la cojera.

Al bloqueo sesamoideo abaxial dio respuesta positiva, así como a la anestesia de la articulación interfalangiana distal. Al realizarle radiografías de la zona anestesiada, no se apreciaron cambios por lo que se pensó que podía tener una artritis a nivel de la interfalangiana distal. Se decidió tratar con acetato de prednisolona y amikacina.

A partir de esto el caballo tuvo una leve mejoría, pero dos meses después seguía estando cojo y volvió al hospital para ser examinado de nuevo.

### Diagnóstico:

En la exploración se comprobó que el absceso se le había vuelto a abrir. Con las pinzas de casco mostraba una respuesta mayor que la vez anterior. Se decidió hacer unas radiografías para poder ver el alcance del absceso, y realizar su diagnóstico diferencial entre un simple absceso cronificado, o un absceso producido por un queratoma, osteomielitis, hematoma, carcinoma o hemangioma.

Con las placas no se pudo llegar a un diagnóstico concreto, aunque observamos cambios radiológicos de remodelación ósea en la tercera falange.

### Tratamiento:

Debido a la cronicidad de la cojera y a los cambios radiológicos se decidió el tratamiento quirúrgico.

Antes de la cirugía se legró toda la zona de drenaje del absceso. Se colocó un torniquete con una venda de Smarch por encima del menudillo. Tras esto se procedió a limpiar toda la zona necrosada del absceso, hasta que se encontró una masa dura con aspecto de queratoma, (diagnóstico definitivo se consiguió enviando una muestra de la masa para Histopatología). Para extirparla se hicieron dos cortes paralelos en la pared del casco a ambos lados del posible tumor. Después se eliminó dicha pared, la cual se fue separando poco a poco de la dermis hasta llegar a la corona. Finalizada la extirpación se realizó un curetaje de toda la zona afectada.

A continuación se tapó toda la zona extirpada con gasas impregnadas en povidona yodada y metronidazol. Cubriendo estas se puso un vendaje, con una inmovilización a base de fibra de vidrio para impedir que se abriera el estuche corneo. También se le colocó una plantilla de yeso con el objetivo de minimizar la expansión del casco al apoyar.

Este vendaje se retiró a las 48h. La zona se limpió utilizando la water-pick con suero y povidona yodada. Cubriendo la zona se colocó una venda de gasa dejando su borde superior libre del ven-

daje, para curarle cada dos días impregnando dicha venda con un antiséptico. Todo esto se volvió a recubrir con fibra de vidrio y se le puso una nueva plantilla de yeso.

El tratamiento se completó con penicilina procaínica (22.000 U.I./kg/12h), gentamicina (6,6mg/kg/24h) y fenilbutazona (2mg/kg/12h).

A los 10 días se le retirará el vendaje y se le volverá a lavar toda la zona a presión. Después de esto se le adaptará una placa ósea ortopédica que se atornillará en la pared del casco para salvar el espacio y estabilizar el casco. También se le pondrá una herradura con pestañas y se le rellenará la suela con acrílico sustituyendo la plantilla de yeso. Para añadir más estabilidad al casco se le repetirá el vendaje con fibra de vidrio, dejando salir por el borde superior las gasas para continuar con las curas.

## Conclusión:

Los abscesos en el casco son una patología muy frecuente en la clínica de caballos. Habitualmente los diagnosticamos por el grado y aparición repentina de la claudicación y por los signos externos; como calor en la zona, respuesta positiva al bloqueo abaxial bilateral y a las pinzas de casco, pulso digital, etc. Pero además debemos pensar en el alcance y la causa del absceso. El estudio radiológico nos ayudará a ver la gravedad de la lesión, ya que como hemos visto no son siempre lo que parecen. La mayoría de los abscesos se suelen resolver de manera satisfactoria; si bien unas veces por que se complican y otras por que son secundarios a otra patología requieren un tratamiento mucho más largo y costoso, además de un pronóstico reservado; de ahí la importancia del diagnóstico.

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