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AN UPDATE ON INSECT BITE HYPERSENSITIVITY (IN THE NETHERLANDS)
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Insect bite hypersensitivity (IBH), going by one of a wide variety of names, has been around for a long time, with much preliminary work being performed in Australia during the middle of the last century. There has however been renewed interest lately, due to the disease itself and the welfare issues it raises, but also to the fact that the insects involved may act as vectors for a number of (emerging) diseases.

As the name suggests IBH is the result of a (type I and IV) hypersensitivity reaction to antigens (probably salivary) of certain insects, causing intense pruritis, which recurs seasonally. Pruritis, occurring predominantly around the mane and the base of the tail, leads to auto-mutilation and severe discomfort in affected horses. Disease severity is often progressive and permanent changes to the skin and subcutaneous tissue can occur. A genetic predisposition has long been suspected and has now been confirmed in several horse breeds.

A number of surveys have been performed to establish in which regions IBH (and thus Culicoides midges) is most prevalent and to identify factors influencing this distribution. The different studies show a similar distribution, reflecting conditions favouring survival, activity and breeding of Culicoides midges. Culicoides require a minimum temperature to survive. This varies per species but generally lies in the range of 10-15ºC, explaining the seasonal occurrence. They are poor fliers, being active when there is little wind and no rain and more suited to areas offering protection. Finally, they require (stagnant) water to reproduce. Even within a small country like the Netherlands there are considerable regional differences in the number of Culicoides midges, and hence in the prevalence of IBH. In general, Culicoides numbers are lowest in coastal areas and increase further inland, especially when protection from wind and rain is present (4). It is likely that there are also large variations more locally.

Although the typical lesions, their distribution and seasonality can be used to diagnose IBH in the ‘IBH season’, a (reliable) diagnostic test has been lacking until now. Ideally, such a test can also discriminate between horses susceptible to IBH and healthy horses in the winter, when Culicoides are not present/active. Intradermal testing in horses is notoriously difficult to interpret and the matter is complicated further by the fact that little is known (with certainty) about the cross-reactivity of the different Culicoides species. However, recent research has shown that when an extract of native Culicoides midges is used an intradermal test can identify horses suffering IBH, at least in the summer. Further studies are required to confirm the usefulness of this test and to determine if it is also usable in the winter. In addition work is continuing on the development of a reliable blood test for IBH.

It is well established that Culicoides midges are responsible for causing IBH but there is less certainty as to which species are to blame, and indeed if these are the same species in different countries. Studies involving the trapping of insects around horses have shown that the vast majority of the Culicoides attracted to horses are C.obsoletus and, to a lesser degree, C.pulicaris (1,2). Intradermal testing has confirmed the role of these species in IBH (in the Netherlands), with C.obsoletus appearing to be more important (3).

In theory treatment of IBH is simple: separate the horses from the midges, but everyday practice confirms that things are rarely as simple as they seem.
bling horses during certain times of the day can help, but for this strategy to be effective the times when the (native) Culicoides midges are most active need to be known. C. obsoletus and C. pulicaris are mainly active around sunset and, to a lesser degree sunrise. Stabling horses in the early morning and from 3 pm until after dark seems appropriate. Also, use of rugs and blankets can help protect horses from being attacked by midges. It is important that all susceptible areas are covered and they work best when applied before the first symptoms appear. Insecticides (which can be used together with rugs) can also play a role in controlling IBH symptoms. We have tested a permethrin containing insecticide and although we could not show a significant reduction in Culicoides numbers attracted to horses 24 hours after application (2) our clinical impression is that this insecticide reduces the severity of IBH symptoms. Insecticides can also be used together with rugs to help protect horses from mites and may also play a role in reducing IBH symptoms. We have tested a permethrin containing insecticide and although we could not show a significant reduction in Culicoides numbers attracted to horses 24 hours after application (2) our clinical impression is that this insecticide reduces the severity of IBH symptoms. The composition of the diet, especially with regard to fatty acids, has also been suggested to influence IBH and a dietary supplement containing vitamins, amino acids, polypeptides and natural oils (Hippo-Ex-Cema®) reduced the severity of IBH in a placebo-controlled, double blind trial (manuscript in preparation).

In the future we intend to investigate further (and optimise) the sensitivity and specificity of intradermal testing, with the ultimate aim of developing successful desensitisation programmes. We hope to be able to characterise the genetic background in susceptible horses, which could lead to breeding guidelines aimed at reducing IBH prevalence. Field trials will (continue to) be performed to evaluate therapeutic interventions, including another dietary supplement and a topical gel, and finally, research will continue into the (feeding) activity of various Culicoides species.

References

Tendons are a specialized form of fibrous connective tissue that are responsible for transmitting mechanical forces from muscle to bone but also act as joint stabilizers and as ‘shock absorbers’ to limit muscle damage. Tendons consist of a relatively small cellular fraction and a correspondingly large extracellular matrix (ECM), which is composed of densely packed and hierarchically arranged collagen filaments, embedded in a hydrophilic, proteoglycan rich matrix that provides tendons with their characteristic properties. Blood vessels enter tendons at three sites: at the myotendinous junction and osteotendinous insertion and via the paratenon. However the blood supply is scarce and even decreases further with increasing age and mechanical loading.

In normal tendons there is a finely tuned equilibrium between catabolic and anabolic processes. There is a balance between the breakdown of damaged ECM and the production of new ECM constituents. This balance, essential for tissue maintenance and repair, is maintained by matrix metalloproteinases (MMPs), a group of proteolytic enzymes and their inhibitors (TIMPs). Gradual increase of mechanical stress, causing microtrauma, may result in a physiological adaptation to the increased demand, leading to a stronger tendon. However, the capacity of tendon tissue to adapt to increasing demands is relatively small, especially at an adult age.
It has now been well accepted that tendon injuries are most commonly caused by accumulating micro-trauma through repetitive overload rather than by a single macro-trauma. When repetitive loads create damage to the ECM at a rate that exceeds the repair capacity of the tendon cells, the damage accumulates and becomes clinically apparent. This repetitive micro-trauma causes the release of pro-inflammatory cytokines that enhance tissue breakdown, causing weakening of the tendon and predisposing it to major trauma at a later stage.

Many factors influence the delicate equilibrium of tissue homeostasis within tendons. Biomechanical factors appear to play a central role in this. Not only can overload of a tendon cause micro-trauma and tissue degeneration, it appears that also under-stimulation (stress deprivation) of tendon tissue can be the onset of degenerative events. In stress deprived tendons an increase in MMP/TIMP ratio can be observed, which causes a possible increase in degeneration of collagen and other ECM constituents. Other factors that may play an important role in tendon homeostasis include hyperthermia that develops during exercise within the core of the tendon and hypoxia and the production of free radicals during exercise due to the poor vascularisation of tendons. The conformation of the horse, its fitness and the training surface are suggested to also play a role in the development of tendon injuries. Presumably there is a combination of factors involved and indeed no one factor may be totally responsible.

With increasing age tendons appear to be at higher risk for sustaining an injury. The quality of the tissue deteriorates due to accumulating micro-trauma whereas the capacity to adapt to increasing workloads decreases. It will be impossible to avoid all factors that negatively influence tendon homeostasis during a horse’s career. However management factors and adequate training regimes might significantly reduce the risk of obtaining a tendon injury.

NEW TREATMENT OPTIONS FOR TENDON INJURIES
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Tendon healing takes place in three distinct, but partially overlapping phases; the inflammatory (acute) phase, the proliferation phase and the maturation and remodelling phase. The mechanisms at work are very characteristic in each of these different stages, implying that therapeutic requirements in each stage also differ. Therapies in the acute phase should aim at damage control by protecting the affected area against secondary injury and at reduction of inflammation, which is mostly done by a combination of physical therapies, such as cooling and rest, with Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). Recently it has been suggested that applying a rigid lower limb cast can be beneficial in the treatment of tendon lesions by preventing further propagation of the initial lesion.

During the proliferation phase new matrix starts to form and the characteristics of the new tendon matrix are determined. Therapies in this stage should aim at accelerating the formation of a fibro-proliferative callus and improving the quality of the ECM that is laid down. This process can be enhanced by the implantation of mesenchymal stem cells or intratendinous treatment with growth factors. Mesenchymal stem cells, which are prevalent in bone marrow but which are also found in significant numbers in fat, are capable of differentiating into tenocytes. For clinical applications, bone marrow, containing mesenchymal stem cells can be directly implanted into the tendon lesion, but preferably the stem cells are expanded \textit{ex vivo} before implantation. Platelet rich plasma (PRP), an autologous concentrate of thrombocytes, contains high levels of various growth factors that play an important role in tissue repair in general and in tendon healing in particular. PRP is simple to produce at any time during the healing trajectory. Both stem cell expansion and PRP concentration kits are commercially available and fairly easy to perform in practice. Although still scarce, more and more evidence for the effectiveness of both therapies becomes available.

During the remodelling and maturation phase the ultimate structural and biomechanical properties of the tendon will evolve and this process can be significantly accelerated.

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repair tissue are defined. A gradual increase in mechanical loading has been shown to be beneficial to tendon healing in this phase, making controlled exercise still the cornerstone of tendon healing in this phase. Too early or too intensive mechanical loading might be just as detrimental to tendon repair as too late or too moderate exercise levels, as both stress deprivation as well as repetitive mechanical stress can induce metalloproteinase activity and induce collagen damage. However, little is known about the relationship between stage of tissue integrity during recovery from a tendon injury and the functional loading capacity of the injured tendon. The recently introduced therapies that enhance matrix anabolism probably do not only influence the quality of repair but also the time course of the healing process, underlining the crucial role of and need for a balanced rehabilitation programme.

For adequate treatment of tendon injuries it is crucial to know the phase of repair the lesion is in, which urges the need for imaging modalities that are able to distinguish between the different phases. Furthermore the introduction of new and promising treatments make the reconsideration of rehabilitation programs essential.

HOW TO ASSESS AND TREAT UMBILICAL HERNIAS
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Umbilical problems are quite often seen in young born foals. Umbilical swelling can be caused by different conditions such as infection and/or abscess formation, simple or large congenital hernias, and complicated hernias associated with incarcerated bowel or abdominal abscesses.

Each swelling in the umbilical region should be examined closely for drainage and its relationship to the umbilicus. The umbilical area should be carefully palpated to assess the general consistency of the swelling, the presence of pain or heat, the size of the hernial ring and the reducibility of the contents of the hernial sac. In doubt, an ultrasonographical examination should be carried out.

Most of the small, simple umbilical hernias will heal spontaneously within the first weeks of life. Complications such as strangulations of intestine into the hernial ring will rarely occur during the neonatal phase. These complications will be seen more frequently in foals of 3 to 6 months of age. Close monitoring of these foals will be very important since only 30% of foals with a strangulation of intestine into an umbilical hernia will show signs of colic. Sudden local swelling and local pain is a more reliable symptom of this condition.

The choice of treatment will depend on the presentation of the hernia, the age of the foal, the desires of the owner and the surgeon’s preferences. As mentioned earlier, small hernias (< 5 cm) tend to heal spontaneously. Close monitoring these hernias is a must and owners should therefore be instructed how to reduce the contents of the hernia and to do so regularly. They also should be alerted to call for veterinary assistance in case of sudden swelling, pain and irreducability.

In case the hernia persists or enlarges, other treatments should be attempted. Although I am personally not in favour of non-surgical treatments, I have to mention their existence. Bandages or corsets do support the hernial sac and stimulate abdominal wall closure of persistent small hernias. Care should be taken not to cause pressure wounds, especially on the back of the foal. Small rubber bands or umbilical cord clamps should be used very cautiously and are not recommended by the author. These methods cause necrosis and sloughing of the hernial sac, inflammation and fibrosis of the abdominal wall, and a skin defect that heals by second intention.

Persisting or enlarging hernias should be treated by elective umbilical herniorrhaphy. This surgical treatment is routinely performed in older foals for cosmetic reasons and/or to prevent intestinal strangulation. Reasons to perform an umbilical herniorrhaphy in neonates are: umbilical herniation in combination with infection, irreducible strangulation of intestine into the umbilical hernia, large size of the hernia and rupture of the hernial sac with subcutaneous presence of intestine. Most umbilical hernias are treated when the foals are 2 to 3 months of age, just before weaning. At this age, foals are still easy manageable. Alternatively, surgery can be postponed until the foal is weaned and halter broken. During this period, close monitoring should be continued.

Inhalation anesthesia is preferable. The foal is placed in dorsal recumbency and prepared for aseptic surgery in a routine manner. Umbilical herniorrhaphy can be performed in different ways. The author prefers a closed reduction, avoiding opening and possible contamination of the abdominal cavity. Closure of the hernial ring can be achieved in numerous manners: simple continuous suture patterns, simple interrupted sutures, overlapping

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Fractures to the rostral part of the mandible are one of the most encountered fractures in horses. The cause of the fracture is in most cases a violent pull back escape attempt while the incisors or lower jaw is trapped on a fixed object, resulting in a typical avulsion fracture.

Diagnosis is straightforward and is made based on a thorough clinical examination of the oral cavity. Clinical signs of mandibular fractures are: excessive drooling, lack of appetite, local swelling of the soft tissues, hemorrhage, instability, malalignment and halitosis. The clinical examination should always be extended to other structures to assess the integrity of the nasal passage and the cranial nerves. Also the general condition of the patient has to be checked since mandibular fractures can lead to the development of a severe dehydration.

In some cases radiographic examination can be applied, especially to reveal damage to tooth roots. Surgical treatment of mandibular fractures is indicated for the following reasons: instability of the fracture, malocclusion and bilateral configuration. With surgical fixation, a better cosmetic outcome and a more rapid healing can be expected.

Simple mandibular fractures, not requiring external fixators and/or internal implants and not extending beyond the canine teeth, lend themselves to be treated on the standing horse, using sedation and regional anesthesia. Desensitization of the rostral part of the mandible is achieved by blocking the mandibular branch of the trigeminal nerve. This can be carried out as this nerve exits the mental foramen on the lateral surface of the mandible. The foramen is usually palpable just ventral to the level of the commissure of the lips, underneath the tendon of depressor labii inferioris muscle. If for one or another reason more caudal desensitization is needed, the nerve must be blocked as it enters the mandibular foramen at the medial surface of the vertical ramus of the mandible. Regional anesthesia is in most cases applied to both sides of the mandible. General anesthesia may be necessary for the treatment of young and/or unmanageable horse.

**Surgical technique:**
The decision what technique will be used to treat a mandibular fracture, will depend on its localisation, its complexity and configuration and the surgeons preferences. Unnecessary to say that pre-operative planning will be of great importance.

Preparation of the patient consists of flushing of the mouth, curettage of the fracture line to remove food, blood clots and devitalised tissues. General administration of NSAID’s and antibiotics is obvious as is the controle of the vaccination status of the horse.

Two considerations have to be made before starting the surgery: all loose teeth should be preserved and never forget you are working in a contaminated area with an open fracture.

The goals of the treatment are to restore the anatomical alignment and to achieve a stable fixation. To reach these goals, the simplest method of repair should be applied. This usually involves the application of intraoral wires.

Materials needed are:
- Different sized curettes
- Flushing fluids (+ pressure bag, fluid line and needle)
- 14G needle or Steinmann pins of appropriate diameter.
- Jacobs chuck
- Cerclage wire (1 to 1.25 mm)
- Pliers
- Wire cutting forceps
- Protection materials

After thoroughly cleaning of the fracture line and manual reduction of the fracture, the cerclage wire will be applied in such way that the fractured part is fixed to the adjacent healthy part. This can be achieved by applying one wire in a figure of eight configuration or with multiple overlapping wires.

A continuous wiring technique, as used in human surgery, will provide a very rigid and stable fixation. Wire loops are inserted between each tooth from the lingual to labial side. One end of the wire is fed through
A practitioner’s guide to bronchoscopy in the horse

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Bronchoscopy using either fibreoptic or videendoscopes is now readily available to most equine practitioners. This presentation will address clinical abnormalities distal to the larynx. At least a 145 cm scope is necessary to examine the trachea and its bifurcation in large horses; a 165 cm instrument is better and a 200-300 cm gastroscope is helpful for examination of the more distal bronchi. 8 mm external diameter scopes are ideal for all except small ponies/foals.

The indications for bronchoscopic investigation are coughing, nasal discharge, epistaxis, abnormal respiratory noise, abnormal respiratory effort either at rest or after exercise and loss of athletic performance.

As with any imaging technique familiarity with normal structure and function is necessary to interpret abnormal findings. Structural abnormalities of the trachea (collapsing, scabbard, corkscrew) are important to recognise. Deformity of the carina and/or conducting airways may be seen with severe inflammation or infection, abscessation, interstitial pneumonia and neoplasia. Through post mortem latex casting precise bronchial mapping is available for the horse but most investigations and re-examinations are lesion directed. Intrabronchial foreign bodies and neoplastic masses are occasionally detected. Assessment of thickening or swelling of the carina due to airway inflammation is useful in severe cases of Recurrent Airway Obstruction (RAO) but in general does not correlate well with disease severity. Discharge of blood, mucopus or both from a single mainstem or segmental bronchus is highly significant and indicates focal disease which can be directly inspected and sampled. This can also inform external imaging with ultrasound or radiography.

Assessment of inflammation within the bronchial tree by visual inspection, grading of tracheal mucopus accumulation at the thoracic inlet and harvest of tracheal secretions and/or bronchoalveolar lavage fluid (BALF) for cytology and bacteriological culture is the most frequent use of bronchoscopy. Attempts have been made to correlate the amount of tracheal blood and/or mucopus to performance in racehorses with variable success. In northern Europe the prevalence of airway inflammation in housed animals during the winter months is high but in animals competing in lower speed disciplines it may not necessarily be performance-limiting.

Visual inspection of tracheal secretions alone is often diagnostically unrewarding as the response of the airways to noxious stimuli is often generic in nature and thus the appearance of secretions is similar whether primarily inflammatory or infectious in origin.

Cytological examination and bacteriological culture are required for more detailed evaluation and diagnosis and should be correlated with the animal’s history and clinical examination. Transendoscopic tracheal or bronchial secretion samples for bacteriology should be collected rapidly at the start of the procedure using a sterile (preferably agar-plugged) catheter to minimise contamination. A direct aspirate or lavage may be collected and the sample divided into appropriate sterile containers with and without cytological preservative (e.g. EDTA or Cytospin fluid) for bacteriology and cytology respectively. Tracheal wash analysis provides information about secretions originating from the entire bronchial tree.
whereas BALF is collected from a single lung segment and unless specifically guided to sample a diseased segment, this assumes that diffuse lung inflammation is present. BALF can be collected "blind" with a proprietary tube, which usually samples the right dorsocaudal lung segment or transendoscopically from a selected bronchus. In normal horses and those with RAO, BALF cytological profiles are generally homogeneous throughout the lung so the sample site is not important; less is known about inflammatory airway diseases (IAD) and clearly focal pneumonia and abscesses are segmental diseases. However for routine transendoscopic BALF collection it is useful to always use the same bronchus. This author uses the right apical bronchus; 1st bronchial branch on the lower axial aspect (5 O’clock) of the right mainstem bronchus which gives reliable returns and wedging of the endoscope is easy. Bacteriological culture of tracheal washes is useful to identify potentially causative bacteria in suspected parenchymal or airway infections but culture results must be carefully correlated with the clinical and cytological picture. Streptococcus equi subsp zooepidemicus, S. pneumoniae, Actinobacillus/Pasteurella spp, Bordetella bronchiseptica and Mycoplasma fells and M. equirhinis cultured in significant numbers either alone or as mixed growths from tracheal secretions have all been significantly associated with IAD. BALF is more readily contaminated and unless harvested by directed sampling of a suspected focal septic, lesion culture results can be very misleading for the clinician. Detection of bronchial foreign bodies, often in association with coughing, fetid breath, infection and haemorrhage may be identified and removed transendoscopically. Focal swellings and discrete masses associated with infection, abscession or neoplasia may be investigated by biopsy or needle aspirate. Some lesions may be amenable to transendoscopic laser ablation. This presentation will discuss examination and sampling techniques, illustrate the normal anatomy and demonstrate common disorders of the lower airways.

**ANAPLASMA PHAGOCYTOPHILUM IN HORSES...AN EMERGING DISEASE OR UNDERDIAGNOSED?**

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**Introduction**

Equine granulocytic anaplasmosis (EGA) is a tick-borne disease, caused by the obligate intra-cellular bacterium *Anaplasma phagocytophilum*, which can elicit febrile disease in animals and humans. The disease has been referred to as equine granulocytic ehrlichiosis in the past, and is transmitted in Europe by *Ixodes ricinus* ticks. Ticks of the *Ixodes ricinus* complex also act as vectors to spread *Borrelia burgdorferi* from one animal to another, and co-infections of *A. phagocytophilum* and *Borrelia burgdorferi* have been confirmed in horses (Chang and others 2000).

EGA was first described in the United States in 1969 (Gribble 1969) and has since also been reported in Europe (Artursson and others 1999; Bermann and others 2002; Korbutiak and Schneiders 1994; Von Loewenich and others 2003; Butler et al 2008). Following an incubation period of around 10 days (Gribble 1969; Pusterla and others 2002; Korbutiak and Schneider 1994; Von Loewenich and others 2003; Butler et al 2008), an incubation period of around 10 days (Gribble 1969; Pusterla and others 2002; Pusterla and others 1999b), infected horses may experience sub-clinical disease or develop overt signs that include fever, depression, inappetence, reluctance to move and distal limb oedema. The disease can be self-limiting when untreated, and clinical signs usually last from 7 to 14 days (Gribble 1969). These clinical signs however are not pathognomonic for the disease, and demonstration of granulocytic inclusions, either morulae or initial bodies, in Wright-Giemsa or haematoxylin and eosin (H&E) stained blood smears can confirm a clinical diagnosis (Engvall and others 2002; Gribble and others 2002; Hutton et al 2008). Following an incubation period of around 10 days (Gribble 1969; Pusterla and others 2002; Pusterla and others 1999b), infected horses may experience sub-clinical disease or develop overt signs that include fever, depression, inappetence, reluctance to move and distal limb oedema. The disease can be self-limiting when untreated, and clinical signs usually last from 7 to 14 days (Gribble 1969). These clinical signs however are not pathognomonic for the disease, and demonstration of granulocytic inclusions, either morulae or initial bodies, in Wright-Giemsa or haematoxylin and eosin (H&E) stained blood smears can confirm a clinical diagnosis (Engvall and others 2002; Gribble and others 1999). These organisms can be found microscopically in peripheral blood only for a few days in the acute stage of the disease (Gribble 1969; Rikihisa 1991; Stannard and others 1996), and molecular techniques like polymerase chain reaction (PCR) (Engvall and others 1996; Pusterla and others 1999a), or serology (Artursson and others 1999; Van Andel and others 1998) can be valuable tools to confirm infection.
Recent research:
In 2007-2008 we have collected 147 ticks from 72 healthy horses. We have analyzed 104 peripheral EDTA- and serum blood samples, derived from these 72 horses, for the presence of *Anaplasma phagocytophilum* (and other tick-borne pathogens) by use of stained blood smears, PCR combined with RLB and IFAT (+ 4Dx Snaptest IDEXX). The ticks were identified and prepared for DNA extraction and PCR-RLB analysis. Our results show that a high percentage (15-20%) of horses has antibodies against *Anaplasma phagocytophilum* and 4% of these horses have antibodies against *Borrelia burgdorferi* as well. Identification of the ticks showed besides a lot of *Ixodes ricinus* also a few *Dermacentor reticulatus* ticks (vector for piroplasmosis). PCR-RLB performed on the ticks detected predominantly infections with *Borrelia burgdorferi*. *Anaplasma phagocytophilum* has been detected in only a few of the ticks we have collected from these horses. These results show that *Anaplasma phagocytophilum* is present in a high percentage of horses and a low percentage of ticks, which raises the question if ticks are the only vector for this pathogen.

References available from the author upon request

**NEW INSIGHTS IN DIAGNOSTIC IMAGING OF TENDON INJURIES**
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**Introduction**
Tendon injuries represent a common cause of poor performance and lameness in sport and race horses. Alterations in tendon structure can sometimes be difficult to diagnose based on history and clinical examination. Ultrasonography (US), tenoscopy/bursoscopy (TS), magnetic resonance imaging (MRI) and/or computed tomography (CT) are modern imaging methods used by equine practitioners to accurately define tendon injuries and to extrapolate the biomechanical function of the damaged tissue. Indications and advantages of each method will be discussed in this abstract with regard to the latest technology.

**Ultrasonography (US)**
US remains the technique of choice for first opinion evaluation when:
- Soft tissue swelling/deformation, pain or wound over a tendinous area
- Positive response to diagnostic regional analgesia with/without radiographic signs
- Effusion of tendon sheath or bursa

Although US is an operator dependent imaging tool, where excellent knowledge of anatomy is required, the equine practitioner can make a fast and non-invasive tendon evaluation for a relatively low cost. Linear and high frequency transducers (7.5-20MHz) are preferred. In some specific anatomical areas, curvilinear transducers, specialized preparation techniques and acoustic stand-offs are required.

The low water content of the tendon tissue produces an image of medium echogenicity on US. Scanning perpendicular to the tendon axis avoids false diagnosis of acute hypoechoic lesions (anisotropic artifact). However, permanent rocking of the probe can help to differentiate normal hyperechoic areas (endotendon septa) from scar tissue (such as observed in chronic injuries). Measurement of the cross sectional area is helpful when diagnosing diffuse and/or chronic tendon injuries.

It is very important to use a strict and systematic approach with US; which should include transverse and longitudinal scans. Technical difficulties can be encountered in some anatomic regions. Comparative examination of the contralateral limb is highly recommended. Dynamic evaluation can also provide information for partial tendinous tears and adhesions.

Although great progress has been made on image quality and spatial resolution, the operator should be aware that US does not generate an image containing 100% of the ultrastructural information. The image is not a direct view of the collagen fibers; however it does provide a pictorial representation of the changes in acoustic impedance throughout the tendon. 3D transducers, computerized US, ultrasonographic tissue characterization (UTC) and power/colour Doppler are novel techniques that can provide a more objective and quantitative evaluation of the tendon structure.

**Tenoscopy/bursoscopy (TS)**
TS is indicated when there is a positive response to diagnostic intrathecal/intrabursal analgesia. Tendon sheath/bursa effusion with/without US findings is also a clear indication for TS. Early referral of these cases is
recommended as the quality of exploration decreases with chronicity due to thickening of peritendinous tissues. Chronic inflammation also induces several irreversible changes that may affect the final outcome.

Although more invasive, TS involves exploration of various tendon sheaths and bursae with a small rigid tenoscope. This minimally invasive surgical technique allows evaluation of the tendon’s periphery (epitendon), mesotendons, synovial lining and gliding bone surface (bursa). Marginal tears and manica flexoria anomalies are often overlooked with US but can be better visualized by TS, as well as adhesions between tendons or between tendon and synovial lining. The absence of constriction syndrome can be confirmed in TS by the ability to freely move the tenoscope in specific areas such as the metacarpo/metatarsophalangeal and carpal canals. Advanced surgical expertise and excellent knowledge of anatomy is required to perform TS. The cost of the procedure, and general anaesthetic risk are possible shortcomings of this technique.

**Magnetic resonance imaging (MRI)**

Deep digital flexor tendon injuries in the foot and distal pastern are difficult to diagnose with US due to anatomical restrictions. MRI is then indicated for any lameness localized to the foot without radiographic abnormalities present. As disease modalities can potentially be complex, MRI is also indicated in the presence of foot pain with radiographic signs that do not fully explain the clinical signs and response to diagnostic analgesia.

The lower water content of tendons makes these structures appear as hypointense and homogeneous signals in all MR sequences. Acute tendon injuries appear hyperintense on T2- and T1-weighted sequences (although less intense/obvious on T1). Morphological changes associated with mild alterations in the intratendinous signal (hyperintense on T1, hypointense on T2) are characteristic of chronic tendon injuries. Tendon necrosis or diffuse/chronic injuries can be difficult to diagnose with US. Changes in the MR signal homogeneity may help to confirm such a suspicion in the metacarpo/metatarsophalangeal area.

MRI presents significant advantages over US and CT because of better contrast resolution of soft tissues and direct multiplanar imaging. However, several artifacts such as the magic angle can lead to false diagnosis. Interpretation of MR studies should be performed by well-trained operators. Elevated costs, relatively long examination time, poor availability of equipment and general anesthesia required for high-quality images are all potential disadvantages of this technique.

As shown for cartilage and early diagnosis of osteoarthritis, spatial characterization (mapping) of T1 and T2 relaxation times may have a role to play in early detection of degenerative changes in tendons.

**Computed tomography (CT)**

Indications for CT are similar to MRI. Normal tendons present a homogeneous low density (80-110 Hounsfield Units (HU)). Acute injured tendons appear larger, with reduced density, however this decrease in radio-density can be difficult to appreciate unless severe necrosis has occurred (40-50 HU). Injection of iodine-based contrast material in a peripheral artery can compensate to some extent for the lack of soft tissue contrast compared to MRI.

Neovascularization, increased blood flow or tissue permeability can be highlighted with contrast enhanced CT and improve detection of tendon injuries. Ionizing radiation, cost, availability of equipment and general anesthesia are all pitfalls of this technique. However, CT examination is short compared to MRI and offers an immediate therapeutic option to the clinician in the form of CT-guided injections.

**HOW TO MANAGE TENDON INJURIES SURGICALLY?**

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**Introduction**

Laceration, direct trauma from interference, peritendinous injury caused by over-tight bandages, and overload injury during exercise represent common causes of tendon injuries in sport and race horses. Although it remains unclear what is the best treatment for each type of injury, tenorrhaphy, tenectomy, tendon splitting, proximal check ligament desmotomy, tenoscopy/bursoscopy are surgical procedures commonly used in the man-
agement of tendon injuries with medical treatment and rehabilitation program. Indications, advantages and shortcomings of each surgical technique are discussed in this abstract.

**Tenorrhaphy**
Complete lacerations or lacerations involving greater than 50% of the flexor tendons are candidate for tenorrhaphy. The aim of this procedure is to restore tendon gliding function, and to minimize gap and adhesion formation. Following local debridement and extensive lavage of the wound +/- tendon sheath, the tendon edges are re-apposed using USP 2-0 absorbable monofilament as important as this tissue has a crucial role in healing process. Closure of the paratenon or tendon sheath also prevents adhesion formation between tendon and surrounding tissue. Following repair, the injured limb is immobilized in a cast for several weeks to allow the tenorrhaphy scar to gain sufficient strength. Then special shoe with high heel is applied and the foot is progressively brought back to a normal angle. Although the gap is minimized, the gap formation is unavoidable even with the best suture and immobilization technique. Disturbance of external/internal vasculature and tendon necrosis, local infection, cost and risk associated with general anesthesia are shortcomings of this procedure. 40-60% of the horses return to athletic function, depending on type/level of activity.

**Tenectomy**
Septic common digital extensor tenosynovitis can represent a therapeutic challenge in chronic cases. Long-term soundness without gait abnormality has been reported after radical resection of the entire intrathecal component of the common digital extensor tendon. Careful dissection of the palmar aspect of the sheath is important to respect the carpals joints integrity. Placement of a Penrose drain and application of a full limb palmar splint are part of the post-operative management.

**Tendon splitting (Ascheim)**
Longitudinal separation of tendon fibers is performed with ultrasound guidance/assistance on acute anechoic lesions to decompress intratendinous areas of hemorrhage. This technique can also be used on chronic lesions (granulation tissue or more mature scar tissue) to improve blood flow and/or to create some space 48h before intralesional injections. However deleterious effects on healing have been noticed on experimental lesions. Although indications remain unclear, this controversial technique is commonly performed on the standing horse under sedation and local anesthesia with nº11/15 scalp blades or 18-20G needles.

**Proximal check ligament (PCL) desmotomy**
PCL desmotomy is indicated for moderate to severe cases of superficial digital flexor (SDF) tendonitis, 3-6 weeks post-injury. This technique utilizes in the early phase of healing, the whole SDF musculotendinous unit as an elastic structure to protect the injured tendon. After 4-6 weeks, the transected PCL heals but in an elongated fashion. In theory this would negate the loss of elasticity of the scarred tendon. PCL desmotomy allows 51-71% of Thoroughbreds and Standardbreds racehorses to complete at least five or more starts. Open or tenoscopic approaches have been described but tenoscopic techniques largely decrease incisional complications. Increased risk of suspensory ligament injury, cost and risk associated with general anesthesia are shortcomings of this procedure.

**Tenoscopy & bursoscopy (TS)**
Exploration of digital flexor tendon sheath, navicular bursa, carpal and tarsal sheaths, bicipital and calcaneal bursae with a rigid tenoscope are minimally invasive surgical techniques that allow evaluation of the tendon’s periphery (epitenon), mesotendons, synovial lining and gliding bone surface. TS is indicated with acute or chronic tendon sheath/bursa effusion with/without ultrasonographic or MRI findings. Positive response to diagnostic intrathecal analgesia and suspicion of constriction syndrome represent other indications for TS. Longitudinal and marginal tears, *monica flexoria* anomaly and adhesions between tendons or between tendon and synovial lining are often overlooked with US. Mechanical debridement of fibrillated tendon fibers, surgical division of restrictive adhesions, annular desmotomy and flexor retinaculum release are performed under tenoscopic visualization. Advanced surgical expertise and excellent knowledge of the anatomy is required for those procedures. Cost and risk related to general anesthesia are shortcomings of these techniques. Prognosis varies with location, lesion type, duration, and activity type/level.

References available from the author upon request.
DIAGNOSTIC AND INTERVENTIONAL ULTRASONOGRAPHY OF THE SHOULDER REGION
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Shoulder region anatomy
The scapulohumeral joint is a large spherical joint surrounded by a relatively thin joint capsule. Cranially, the joint capsule is re-enforced by two thin glenohumeral ligaments but the vast majority of the joint stability is provided by the medial (subscapularis m.), cranial (brachiocephalicus m. & bicipital m.), lateral (supraspinatus m., infraspinatus m., teres minor m. & deltoid m.) and caudal (triceps brachii m.) musculotendinous units.

Indications for shoulder ultrasonography
Any lameness localized to shoulder area by obvious clinical signs (markedly reduced cranial phase of the stride, swelling or pain over the shoulder area, shoulder muscle atrophy). Any lameness that does not improve with diagnostic analgesia of the limb distal to the elbow. Ultrasound evaluation of the shoulder is advised before performing intra-synovial anesthesia of the region (joint or bicipital bursa) as it may confuse interpretation. Any history of trauma, wound or draining tract in the shoulder region.
Any lameness with radiographic or nuclear scintigraphic signs in the shoulder region.

Ultrasonographic technique
The shoulder joint is covered by a thick muscle mass and anatomic structures to image are relatively big, therefore a 3.5-5 MHz macroconvex transducer is the probe of choice to start the examination. Complete ultrasonographic examination of the shoulder region includes evaluation of the bicipital apparatus, the scapulohumeral joint and surrounding muscles and tendons.

The bicipital apparatus is best imaged with a cranial approach. The examination is started in transverse section at the level of the supraglenoidal tubercle. As the transducer moves distally, the bicipital tendon becomes bilobed as it runs between the cranial heads of the greater and lesser tubercles and over the underlying intermediate tubercle of the humerus. The tendon is heterogenous as it composed of fibrous and fibrocartilaginous fibers. The musculo-tendinous junction is also irregular. Careful comparison with the contralateral leg is advised to confirm any abnormal findings. Both transverse and longitudinal scans are necessary to fully evaluate this tendon. The bicipital bursa lies between the humeral fibrocartilage and the bilobed tendon. Fluid in the bicipital bursa is hardly visible in normal horses. In young animals, the proximal growth plate of the humerus is visible. The fibrocartilage covering the humeral tubercles has a medium echogenicity with regular hyperechoic spots. This transverse growth plate is in direct contact with the distal limit of the bicipital bursa. A longitudinal notch is also visible in the intermediate tubercle in foals, as a result of this complex growth plate. The scapulohumeral joint is better imaged with a lateral and caudolateral approach than a cranial one. As the articular surface of the scapula cannot be imaged, the lateral and caudal parts of the humeral head are the only articular surfaces visible ultrasonographically. This can be improved with protraction and adduction of the limb. With the probe in longitudinal section, the sharp edge of the glenoidal margin is visualized, followed ventrally by the smooth and rounded articular surface of the humeral head. A thin joint capsule covers the humeral head and synovial fluid is barely visible in normal horses. The scapula spine is then identified and the supraspinatus and the infraspinatus muscles are individually evaluated from their proximal to distal extremities. This evaluation includes the short and strong distal tendons and the small infraspinatus bursa located between the convexity of the greater tubercle of the humerus and the tendon. This bursa is invisible ultrasonographically in normal horses.

Bicipital apparatus – Abnormal findings
Special attention should be paid to the size and echogenicity of the biceps brachii tendon. The edges should also be carefully evaluated for tears (as with tendons running in the digital tendon sheath). Presence of hypoechoic areas or calcification is abnormal. The presence of fluid in the bursa is also usually considered abnormal. Thickening and proliferation of the synovial lining represent signs of inflammation. Accumulation of heterogenous material (fibrin) within the bursa is highly indicative of severe inflammation or sepsis. Fibrocartilage and growth plate evaluation is of paramount importance in young animals. Accumulation of fluid or heterogenous material in the fibrocartilage or cystic lesions in the subchondral bone is often indicative of seeded infection.
Scapulohumeral joint and surrounding muscles – Abnormal findings
Increased amounts of synovial fluid are commonly seen with articular inflammation. This is best seen at the caudolateral aspect of the joint below the margin of the humeral head. Pressure on the probe should be minimal to prevent collapse of the joint recess. Evaluation of the echogenicity can be helpful in estimating the degree of fluid cellularity. As for the bicipital bursa, thickening and proliferation of the synovial lining and thickening of the joint capsule represent signs of inflammation. Irregularities of the joint margins represent periarticular osteophytes, indicators of chronic inflammation. In general, ultrasonography is more sensitive and allows earlier osteophyte detection than radiography; but this is particularly true for this joint. Fracture of the scapular neck and glenoid margin are easily overlooked on radiographs. Interruption in the cortex of the scapula is strongly indicative of a fracture. Irregularity of the subchondral bone of the caudal lateral part of the humeral head, effusion and the presence of loose bony fragments are strongly indicative of osteochondrosis dissecans in young horses. Infra and/or supraspinatus muscle atrophy (decreased muscle volume, increased muscle echogenicity) and/or tendinitis are often associated with scapulohumeral instability as a cause or a consequence.

Ultrasound-guided procedures
As brachial plexus paralysis is possible with blinded local anesthetic injection, arthrocentesis of the scapulohumeral joint and bicipital bursa are more safely performed with ultrasound-guidance.

References available from the author upon request.

Stifle anatomy
The stifle comprises the femoropatellar joint (FPJ), where the patella glides over the femoral trochlea and the femorotibial joint (FTJ), where the femoral condyles glide over the tibial plateau. The congruence between these bones is improved by the strong fibrocartilaginous parapatellar apparatus and the presence of two menisci inserted between the femur and tibia. The FTJ is made up of a medial (MFTJ) and a lateral compartment (LFTJ). The fibula, strongly attached to the tibia, has no direct contact with the femur in horses. Fourteen ligaments provide joint stability but only 7 (3 tibiopatellar ligaments, 2 collateral ligaments and 2 cranial meniscotibial ligaments) are relevant for ultrasonography as they can be imaged properly. Images obtained from the cruciate ligaments are unfortunately not diagnostic. Communication between different synovial cavities has been reported to be 85% for FPJ-MFTJ, less than 20% for FPJ-LFTJ and 5% for MFTJ-LFTJ. The LFTJ has a wide communication with the subextensorius sheath.

Indications for stifle ultrasonography
- Any lameness localized to the stifle by obvious clinical signs (joint effusion or swelling, pain on palpation, flexion or retraction of the limb).
- Any lameness that improves with intraarticular anesthesia of the region as it may confuse interpretation.
- Any history of trauma, wound or draining tract in the stifle region.
- Any lameness with radiographic or nuclear scintigraphic signs associated with the stifle region.

Ultrasonographic technique
The anatomy of the stifle region is complex; therefore a standardized and systematic approach is required to evaluate each joint and compartment accurately. A 7.5 MHz linear transducer is the standard probe of choice. With the horse fully weight bearing, the FPJ is the first joint to be evaluated, starting with the 3 tibiopatellar ligaments (TPLs) scanned in transverse and longitudinal sections, from proximal to distal. The medial TPL has a triangular shape, the middle or central TPL is rounded and the lateral is wide, flat and relatively close to the middle TPL. The proximal parts of the TPL are fused with the joint capsule whereas the distal parts are surrounded by infrapatellar fat. It is not uncommon to see some hyperechoic lines within normal TPLs. It represents intraligamentary fat deposition and should not be mistaken for chronic pathologic changes. Comparison with the contralateral leg is advised. The medial TPL bends 90° proximally and fuses with the parapatellar fibrocartilage as it hooks onto the medial ridge of the femoral trochlea. The fiber pattern in this region is lost and can appear heterogeneous. The origin of the lateral TPL fuses with the gluteobiceps tendon. Careful evaluation of the bony origin and insertion is advised;
which should be regular and smooth as the patella apex. The lateral and medial and trochlea ridges of the femur are then evaluated in transverse and longitudinal sections. The subchondral bone should appear smooth and regular and be covered by a thin layer of cartilage on the medial ridge (<2mm) and a thicker layer of cartilage on the lateral ridge (2-4mm) in adult horses. In the intertrochlear groove subchondral bone and cartilage are frequently folded and irregular. In foals, the cartilage is thicker and the subchondral bone regularly irregular, indicating a normal ossification front. The synovial recesses are visible on the caudal aspect of the medial and lateral TPLs and they should normally dissipate when pressure is applied with the transducer. The synovial villi in the medial recess are also well developed in normal cases. The location and amount of synovial fluid and the appearance of the synovial membrane should be carefully evaluated. Comparison with the contralateral limb is often helpful. The suprapatellar recess can also be visualized through the quadriceps muscle insertion. This recess is particularly well developed on the medial aspect of the joint.

The MFTJ and LFTJ are initially evaluated with the horse weight bearing. The 7.5MHz transducer is first applied vertically between the medial TPL and the medial collateral ligament (MCL). The medial femoral and tibial condyles are covered by a thin layer (1-2mm) of cartilage and delineate a space where the triangular medial meniscus (MM) perfectly fits. The MM has a medium, relatively homogenous echogenicity. However, some small vessels running in the synovial membrane can produce enhancement artifacts that should not be mistaken for meniscal heterogeneity/injury. The proximal portion of the MM is less defined due to a small covering of synovial villi in normal joints. The abaxial surface of the MM is adherent to the joint capsule, delimitating 2 synovial spaces that communicate axially. Only the proximal space is filled with synovial fluid in normal weight bearing horses. The synovial membrane is thin and limited synovial villi are visible. The synovial fluid is hypoechogenic, with rare hyperechoic spots visualized in normal mature horses. The size of this recess should be less than 1.5-2 times the MM proximo-distal length. The MCL lies directly against the meniscus and should be evaluated from its origin to its very distal insertion (tibial metaphysis) in both longitudinal and transverse sections. As the MM curves cranially and caudally, slight tilting of the probe is necessary to remain perpendicular to the fibers.

On the lateral aspect, the same technique is employed, but the transducer is applied vertically between the peroneus tertius tendon (PTT) and the lateral collateral ligament (LCL). The lateral femoral and tibial condyles delineate a larger space where the trapezoidal lateral meniscus (LM) and the popliteus tendon fit. The LCL extends from the femur to the fibular head. Definition of the LM is always less clear than the MM. The weight bearing evaluation is concluded with examination of the PTT, its origin and the subextensorius recess that communicates with the LFTJ. No fluid should be visible in this recess in normal joints. The caudal stifle (curvilinear transducer) is not evaluated routinely as the image quality obtained is poor and generally non-diagnostic.

With the joint in 90º flexion and the transducer applied vertically, the cranial horn of the menisci is identified. By moving the transducer axially, the meniscotibial ligaments and tibial insertions are visualized. Slight tilting of the probe is necessary to get a proper image of the tibial attachment. The weight bearing surfaces of the condyles are then evaluated. A curvilinear transducer may be necessary in large horses. The subchondral bone, the condyle curvature and the cartilage should all be evaluated.

**Ultrasoundographic abnormal findings**

Increased amount of synovial fluid, thickening/proliferation of the synovial lining, and thickening of the joint capsule represent signs of articular inflammation. Evaluation of fluid echogenicity can be helpful in estimating the degree of fluid cellularity. A large number of hyperechoic spots in the synovial fluid may represent floating cartilage or meniscus debris. Irregularities of the joint margins represent periarticular osteophytes; which are indicative of chronic inflammation. Small chip fractures of the medial, lateral or proximal edge of the patella (which are difficult to diagnose on radiographs), are more easily identified on ultrasound as a clear interruption from the parent bone. Hypoechogenic lesions, loss of fiber pattern and irregular bone reaction on origins/insertions are indicators of TPL and CL desmitis. Periligamentous fluid accumulation can be seen in acute case of TPL desmitis. Interruption in the collateral ligament structure with/without avulsed fragments is seen with complete collateral disruption and severe joint instability. Subchondral bone irregularity associated with effusion and presence of mineralized cartilage flaps on the femoral trochlea and/or presence of loose bony fragments are strongly indicative of osteochondrosis dissecans in young horses. Any abaxial displacement of the menisci is considered abnormal as it is a sign of either joint collapse or serious damage to the meniscus or its fixation points. Other changes indicative of injury are changes in heterogeneity, fissure or tearing of the meniscus or its tibial attachment. Flattening of subchondral bone and...
cartilage thinning are indicative of femoral condyle disease. Thickening/thinning of cartilage with interruption of the subchondral bone plate is strongly indicative of a subchondral bone cyst in the femoral condyle. The defect is usually filled in by material of mixed echogenicity.

**Ultrasound-guided (UG) procedures**

Intrasynovial injections could be performed with ultrasound-guidance to improve accuracy of injection, especially when effusion and/or experience are limited. UG techniques decrease the risk of iatrogenic meniscal and cartilage injuries. UG injection of subchondral bone cyst in the femoral condyle can be performed with the horse anesthetized and placed in dorsal recumbency. The leg is fully flexed and using a microconvex transducer, a needle can be guided in through the cyst opening and inserted into the cyst wall.

References available from the author upon request.

**Ultrasoundographic work-up of the Acute Abdomen and an Ultrasound Glimpse at the Equine Thorax**

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Veterinary medicine has made significant advances over the last 20 years in the use of ultrasound for diagnostic work-up of the equine patient. Ultrasound was first introduced in the 1970s for reproductive imaging purposes, to monitor the mare’s reproductive tract and to optimize the time for breeding. In the early 1980s tendon imaging began to find its way, followed by not only echocardiography, but also diagnostic work-up of the equine abdomen and thorax. Nowadays many veterinary practices have their own fully equipped ultrasound apparatus, which makes the application of transabdominal and transthoracic ultrasonography more and more feasible not only in referral hospitals but also in first line equine practices. The goal of both presentations is to give a short review of specific landmarks that can be checked, backed-up with examples of characteristic images of the normal situation and a set of pathological conditions with which we can be confronted as equine clinicians and for which ultrasonography can provide important extra information. Performing routine transabdominal and thoracic ultrasound would be very time consuming in the hands of a non experienced person; however practical strategic use of transabdominal and thoracic ultrasonography, to check specific landmarks and even to follow-up certain pathologies in patients can offer interesting advantages.

**THE MARE – FOAL BOND: PRINCIPLES AND PRACTICES**

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In natural circumstances the mare-foal bond typically covers around 8-9 month, with birth – imprinting and weaning as the most vulnerable periods. The maternal bond still gives the youngster protection between 9 month and 18-30 month when he disperses from its birth family band. Birth is an important and critical period especially for the foal, not only physically but also behaviourally. A normal birth with an undisturbed imprinting period of several hours of both mare and foal leads to a close dam-foal binding (figure 1).

![Figure 1. Neonate foal maternal imprinting and - labelling](image)

Disruption of the imprinting and bonding process can for instance take place by accident (usually death of dam or foal); in case of the pathology of the mare or foal which requires essential neonatal veterinary care; unprofessional early handling; unsuitable housing; etc. Disrupted imprinting can lead to problems in bonding,
milk intake, insufficient maternal and protective behaviour of the dam as well as misdirected social and sexual behaviour later in life of the foal and therefore must strongly avoided. Depending on the time misimprinted foals are denied contact with other foals and conspecifics they will have increasing problems in understanding horse-specific behaviour. They can even become afraid of their own species and often will be incapable of reacting properly to other horse’s signals towards them, increasing the risk of conflict and injury. In case of unintentional disruption of the imprinting due to serious illness or death of the dam at or shortly after parturition, proper measures should be taken to minimise the above described effects. This can be done either by fostering the foal to another mare or bucket feed it in a herd of other mares and foals (figure 2).

Figure 2. Orphaned foal bucket fed but raised with other dams and foals

Various techniques which can used to accomplish this fostering will be shown during the presentation. Basically it is important to concentrate to create positive associations between the foster mother and the new foal. Some mares show various levels of aggressive behaviour against their new born foal to suckling (suckling rejection) or even are aggressive in general (foal rejection). Often these mares are either maiden mares or Arabian mares. It is possible that a combination of pain experienced during parturition, pain when touching a very full udder, neophobia and/or fear of the foal induces the aggressive reaction in the maiden mare. Together with appropriate clinical care, professional counter conditioning of both mare and foal the dam – foal bond can be (re-)established. During periods of separation of dam and foal due to veterinary care of a (neonate) foal, it is important to keep visual (and possibly olfactory) contact between dam and foal to avoid that the mare does not receive enough stimuli from its foal and the laborious (and risky) re-establishment of their band is needed. Care should be taken of linking the visual observation of the suckling behaviour since this is not correlated with the quantitative milk intake.

Many forms of handling methods at (very) early age or around weaning have been subject to research in order to see if and at what age this methods could be positive to the human-horse relationship or trainability later in life. They reveal that the time and type of contact all play a role, while recent studies suggest that the use of familiarized social models might be a great help through social facilitation.

Natural weaning takes place through gradual rejection of suckling by the dam around 8-9 month, dispersion from the family band takes place around 1.5-3 years. Waran et al (2008) have published a good review of the literature of the effects of weaning on the domestic horse. Under domestic conditions, weaning tends to take place normally between 4 and 6 months of age. The weaning process has been identified as associated with potential psychological, physical and nutritional stressors that are of welfare concern. The different types of stress especially associated with early weaning can also have long term impact on their performance, it can stimulate the development of stereotypic behaviour, own maternal behaviour, trainability, and it also can influence the anatomical development of the extremities.

The current best practice in domestic weaning with respect to foal and mare welfare will be presented and
is depending on many different local factors. However, several variables can be managed relatively easily during weaning in order to minimise stress responses. These include: early creep feeding to familiarise the young animal with the food it will be exposed to during weaning, feeding a high fibre diet, keeping the animal in extensive conditions using a gradual approach to weaning and allow to have social contact either with (preferably familiar) other foals or with older horses. The use of Equine Appeasing Pheromones during weaning will be presented.

Summarising, the quality of the natural strengthening (imprinting) maintenance (mare-foal husbandry) and weakening of the dam-foal bond (weaning) is of strong importance of the performance and human-horse relationships later in life and therefore measures should be taken to prevent undesirable side effects.

References
17. van Heel MCV, Kroekenstoel AM, VanDierenndonck MC, van Weeren PR, Back W. Uneven feet in a foal may develop as a consequence of lateral grazing behaviour induced by conformational traits. Equine Vet J 2006; 38:646-51.

DILEMMA’S IN EQUINE SPORT: BALANCING PHYSICAL HEALTH, MENTAL HEALTH AND PERFORMANCE
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Equine welfare in equine sports is a continuing topic for discussion and can have emotional advocates or protesters. The research concerning the welfare of animals (including horses) has developed from the declaration of the Five Freedoms in 1965 towards a more neurobiological based approach of the last decade2-5: the behaviour of an animal is regulated by its motivation and external stimuli. A motivational system is activated if the expected (genetically and/or ontogenetically determined reference) state differs from the actual or current state. Every response which diminishes the difference between the actual state and the expected state can be regarded as having a rewarding property (pleasure; positive affective state) and will
reinforce ongoing behaviour. In contrast, failure of doing so, which can be regarded as having no rewarding property (displeasure, stress; negative affective state) will lead to termination of ongoing behaviour and searching for alternatives, often leading to abnormal behaviours and/or abnormal physiological values. There are limits to adaptation, c.q. limits to the capacity to cope with external and motivational internal stimuli. The individual’s welfare is impaired at the moment that the individual is unable to decrease the discrepancy between the actual and expected state on the long term. It is often stated that every individual “aims” to maintain its homeostasis in continuously changing environments and situations and herewith controls its situation. Stress can be seen as an environmental effect on an individual which overtaxes its control systems.

Behaviours that are essential for an animal to perform have either short term goals (like increase blood glucose concentrations etc.) or long term goals (reproductive - and social behaviour, exploration). Studies have shown that these long term goals are safeguarded because they are regulated in the reward system in the brain (in the hippocampus): the release of endorphins (or dopamine) when performed. To determine which behaviours are essential an Ethological Need analyses can be done. For horses the outcome of this analyses shows that allogrooming and play, semi permanent access to forage (c.q. balancing saliva production through chewing with permanent secretion of gastric acids) and locomotion are essential basic needs. Because the brain structure in all mammals are similar and can not be changed by a few hundred years of selective breeding, this must be valid also for sport horses.

Many sport horses have to balance their essential basic needs with the challenges of their environment, usually determined by their riders/grooms (and/or owners). Not only their husbandry and management are often only remotely associated to the environment the horse needs to fulfil his basic needs, but they are also subject to regular (long distance) travel, sometimes large different atmospheric conditions in a short time span, repeated training or competition to the limits of their physical abilities, inconsistency of aids etc. It must be difficult to for the horses to continuously adapt and perform at their best and not get (chronically) stressed. There are numerous studies to the physical effects of all these challenges, but there are a limited studies looking into the mental challenges and mental possibilities to adapt. Controlled systematic studies of the behaviour of horses combined with physiological measurements (like Heart Rate Variability; changes in plasma concentrations of f.i. cortisol, ACTH or beta endorphin; lactate and growth hormones) can give insight in the mental (emotional) state of the horse in relation to performance, physical health and environmental challenges.

In one large study to determine parameters which can be used to determine early overtraining, controlled systematic behavioural observations were an important part of the study. In this study it was shown that overtraining was associated with neuro-endocrine imbalance and especially with behavioural changes in a standardised test. Many more of such studies need to be done in order to be able to develop reliable and pragmatic (practical) parameters to measure the mental state in a horse in relation to its performance, its possibility to adapt and its environmental challenges.

Another topic in the life of a sport horse is related to training methods. Several methods are criticised, but also in this field reliable scientific (mental) parameters are often lacking. The most recent discussion is the use of hyperflexion/Rollkur/LDR. The outcomes of a recent (political) discussion within the FEI lead to the conclusion that hyperflexion/rollkur should be “redefined as flexion of the horse’s neck achieved through aggressive force, which is therefore unacceptable. The technique known as Low, Deep and Round (LDR), which achieves flexion without undue force, is acceptable” (FEI release statement on rollkur dd 09/02/2010). However, it is not defined how much pressure (in N) represents “aggressive force” and what is an “acceptable force”. While there are suitable validated instruments to measure forces at different locations are developed in Australia, US and the Netherlands. But moreover the mental (and some physical) challenges (for instance lack of vision in the direction of movement etc.) related to both hyperflexion and LDR are not yet studied reliable enough to...
know how stressful or relaxing these are, and thus what role these play in the possibility for a horse to adapt to the stressors and challenges for this training method.

Veterinarians have a large toolkit to help the horses to overcome all these challenges, but there is a thin line between helping the horse to overcome the normal effects of training/performance and doping. Doping in performance horses can be defined as the “illegal application of any substance, except normal diet, that might modify the natural and present capacities of the horse at the time of the performance”. The discussion about the new rules from the FEI and other equine sport regulating bodies can be viewed from many different angles, but in most discussions the angle from the possibilities of the horse to be able to balance his essential basic needs with possibility to adapt to its challenges is lacking. A solution to start to get insight in this dilemma’s could be to develop a logbook in which daily (para)veterinarian treatments, physical and mental states – additionally assessed with repeated standardized (validated) behavioural and physical tests – are recorded.

References

HOW TO RECOGNISE AND TREAT PAIN IN THE EQUINE PATIENT
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Pain can be a very debilitating condition in the horse, which can lead to undesirable side effects, chronic pain or even euthanasia. Recognition and treatment of pain is a fundamental objective in veterinary practice. Pain management in the horse has been underestimated for a long time, but it is encouraging to see that an increasing interest has developed in this area. However, there are still considerable variations in the attitude and approach of pain management in horses. This is likely due to difficulties in assessing and quantifying pain in this species, financial considerations, unfamiliarity with certain drugs or techniques, and fear for negative side effects of certain analgesics. Colic and lameness are specific pain symptoms that are recognised relatively easy. Other signs related to pain, such as inappetence, reduced exploratory behaviour, weight shifting and tooth grinding are more easily missed. Pain scoring systems have been developed and evaluated for more objective and reliable measurement of pain. They vary from simple subjective scales.
to composite objective behaviour-based systems, each with their own limitations. The incorporation of a practical pain scoring system into daily pain evaluation might help to improve adequate pain therapy. The pain pathway which leads to the perception of pain after a noxious stimulus consists of transduction, transmission, modulation and perception. Analgesic therapy can be instituted at each level of the pain pathway. Early intervention and pre-emptive use of analgesics is advantageous and will reduce ‘wind-up’ and central sensitization. Depending on the type, intensity and duration of pain a specific method or therapy can be more desirable than another. The combination of two or more drugs or analgesic techniques that act on different sites in the pain pathway is called multimodal analgesia. This approach to pain management offers several advantages as analgesic drugs often work synergistic or additive. This results in use of a lower dosage of individual drugs and reduces the risk for unwanted side effects and toxicity.

**UPDATE ON THE USE OF NSAIDS AND OPIOIDS IN THE HORSE**

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The presentation will start with evaluating current clinical practice concerning use of NSAIDs and opioids in the horse, as we found in the survey that was performed last year. From the results of this survey we can ask ourselves the question whether there is need for more and newer NSAIDs for the horse. The release of more COX 2-selective NSAIDs like Firocoxib will be performed in the near future and we know that for instance Carprofen is also registered for horses in countries not far from ours. What will these new NSAIDs bring us that we do not currently have? Recent literature describes the efficacy of Firocoxib in orthopaedic (Doucet 2009) and in visceral pain (Cook 2009) in comparison to other NSAIDs.

Results of the survey learned that most of the equine practitioners use butorphanol as their favourite opioid. This probably has to do with the indications for registration: combination with a sedative for standing surgery and premedication and treatment of acute visceral pain. There are still practitioners that use methadone for these purposes, especially in combination with sedatives for small surgical procedures. There are very small numbers of veterinarians that use morphine and epidural techniques with opioids are also encountered very little. This means that opioids do not have widespread use for postoperative analgesia. The options with short acting systemically administered opioids like butorphanol are not very good. At this stage the side effects of opioids come into play. Therefore, local application and other administration techniques need to be used. Fentanyl-patches are being used in veterinary medicine for long time, mainly in cats and dogs (Hofmeister 2004). Recently, the clinical use of fentanyl patches in horses has been studied and various publications have described transdermal penetration through equine skin (Mills 2007), pharmacokinetics and analgesic properties of fentanyl in conscious horses (Orsini 2006).

Apart from NSAIDs and Opioids, there is some literature on other agents that could be used for equine analgesia. Tramadol, a synthetic analogue of codeine, has been used clinically for the last two decades to treat moderate to moderately severe pain in humans. Shilo et al. (2007) describe the pharmacokinetics in horses. Gabapentin, an anticonvulsant drug that is a structural analogue of GABA has been frequently used to treat neuropathic pain in people. The analgesic effects of Gabapentin in equine neuropathic pain have been described by Davis et al. (2007).

**ADVANCED PAIN MANAGEMENT: CONSTANT RATE INFUSION IN THE CONSCIOUS AND ANAESTHETISED EQUINE PATIENT**

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Constant rate infusion (CRI) maintains consistent plasma concentrations of IV analgesics and consequently produces a more constant analgesia. It also allows for reduction of the total dose that is administered and leads to fewer side effects. MAC of halothane and isoflurane in horses is reduced by 25-50% when lidocaine is administered intravenously by CRI (Doherty 1998, Dzikiti 2003). The antinoceptive effect of IV lidocaine was confirmed in an electroencephalographic study of castration in ponies under halothane anaesthesia (Murrell 2005) and a somatic thermal threshold study in awake ponies (Orsini 2003) and ponies with experimental dermatitis (Murrell 2005).
horses (Robertson 2005). A CRI of 50 mcg/kg/min after a loading dose of 1.3 mg/kg is generally used in awake horses. Ketamine, besides being a dissociative anaesthetic, is an NMDA receptor antagonist and at sub-anaesthetic doses possesses anti-hyperalgesic effects. It can be administered as a CRI in anaesthetized and awake horses at 0.4 to 0.8 mg/kg/h (Fielding 2006). In awake horses it was found to depress the nociceptive withdrawal reflex in an experimental setting (Peterbauer 2008). In a clinical setting it provided good analgesia for horse with burns and improved appetite and comfort in severely lame horses, although lameness severity did not change (Matthews 2004).

α₂-agonists like xylazine, detomidine, romifidine and medetomidine are potent sedatives, but also have analgesic effects. Their use as a single dose or CRI is mostly restricted to the peri- and intra-operative period, particularly if prolonged sedation is not desired. CRI of detomidine (0.18 mcg/kg/h) (Wagner 1992) and medetomidine (3.5 mcg/kg/h) (Bettschart-Wolfensberger 2001) both produce a MAC-sparing effect on inhalant anaesthetics of approximately 30%.

The systemic use of μ-opioids has been negatively influenced by inconsistent findings of their analgesic effect in MAC studies of inhalant anaesthetics and by reported side effects, such as excitement and increased locomotor activity in non-painful horses. In addition concerns about potential gastrointestinal stasis and colic exist (Senior 2004), although other reports did not find an increased risk related to the perioperative use of morphine. Butorphanol is a κ-agonist opioid that is widely used in horses. Although there is some controversy regarding its analgesic potency, a beneficial analgesic effect has been shown more recently in horses after celiotomy (Sellon 2004). Due to its short duration of action, it is recommended to be used as a CRI when prolonged analgesia is desirable.

Local analgesic techniques can perfectly be combined in the analgesic multimodal approach because they act synergistically with other analgesic classes of drugs and do not add to systemic side effects. This lack of systemic side effects is very beneficial when opioids are used for postoperative analgesia. Morphine results in relatively long lasting analgesia (18-24 hours) when administered epidurally or intra-articularly. Furthermore, it can be administered in combination with other analgesic agents like α₂-agonists (Fisher 2009) or local anaesthetics (Santos 2009).

Local anaesthetic blocks can be used to perform head surgery in the standing horse and long acting local anaesthetics like bupivacaine and ropivacaine can be used to prolong this effect in the first postoperative phase. Tremaine (2007) describes various local techniques for the equine head. Driessen et al. (2008) describe a technique for continuous perineural blockade of the palmar nerves in the distal equine thoracic limb. This technique is only at an experimental stage, but may be very interesting in patients with severe distal limb pain that don’t respond sufficiently to initial therapy with for instance NSAIDs. We encounter these patients regularly and local anaesthetic techniques, in conjunction with routine analgesic treatment, could probably lead to improved analgesia and welfare in our equine patients.

References


• Santos LC, de Moraes AN, Saito ME. Effects of intracutaneous ropivacaine and morphine on lipopolysaccharide-induced synovitis in horses. Vet Anaesth Analg 2009;36:280-286.


WHAT CAN YOU DO TO PROLONG IV ANESTHESIA?

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“The Usual”

Xylazine (0.4 to 1.1 mg/kg IV) is widely used for sedation and combined with ketamine (2.5 mg/kg IV) for short term parenteral anaesthesia because of ease of administration, cost efficacy, rapidity of onset and a short and generally predictable recovery following single doses. In stressed horses, usual doses of xylazine and ketamine may not cause adequate sedation and recumbency or result in a shorter than expected duration of anaesthesia.

Problems with Prolonged Field Anaesthesia

• limit anaesthesia time to 1 hour because prolonged, rough recoveries may result
• in the field, padding is minimal and positioning often poor
• in previously injured horses, post anaesthesia myositis is more likely to occur
• for prolonged anaesthesia administer supplemental \(O_2\) (eg 30 ml/kg/min via nasal tube)
• IV crystalloid fluids administered during anaesthesia to anaesthetised horses that have been injured will improve perfusion, tissue oxygenation and reduce the possibility of post-anaesthesia myopathy – typical anaesthesia rate = 10 ml/kg/hr.

Increasing duration of Xylazine & Ketamine anaesthesia

• If diazepam has not yet been administered, give 0.1 mg/kg IV
If too light (typically 10 to 15 min post induction),
• first give ketamine alone (about ¼ induction dose or
  0.5 to 1.0 mg/kg IV)
If need longer (typically 20 to 30 min post induction)
• then give another ¼ dose of ketamine PLUS ¼ dose
  xylazine (0.3 mg/kg IV).
Thiopentone 0.4 to 1 mg/kg – will stop swallowing
• and movement for 5-10 min.
Consider using Guaifenesin alone (25 to 50 mg/kg) IV to effect
• Start “Triple Drip” – takes 5 min to make up and
  should administered via a catheter
• For painful procedures, consider addition of a local
  anaesthetic infiltrative or regional nerve block
• Consider IV or IM administration of an opioid

“Triple Drip” for maintenance of anaesthesia

Problems with Guaifenesin:
• Administration of large volumes
• Perivascular irritation: GG is administered as a 5 or
  10% solution, which (esp 10%) causes haemolysis
  and endothelial injury. Perivascular administration
  will cause cellulitis and phlebitis. This can be mini-
  mized by dilution with similar or larger volumes of
  “perivascular” 0.9% NaCl.
• Phlebitis with 10% solutions “in irritated veins”: con-
  sider use of 5% solutions
• High doses (esp foals & miniature horses) result in
  renal damage (tubular obstruction from cast forma-
  tion and haemoglobinuria): give IV fluids to increase
  urine output
• Cardiac toxicity - seen at 3 to 4 times the therapeu-
  tic dose. Initial signs of toxicity include limb
  rigidity, limb movement and response to external
  stimuli (“light anaesthesia”).
• Differs from most other anaesthetics in that cardiac
  arrest (probably ventricular fibrillation) precedes
  respiratory arrest. Horses take large breaths prior to
death!

“Triple Drip” mixture for up to one hour of
anaesthesia in a 500 kg horse:
1. 25 gm Guaifenesin (usually available as a 10% solu-
   tion) - The mixture can be diluted with 250 mls of
   5% dextrose (= 5% GG) to increase the volume for
   infusion
2. 500 mg xylazine
3. 1000 mg ketamine

Administer via a standard IV giving set (20 drops/ml) at
a Drip Rate of 3 drops/sec.

What to do if the Triple Drip anaesthetic depth is
inadequate (“movement”)
1. Double the GG drip rate for 3 to 5 min, then slow to
   3 drops/sec.
2. Administer a bolus of ketamine: 0.2 to 0.4 mg/kg =
   100 – 200 mg / 500kg
3. Administer a bolus of thiopentone: 0.2 to 0.4 mg/kg
   = 100 – 200 mg / 500kg

SEDATION FOR DIFFICULT HORSES
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Responses of stressed horses
• They are more likely to injure themselves, handlers
  or veterinarians
• Stress causes catecholamine release and a “flight or
  fight” response
• High circulating catecholamine levels cause CNS
  stimulation, increasing the requirement for general
  anaesthesia drugs
• In addition, preferential perfusion of muscle (vs vital
  organ perfusion) causes redistribution of sedative
  and anaesthesia drugs to this muscle tissue, rather
  than the brain, resulting in a need for large doses of
  drugs to achieve sedation or anaesthesia which is
  generally of poorer quality than in relaxed horses.
• These horses may be at greater risk for post anes-
  thetic myopathy
• The study of anaesthesia related mortality (The Ani-
  mal Health Trust, Newmarket, UK) showed that pre-
  medication with acepromazine decreased the mor-
  tality risk. Acepromazine provides moderate
  sedation with a long duration of action (2-4 hrs) and
  some vasodilation.
• Intractable or stressed horses can be difficult to
  approach to enable injections
• Normally, we would like to do a basic physical exam
  of all patients prior to anaesthesia, although this
  may not be possible in these types of horses. Cer-
  tainly, it is difficult to obtain normal resting parame-
  ters for heart and respiratory rates, body tempera-
  ture and white cell counts.
Administration of sedatives or analgesics then either
allowing the horse some time to itself in a stall gener-
ally or leaving it in the company of its usual equine
“friends” results in better sedation (lower catecho-
larine levels), compared to IV administration and
immediately commencing work on the patient or
attempting to induce anaesthesia.
Commonly in our race track practice we use combinations of acepromazine, an alpha-2 agonist (usually xylazine in the peri-anaesthetic environment) and opioids, most commonly methadone. The following table shows the combinations of sedatives we use on a variety of patient “types” commonly using either 1 & 2 or all 3 drugs in combination.

Useful combinations for Sedation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose mg/kg</th>
<th>Route</th>
<th>Mare or Gelding</th>
<th>Yearling</th>
<th>Colt or Stallion</th>
<th>Foal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acepromazine</td>
<td>0.03</td>
<td>IV</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>0.01</td>
<td>IM</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Xylazine</td>
<td>0.3</td>
<td>IV</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
<td>1</td>
</tr>
<tr>
<td>Xylazine</td>
<td>1.1</td>
<td>IM</td>
<td>+/-</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.1</td>
<td>IM</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

Onset min. 15 15 10 3
Duration min. 120 120 40 to 60 20

Specific Case Examples will include:
1. Sedation of yearlings for radiography or elective orthopaedic surgery
   Yearlings can be difficult to handle and sedate, generally because they are inexperienced.
2. Sedation of traumatized horses
   An example would be a horse that has suffered acute trauma and requires wound repair. Allowing the horse some time to itself in a stall may result in the horse becoming less stressed and administration of sedation or analgesia at that time will produce better results, compared to IV administration and immediately commencing work on the patient. Elevation of circulating catecholamines alters perfusion, pharmacodynamics and also increases the risk of myositis.
3. Sedation of intractable horses
   These horses have the potential to seriously injure the operator either because of fear or aggression.

THE WORK UP AND THERAPEUTIC CHALLENGE OF LAMENESS IN THE SHOULDER AREA
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Lameness associated with the shoulder region is not as common as distal limb lameness. Shoulder pain associated with the swing phase generally results in a shorter stride to minimize shoulder extension and flexion. Horse with shoulder lameness are more lame when the affected leg is at the outside of a circle. Diagnosis of shoulder lameness can be challenging because mild lameness may be difficult to localize. Muscle atrophy at the shoulder region is a typical finding.

Intra-articular analgesia of the shoulder joint usually improves the lameness but rarely alleviates it. 20 ml of anaesthetic solution has to be used, in some horses there is a communication with the bicipital bursa. Rarely an instability of the shoulder appears after applying the block. The bicipital bursa is large so needs also 20 ml of anaesthetic solution.

Osteochondrosis of the scapulohumeral joint
Often clinical evident at young age giving a moderate to severe lameness whereby often the foot changes to a more upright foot. Radiographically the glenoid cavity of the scapula and the humeral head will flatten and results in a loss of congruity, irregular lucent zones in the subchondral bone and blurring of the joint space. The degree of radiographic changes correlates to the clinical signs. The prognosis even after surgery at young age is not favourable.

Subchondral bone cysts
Causes lameness mostly in young horses. Small lesions can respond to treatment, treatment is mostly unrewarding in older horses.

Osteoarthritis
Osteoarthritis in shoulder joints in competitions horses is uncommon. Intra-articular analgesia normally gives a significant improvement of the lesion. The response to therapy depends on the underlying cause (osteocondrosis, intrarticular fracture, tearing of the joint capsule).

Osteoarthritis in miniature breeds
Shetland ponies, falabella and miniature ponies have more frequently an osteoarthritis, sometimes with more advanced radiographic changes. Dysplasia of the
scapulohumeral joint has been described in these breeds.

**Luxation**

Luxation of the scapulohumeral joint results in acute onset severe lameness and is seen more in ponies.

Fracture of the supraglenoidtubercle of the scapula is a fracture that occurs in young horses. Lameness will be acute at onset and severe. More chronic cases can have a more subtle lameness. Conservative treatment will mostly result in a pasture sound animal. Surgical excision or internal fixation in combination with a transection of the bicipital tendon are the treatments options.

**Tendonitis of biceps brachii and intertubercular bursitis**

Horses are presented with mild to severe lameness. Characteristic is the discomfort the horse has with the anterior stride of the leg. The area is painful on palpation.

A significant portion of these horses have an infection of the bursa, the clinical signs of these horses are more severe. Even with severe changes these horses can make a full recovery after surgical intervention.

**Damage to the suprascapular nerve**

The nerve wraps around the cranial aspect of the neck of the scapula and is vulnerable to trauma. Atrophy of the supraspinatus and the infraspinatus muscle can occur within 7 days.

Surgically this nerve can be decompressed

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**THE WORK UP AND THERAPEUTIC CHALLENGE OF STIFLE LAMENESS**

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In our competition horses the stifle is one of the joints that has a major contribution to lameness and poor performances, in young horses but also in older horses in the higher level performances.

The anatomy of the stifle has some typical features as the incomplete ossification of the femoral trochlea and the patella at young age. The reciprocal apparatus with the peroneus tertius dorsally and the digital flexor tendons caudally, the parapatellar fibrocartilage of the medial patellar ligament enabling the hooking of the patella. The stifle contains three joint spaces, the femoropatellar joint and the medial and lateral femorotibial joints, diffusion of mepivicaine between all compartments may occur in about 75% of horses. The medial and lateral meniscus, the caudal and cranial cruciate ligaments and the collateral ligaments can also be the cause of serious injury.

In the lameness workup the anamnesis can elute more typical signs of stifle problems as falling of the lead, difficulty with lead changes, feeling more lame at canter, reduced anterior phase of stride. In the lameness examination it is important to observe the lameness thoroughly and so picking up some of the characteristics of stifle lameness’s. Carrying the stifle more abducted is one of them and stifle lameness can be more obvious under the saddle then in hand, and typical will be more easily noticed by the rider.

It is important to carefully examine the limb for joint distension, capsular thickening, for swelling and pain over the ligamentous structures.

In addition to the flexion test, manipulation tests can be used as the collateral ligament test, stifle stretching, and in some cases the patellar displacement tests.

Diagnostic analgesia is a very important tool to confirm the location of the lameness. Due to the variety of diffusion of local anaesthetic through the three compartments as a general rule a positive block on one of the three can not exclude the other two as the site of injury and for a negative block all three compartments must be blocked with at least 20 ml anaesthetic solution. I do trot up the horses directly after injection and then for every 5 minutes up to 30 min after injection. Severe lameness’s in general and medial collateral ligament injuries and bone cysts can show a more variable response to joint blocking.

Radiography is the imaging modality of first choice, a high powered x-ray generator together with high quality digital system will produce excellent quality views. Lateromedial, flexed lateromedial, caudal 30 lateral craniomedial oblique views, caudocranial and in selected cases skyline of the patella are the standard views of choice.

Soft tissue injuries are a large portion of the stifle injuries and giving ultrasonography a very important diagnostic role in stifle injuries.

**Septic arthritis**

The stifle is one of the more often affected joints in foals with septic arthritis. In adult horses the cause of the infection is not only haematogenous but also traumatic or secondary to joint injection. The specific anatomy of the stifle makes it more difficult to deal with the infection.

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Flushing the joint and arthroscopic cleaning out of the joint is the treatment of choice together with systemic and local antibiotics therapy. In more refractory cases antibiotic impregnated devices can be of help. In osteomyelitis cases in foals it is important to do a careful debridement whereby the essentials debris is removed without damaging the integrity of the joint.

Osteochondrosis
Osteochondrosis of the lateral femoral trochlear ridge is a common feature. It can be asymptomatic but can also cause lameness varying from subtle gait deficits to marked lameness. It development start in foals from 3-9 months of age and signs and severity can vary in time. Foals can be treated with correction of dietary imbalances and confinement. In selected cases it can be of value to surgical fixate detached cartilage flaps in foals. Arthroscopic debridement is the therapy of choice but preferable the horses are more then 15 months of age. The prognosis is reasonable good but depended on the area involved and the secondary changes in the joint.

Subchondral cystic lesions
Subchondral bone cysts and osseous cyst like lesions can be a form of developmental disease but trauma is also an important factor. The therapy of choice has been arthroscopical debridement but arthroscopic guided corticosteroid injections has been suggested as alternative. The prognosis is also depended on the conjoining joint disease.

Upward fixation of the patella and delayed patellar release
The condition is more common in horses with a straight hind limb conformation. It is more common seen in young horses and ponies especially if they are in poor condition, poorly musculated. An important part of the therapy is to get these horses to be shod, change the foot conformation to a more upright foot whereby the medial toe is lowered or there is an unilateral lateral heel wedge applied. The medial patellar ligament is thickened by applying an iodine in oil blister or surgically split the ligament longitudinally. It is important to improve body condition and stimulate the muscle formation by training and/or testosterone derivates. If this therapy is not successfully or the patella can not be unlocked the medial patellar ligament desmotomy has to be performed in the standing sedated horse under local analgesia.

Cranial and caudal cruciate ligament injury
Collateral ligament injuries
Meniscal and meniscal ligament injuries
Three different entities that have a lot in common and are also seen together, often in combination with a certain amount of (secondary) degenerative joint disease. They are often presented with a history of an acute injury, the lameness can vary from mild to severe. Arthroscopic surgery and/or intensive therapeutic and supportive management is necessary to get these horses back on their intended level in their athletic career. These horses are supported by joint treatment with corticosteroids and hylaluronic acid and frequent IRAP treatments. These joints can be supported with intramuscular adequan or pentosan treatment and/or nutriceuticals. Selected ligament and capsular blistering can also be used to improve joint stability.

THE APPROACH TO THE EQUINE DERMATOLOGY CASE IN PRACTICE
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Dermatological disease in equine practice is always a challenge. Veterinarians will be a called to see chronic skin cases more that those of acute onset; most owners will either have ignored it (in the hope that it would “go away” or will have applied some medications (usually ill-advised and inappropriate). This not only makes the investigation and diagnosis more difficult but often confuses the treatment also. A full history must always be taken! This should establish all owner-applied measures as well as an appraisal of the major clinical presentations and the extent of peer / other species involvement in similar states. A logical clinical approach is also essential – many of the conditions can be easily and definitively diagnosed with clinical and historical information alone. Intuitive supposition can be useful but there are significant dangers because many conditions have a common presenting clinical appearance. Only once a diagnosis has been established can treatment be expected to succeed. The good thing about equine dermatology is that many of the conditions can be treated or at least they have a reasonably well defined prognosis. However, relatively little is known about many of the equine
skin diseases – extrapolation from other species and misleading, unsupported naming of diseases such as Systemic Lupus Erythematosus and Cushing's Disease may lead to problems when the treatment is also extrapolated to the horse. The diseases invariably have major clinical and / or pathological differences from other species and so care should be taken when making the diagnosis; perhaps it is justifiable to emphasise the species specificity and the similarity (rather than the identical nature) of the disease by using the term Equine and “-LIKE” in the names. For example, Equine SLE-like Syndrome implies that the clinical presentation is specific for horses and that a panel of symptoms (hence the term syndrome) shows some similarity to those seen in other species in which the pathology is better established.

Diagnostic aids including specimens of hair, brushings, scrapings and biopsies are frequently taken in equine dermatological investigations; these include skin scrapings, hair plucking and biopsies of various types. In some cases a definitive diagnosis can be established while in others the chronic nature of the condition and / or the complications caused by secondary trauma or infection make them difficult to interpret. Pathologically useful information is usually restricted to early, carefully selected lesions and correct sampling methods.

For the most part equine skin disease can be usefully divided into infectious and non-infectious disease. The former involve virus, bacteria, fungi, protozoa and internal and external parasitic conditions. The latter include traumatic and allergic / immunological skin disease as well as developmental / genetic conditions, endocrinologic changes, and neoplastic disease. A few equine skin conditions can be due to nutritional and neurologic problems and others are secondary to vascular disease, and iatrogenic damage to the skin.
expects the pathologists to “make a firm diagnosis” is both professionally unsound and often counterproductive. Access to a good pathologist is however paramount and provided that all the correct available information is submitted along with the correct samples that truly represent the condition, should facilitate the whole process of diagnosis and treatment.

The six major categories of equine skin disease are:
1. Moist / exudative(crusting) dermatoses
2. Dry (scaling and flaking) dermatoses
3. Nodular skin disease (neoplastic and non-neoplastic nodules)
4. Pruritus
5. Hair coat density alterations (Alopecia & hair loss/hirsutism)
6. Pigmentary changes

Treatment of equine skin disease has been significantly, and adversely, affected by the gradual erosion of the therapeutic measures that can be taken. Nevertheless, the equine skin is tolerant of damage and will usually recover reasonably well if the inciting cause is removed. In some cases this is easier to say than do – secondary changes and iatrogenic interference confuse the treatment markedly and in some conditions the cause cannot be treated. Secondary skin disease, such as hepatic-derivation photosensitisation (HDP) simply points the clinician in the direction of the (usually) more important underlying disease. Secondary changes resulting from iatrogenic or self traumatic interference, may force the clinician to address the secondary effects first and then to restart the diagnostic process when the clinical syndrome is clearer.

SUMMARY
A logical and thorough clinical investigation should provide the best basis for the diagnosis of skin diseases. Where no diagnosis can be reached in spite of a full range of investigations, the clinician can justifiably attempt symptomatic treatment but otherwise it is far better to focus treatment on a specific condition. However, in equine dermatology there are few text descriptions of the majority of the conditions encountered in practice and whilst a few diseases are well recognised there is a still little consensus on the best treatments. There is no substitute for experience and reference to text books and colleagues who might have encountered the condition before.

Further Reading
Knottenbelt DC, Pascoe’s Principles and Practice of Equine Dermatology WB Saunders London. 2009

AFRICAN HORSE SICKNESS - CURRENT PERSPECTIVES FOR EUROPE
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Introduction
African Horse Sickness is a non-contagious, seasonal, Orbivirus/Arbo-virus disease of horses which is biologically transmitted by a restricted range of Culicoides spp. insects., and which is presently confined to areas of Africa and the Middle East. The disease is endemic in East and central Africa; it is not endemic in most regions of South Africa but makes regular incursions into southern Africa during the rainy season (January to March).
likely to be the first to encounter the disease (i.e. veterinarians) are well versed in recognition of the clinical signs.

Nine distinct strains and 42 sub-strains (which have some serologic cross over) have been identified - all 9 have been identified in southern sub-Saharan Africa. Each strain has its own inherent pathogenicity / virulence. Serotype 9 is the predominating type in northern Africa and serotype 2 has recently been isolated in some West African countries. These outbreaks indicate that there may be some changes developing in the virus and its traditional distribution. Whether this is related to adaptation to different vector species is uncertain but the consensus is that *Culicoides imicola* is the most dominant vector; *C. bolontinos* may also be involved but there is no suggestion that other species are significant at present at least. Outbreaks have occurred outside Africa, such as in the Near and Middle East (1959-63), in Spain (1966, 1987-90) and in Portugal (1989) and Morocco (2006). So far no further incursions have occurred into Europe.

Figure 2: Reservoir hosts including zebra (highly significant), elephant (probably insignificant) and donkeys (possibly significant) have been suggested.

The disease has a high morbidity and mortality in horses and more so in naive individuals and is highly seasonal occurring only when the vectors are active. Donkeys and mules are less affected. Most outbreaks are attributed to carrier species such as zebra; zebra populations act as a concentrating and expanding reservoir of infection. Large populations of donkeys may also be a reservoir of infection. There is probably no reservoir status in horses.

Four different clinical syndromes are described but the same strain in the same year can cause very different signs in individual horse. Signs are not pathognomonic in any way, closely resembling other acute viral diseases.

1. **Subclinical Form (Horse sickness Fever):** This is associated with milder strains / infection in partially immune animals and vaccinated horses. The signs, include:
   - ‘FLU like symptoms
   - Fever (40-40.5°C) for 1-2 days

   - General malaise
   - Vaccinated or exposed horses
   - Mild / transient fever
   - Some head / eyelid swelling
   - Conjunctivitis / chemonis
   - Leg filling possible (not common)
   - Transient lethargy and depression

2. **Subacute or cardiac form (‘Dikop’):**
   - Insidious onset with persistent fever / Fever (39-41°C)
   - Oedema of head and neck (Dikop) / Swelling of eyelids, supra-orbital fossae, lips / Swelling of the supraorbital fossa, eyelids, facial tissues, neck, thorax, brisket and shoulders.
   - Congested mucous membranes (petechiation) / Cyanosis
   - Mild colic
   - Dysphagia (pharyngeal / esophageal paralysis)
   - Hydropericardium (cardiac tamponade)
   - Some cases survive -long recovery (>1 year)
   - Appetite remains good!
   - Death usually within 4 – 5 days (often within 1 - 2 days) Slow death (>50%)

3. **Acute / respiratory (pulmonary) form (‘Dunkop’):**
   - Fever (40-41°C), Sweating
   - Pulmonary oedema / dyspnoea, spasmodic coughing, dilated nostrils with profuse nasal discharge (serous) with postural flow
   - Redness of conjunctivae, Cyanosis
   - Recumbency
   - Appetite remains good up to death!
   - Death from anoxia within hours or days

4. **A mixed form (cardiac and pulmonary):**
   - Pulmonary signs of a mild nature that do not progress,
   - Oedematous swellings and effusions,
   - Death from cardiac failure, usually within 1 week

A nervous form may occur, though it is rare and it may in fact be a secondary complication of cerebral oedema and hypoxia. Mortality in horses is around 70-95%, in mules around 50%, and in donkeys around 10%. The disease can also affect dogs which consume infected horse meat.

**Diagnosis**

Any suspicion MUST be reported to the appropriate authority in all European countries. Confirmation relies on clinical recognition and submission of samples including blood and specimens of lung, spleen, lymph...
Abstracts European Veterinary Conference Voorjaarsdagen 2010 - Amsterdam, Netherlands

61 MINUTES – A SPECIAL NEWS PROGRAM

The global economy is in crisis, technology advances are changing the way we do business, the social media craze is taking off, lifestyle and generational trends are new challenges we all face; these topics and more are dominating the news today. Find out how they are influencing the decisions you make as an equine practitioner during “61 Minutes” – a news section tailored to the equine veterinary profession. Modeled after a popular U.S. news hour, this program brings to light current trends in technology, finance, business and lifestyle climates from an equine veterinary perspective. Robert Magnus, DVM, MBA (USA), Mike Pownall, DVM (CANADA) and Joop Loomans, DVM, MS, Ph.D. (NETHERLANDS), will guide you through this thought provoking session. What you hear may surprise you, and provide the insight you need to make proactive changes in your practice management operations. Join us for “61 Minutes” and find out how to reduce the financial impact of our shared economic troubles in the equine marketplace.

BRANDING OUR PROFESSION AND PRACTICE

Where do we stand as an equine veterinary industry? What is our customer’s perception of our profession and of our practice? Follow William Lowell as he guides us on a tour of image and brand awareness. Learn from branding successes and mistakes made by corporations in other industries. This session will give you insights and advantages to your organization, whether you are a solo practitioner or a large multi-vet practice, whether you practice in Europe, Australia, Japan, South Africa or North America.

Summary

There is genuine reason for fearing AHS but we also need to put it into perspective. Currently the disease is not even near to us and provided that we are prepared to monitor our populations of horses and donkeys and provided that we are able to diagnose (or at least suspect) the disease in its early stages we should be able to control its spread across Europe. Any outbreaks that occur in Europe would be short lived - however devastating they were at the time. ‘Further northerly extensions in the range of Culicoides imicola, in response to ‘climatic moderation’, cannot be ruled out and could substantially increase the area of Europe ‘at risk’ to AHS’.

Treatment

Treatment is very difficult. Diuretics which have been used historically, are in fact specifically contraindicated. Fluid therapy exacerbates the severity of the clinical syndrome and corticosteroids are useless. The best approach is to minimise stress and provide very high quality nursing care. The slightest exertion can easily cause sudden death. Concurrent bacterial or protozoal infections are common and so these may also have to be addressed. Surviving horses need at least 6 - 8 weeks of rest before being returned to light work.

Control

A vaccine strategy has been largely successful in sub Saharan Africa. Where the vector extends its range the disease is likely to follow and where the vector exists, the simple act of importing a carrier zebra or an infected horse for example may result in a severe (if localised) outbreak. What is needed is a far better vaccine – fortunately the commercial pressures and almost paranoid fear of the disease in Europe has triggered a real effort to create a better vaccine. A vectorised vaccine using canary pox or vaccinia is currently in the advanced stages of testing. It is a pity that this was not developed many years ago so that the poorer countries animals would be protected better... but then of course we weren't really concerned for either the people or the animals of the endemic countries!

Node and heart muscle collected at necropsy and stored fresh at 4°C. Current tests can provide a definitive diagnosis within a few hours although there are no definitive histological features at any stage. Recently a rapid immunoperoxidase staining method has been reported.

Abstracts European Veterinary Conference Voorjaarsdagen 2010

Proceedings of the European Veterinary Conference - Voorjaarsdagen, 2010 - Amsterdam, Netherlands
**Introduction**

Immunoprophylaxis is the prevention of infectious disease through induction and enhancement of specific protective immune responses. Immunity can be either passively or actively acquired. However, the preferred approach is to induce protective responses actively through the administration of vaccines. Different types of vaccines are available such as inactivated or modified live whole pathogen vaccines, subunit vaccines, inactivated microbial product vaccines such as toxoids, and genetic material encoding for expression of protective antigens (DNA vaccines, vector vaccines).

**Immune response**

Active immunisation starts with the administration of a primary series of vaccinations to prime and subsequently booster the immune system. On subsequent exposure to the specific pathogen, memory cells can be recruited to quickly generate specific antibodies and effector cells such as cytotoxic T-lymphocytes. Only some vaccines are able to induce sterile immunity, in which case infection and replication of the pathogen are completely blocked. Most vaccines induce clinical protection with more or less significant reduction of pathogen excretion in vaccinated animals.

Protective immune responses can be either predominantly antibody mediated, cell mediated or a mix of both arms of the immune response. The nature of protection depends on the pathogen and pathogenesis. In many vaccines adjuvants are included. Adjuvants can be broadly divided in two classes, based on their principal mechanisms of action: vaccine delivery systems and immunostimulatory adjuvants. In general, adjuvants appear to exert their effect by enhancing antigen presentation, improving antigen stability, or acting as immunomodulators. Immunomodulation is achieved by altering the cytokine network, directing the immune response towards a T-helper cell type 1 (cell-mediated) response or T-helper cell type 2 (humoral) response.

**Safety and efficacy of vaccines**

Vaccine safety is established by testing for sterility, toxicity, freedom from extraneous agents, and confirmation of the identity of the antigens or organism(s) included in the vaccine. Field safety studies should be carried out to confirm that the risk of inducing local or systemic adverse reactions is at an acceptably low level. Safety may be to often over-emphasised in comparison with efficacy.

Vaccine efficacy is measured either direct after challenge with the pathogen involved, or indirect after measuring the level of antibodies induced after vaccination. Efficacy is most of times only measured some weeks after vaccination at the peak of the immune response, and information about duration of immunity is consequently lacking in many cases. Other important aspects that can influence vaccine efficacy in the field are interference of maternally derived antibodies (MDAs), diminishing immune responses due to ageing, and more or less risk based vaccination strategies. These aspects will be discussed.

**Passive immunisation**

Passive immunisation can be realised either through the natural passive transfer of maternal immunity through the uptake of colostrum, or through the use of exogenous antibodies. Also active immunisation of the mare during pregnancy can be a strategy to induce high levels of maternally derived antibodies in the foal. This is particularly important in prevention of infection with enteric pathogens such as rotavirus, *E. coli*, and other enterobacteriaceae. Some examples will be given.

**Vaccination of foals**

It has become more and more evident that MDAs exert a profound inhibitory effect in the immune response of foals to vaccines, even if only traces of antibodies are present. Therefore, it is important to realise that there will always be a window of susceptibility between the waning protection derived from MDAs and the protection derived from active immunisation. The best moment for primary immunisation will depend on the primary focus: either to protect the foal and weanling against specific high-risk infectious diseases that infect this age group or to initiate primary immunisation to protect against disease later in life. For the first category, a high and uniform level of MDAs is desirable implicating booster vaccination of the dam before foaling, good transfer of MDAs, management practices to reduce exposure to the infectious agent etc. Specific examples of both categories will be discussed.
Core and noncore vaccines

Core vaccines are directed against diseases against which all horses should be vaccinated in a certain geographic region. Noncore vaccines are vaccines that are not considered “a must” for the equine population as a whole, but can be used and even strongly recommended for certain subpopulations or when serious problems with certain infectious diseases have been and are experienced. The definition of core vaccines will highly depend on the geographic region/country and the equine infectious diseases that are endemic.

Vaccination guidelines

Vaccination guidelines are non-compulsory recommendations intended to assist the veterinary practitioner in the efficient use of vaccines. They complement the official information contained in the summary of product characteristics (SPC) that are found in the package inserts. The authority of these committees is based on the experience of their members, who must be recognized experts in the field of infectious diseases, vaccinology or and internal medicine. There is a need to make a link between the authorized characteristics of the vaccine as described in the SPC and the package insert, the scientific knowledge available in published literature, and the requirements of veterinary practice. To this end, committees have been created upon the initiative of professional associations or experts in the various fields.

Several committees are already at work, in companion animal medicine for example: the Feline Vaccine Advisory Panel of the American Association of Feline Practitioners, and the European Advisory Board on Cat Diseases (ABCD). These committees write general and specific recommendations for good vaccination practice and remain independent of any brand or any particular commercial product. Different committees will arrive at different guidelines and recommendations. These differences can be explained by varying veterinary approaches, depending on the country or region: cultural aspects (e.g. with regard to the importance of animal welfare), the level of medical care and the available vaccines. Also, there are so far no rules about how to control infectious diseases, and the expert opinion, as reflected in the guidelines, is based on a consensus - which may differ, depending on the composition of the expert panel Horzinek, Padova) To my knowledge there is not an internationally recognised Advisory Board on Equine Diseases. The establishment of such a committee should be encouraged.

Suggested reading


EQUINE HERPESVIRUSES 1 AND 4: PATHOGENESIS, EPIDEMIOLOGY, DIAGNOSIS AND (IMMUNO)PROPHYLAXIS

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Introduction

Equine herpesvirus 1 (EHV1) and equine herpesvirus 4 (EHV4) are important ubiquitous equine viral pathogens, causing much damage to the horse industry. EHV1 strains are associated with respiratory disease, abortion, and paresis/paralysis, whereas EHV4 strains are predominantly associated with respiratory disease. In the past decades much research effort has gone into improving knowledge about these viruses. In this presentation the most important aspects of these virus infections with respect to pathogenesis, epidemiology, diagnosis and (immuno)prophylaxis are discussed.

Pathogenesis

Respiratory disease

After inhalation EHV1 and EHV4 multiply in the epithelia of the nasal cavities, pharynx, trachea and bronchi(oli), and subsequently spread to regional lymph nodes. Young horses can develop distinct herpetic lesions in the mucosal membranes of all parts of the upper respiratory tract. In respiratory epithelia and lymphoid germinal centres, necrosis and intranuclear inclusion bodies can be observed.
Abortion and neonatal disease
Abortion can be initiated either by exogenous or endogenous infection, i.e. recrudescence of latent virus. After respiratory infection, EHV1 strains invade quickly the lamina propria and infect leucocytes and endothelial cells of blood and lymphatic vessels. Then infection spreads to regional lymph nodes from which infected mononuclear cells enter the circulation resulting in a cell-associated viraemia leading to placental transfer and infection of the foetus with subsequent abortion. However, foetal infection is not always a prerequisite for abortion. Experimental studies suggest that different EHV1 isolates vary in abortigenic potential. Since both viraemia and endothelial infection seem to play a crucial role in the pathogenesis of abortion and neurological disease, the current knowledge about the mechanisms of infection of blood mononuclear cells and transmission between endothelial cells and blood mononuclear cells at the port of entry and in the target organs will be presented.

Neurological disease
In contrast to several other alphaherpesviruses, e.g. herpes simplexvirus (HSV), bovine herpesvirus 1 (BHV1), and pseudorabies virus (PRV) that can cause encephalitis through primary neurotropism with virus multiplication in neurons and neuronophagia, EHV1 seems to be non-neuropotropic in equine brain, even after intracerebral inoculation. The propensity of certain EHV1 isolates to induce myeloencephalopathy does not reflect specific neurotropism but rather a marked endotheliotropism. EHV1 antigen, however, has also been demonstrated in neurons and astrocytes of horses with acute paralysis, and after experimental infection of SPF ponies chorioretinopathy and neural lesions have been observed, suggesting that at least some EHV1 strains may exhibit neurotropism. Recently a point mutation in the polymerase gene of EHV1 has been shown to be strongly associated with neurological sequelae of infection. Also several host factors seem to be associated with the occurrence and severity of neurological signs.

Epidemiology
Latency
As all alphaherpesviruses, EHV1 and EHV4 appear to establish life-long latent infections. Both viruses could be reactivated experimentally with very high doses of corticosteroids and mild nasal trauma. The most important site of latency is still controversial. Latency has been demonstrated for both viruses predominantly in lymphoid tissues and peripheral leucocytes on one hand, and predominantly in trigeminal ganglia on the other hand. Reactivation and shedding of EHV1 and EHV4 creates the opportunity for transmission to other horses, which is considered important in the epidemiology of EHV1 and EHV4 and might explain why these diseases can occur in closed populations. With the availability of sensitive PCR methods, in recent years more information has become available with respect to the occurrence of latent infections.

EHV1 and EHV4 infections early in life
During the 6- to 8-month period following weaning the majority of foals experience repeated respiratory infections with EHV4. The majority of such infections pass unnoticed. Circulation of EHV1 in unweaned and weaned foals supports the long standing management practices of separating pregnant mares from other groups of horses, especially lactating mares with unweaned foals, and groups of weaned foals, to reduce the incidence of EHV1 abortion. The occurrence of infections very early in life should also have implications for vaccine efficacy criteria and vaccination regimens, as the efficacy of vaccination in already latently infected horses is unknown.

Diagnosis
Respiratory disease
For nasal swabs, PCR appears to be more sensitive than virus isolation. Also local antibodies do not interfere with nucleic acid amplification methods, whereas they may interfere with virus isolation. Therefore, virus shedding can be demonstrated with PCR for a longer time, which is especially advantageous when samples are not taken in the acute phase of respiratory disease or after a relatively long incubation period as described for EHV1 neurological disease. A significant increase in EHV1/4 cross-reactive antibodies or type-specific antibodies by different serological assays can also be considered proof of infection.

Abortion
The foetus is the specimen of choice for diagnosis. EHV1 or EHV4 infection can be demonstrated in relevant organs either directly by IFT or immunohistochemistry in sections from frozen or paraffin embedded tissues, or by virus isolation or PCR.

Neurological disease
In the acute phase an EHV1 infection can be diagnosed by virus isolation or PCR from nasopharyngeal swabs, from white blood cells or from cerebrospinal fluid (CSF). CSF analysis often reveals xanthochromia and increased protein concentrations, reflecting vasculitis and protein leakage into the CSF. Presumptive evi-
dence of infection is provided by showing a seroconversion or significant increase in titre in acute and convalescent sera. However, antibody titres often rise already during the incubation period and may have peaked by the time neurological signs appear. Since a point mutation in the polymerase gene of EHV1 is strongly associated with the neurological potential of EHV1 strains, allele-specific PCRs are nowadays available to differentiate between neurological and non-neurological strains.

(Immun)prophylaxis

Preventive management
Epizootics of EHV1 respiratory disease are often associated with bringing together groups of susceptible horses under circumstances that produce stress, like weaning, long distance transport, intermingling of young horses originating from different locations for sales, training and performing. Preventive management should therefore involve avoidance of stress, prevention of introduction from an exogenous source by isolation practices by keeping incoming horses isolated from the resident population for at least three weeks, and division of the farm or track population into discrete, small units. Management has to be mainly concentrated on prevention of the more serious sequelae of an EHV1 infection like storm outbreaks and outbreaks of neurological disease, and should therefore aim at reducing the chance for exogenous introduction of EHV1 into a population of brood mares, reducing the chance for reactivation from latently infected carriers, and limiting the spread in the case of introduction of infection. A number of recommendations will be given.

Vaccination
Efficacy of vaccination against the different sequelae of EHV1 and/or EHV4 infections will be discussed on an individual level and on a population level. On a population level virological protection, i.e. reduction of virus excretion, is as important as clinical protection. Since latency is important in the epidemiology of EHV1 and EHV4 infections, as for other alphaherpesviruses inducing latent and recurrent infections, the goal of vaccination is different and more ambitious than for many other viruses. The aims for alphaherpesvirus vaccines are not limited to prevention of the first episode of disease but also the control of recurrent infections. Also reduction of re-excretion after stressful events in horses that are first infected and then vaccinated might be an important efficacy criterion for EHV1/4 vaccines. Efficacy of vaccines for protection against clinical disease and reduction of virus excretion after exogenous infection can be different from that after reactivation. Since the ideal EHV1/EHV4 vaccine has not yet been developed, current developments and research priorities will be discussed.

References
A list of references is available on request.

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During 2008 and early 2009 the FEI undertook a full review of its approach to doping and medication issues. This involved the setting up of two commissions. The Lundquist Commission looked at the rules, regulations and penalties around this area, the traditional policies related to prohibited substances, the past approach taken by laboratories, the way in which the FEI communicated with competitors, looking at what had worked well in the past and what had not. The Stevens Commission looked at more structural arrangements, such as stable security, the ability to report and build up intelligence about any lack of integrity, protocols, conflict of interest and systems. The resulting recommendations are now being delivered in the form of the FEI Clean Sport initiative which was intended to protect the sport both now and as the sport is expanding in the future, especially from deliberate doping and preventing accidental medication cases. The Clean Sport Initiative also provides measures in other areas such as who can access stabling areas, a much greater emphasis on the training and education of veterinary and other officials, the use of treatment areas in FEI events, and ensuring that there is monitoring on what substances are given and when. Significantly the term person responsible has been amended to go beyond not just the rider, but also a variety of other support personnel. All these measures are explained in detail at www.feicleansport.org

At the heart of the new approach to the FEI Prohibited List of substances is the 2010 FEI Equine Prohibited Substances List. This is easily the greatest change in policy. It is based on the approach taken to human athletes by the World Anti-Doping Agency (WADA). For the first time substances prohibited during competitions are individually named rather than referred to in...
broad categories. There are two simple categories. Banned Substances which have no regular legitimate use in equine medicine, and Controlled Medications which have common legitimate uses in equine medicine. Detection in the former category has a starting suspension of two years, in the latter category penalties can run from a few months up to 2 years. Because substances are individually named it gives clear guidance to veterinarians and competitors as to what is exactly tested for, but it also allows them to be more confident of what products they can use safely.

Several tools will be used to communicate the clean sport message:
The Equine Anti-Doping and Controlled Medication Regulations (EADCM) - which explain the processes and penalties that will be followed once a substance has been detected. It also introduces a medicines code that outlines what is expected by a rider in this area. The FEI Veterinary Regulations for 2010 - which are very much part of Clean Sport and ensuring fair play. Many of the amendments are recommendations made by the Clean Sport Commissions, have been incorporated, providing Veterinary Regulations that are compatible with the implementation of new EADCM Regulations , the new “List” approach, keeping medicine records, new WADA based terminology, and an important requirement for all FEI Veterinarians and other Veterinarians present at FEI events to undertake a course every 5 years so they can be more confident in their roles. Also being applied in support at many levels is a new clearer FEI website, greater use of email communications and the eventual establishment of a website specifically for FEI veterinarians. The “List ” will also be searchable with a search engine for ease of use. All these measures are intended to change behaviours and bring about a new way of thinking in horse sport.

It is recognised that transmission that originates from outwardly healthy animals is probably of greater importance than from purulent discharges from sick horses because the source of infection is hidden and appears without warning. One obvious group of potentially infectious but outwardly healthy horses includes those that are incubating the disease and go on to develop signs themselves. It is assumed that normal nasal secretions are the source of infection in these animals. The other important category of outwardly healthy potentially infectious horses are those recovered cases that continue to harbour the organism after full clinical recovery.

Alarmingly, in a subcategory of outwardly healthy potentially infectious horses, carriage and at least periodic shedding of S. equi continues for prolonged periods after apparent full and uncomplicated recovery. These horses are referred to as long-term, asymptomatic S. equi carriers and there is strong anecdotal evidence that they can be a significant source of new outbreaks, even in well-managed groups of horses. It is important that if control measures are to be fully effective there must be recognition of the importance of this category of animal and appropriate detection and management of them should be adopted.

It is likely that short lived guttural pouch empyema is the most frequent outcome of uncomplicated drainage of RPLN abscessation. However, in a small but significant proportion of cases (e.g. >10% in three intensively investigated recent UK outbreaks) there is failure of this clearance mechanism resulting in chronic empyema of the pouch.

By the identification, segregation and treatment of these potentially infectious horses, prolonged outbreaks have been successfully controlled and further outbreaks undoubtedly prevented. A systematic programme of repeated nasopharyngeal swabbing (i.e. at least three swabs taken at weekly intervals) of horses following the cessation of clinical signs or during quarantine of incoming horses, using conventional culture

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**EPIDEMIOLOGY AND MANAGEMENT OF STRANGLES**
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Purulent discharges from active and recovering strangles cases are an extremely important and easily recognisable source of new S. equi infections in susceptible horses. Transmission of infection occurs when there is either direct or indirect transfer of these S. equi carrying
in conjunction with PCR, has successfully identified carrier horses.

Confirming the diagnosis of guttural pouch empyema is generally straightforward and is best achieved by direct visual assessment of the inside of both pouches by endoscopy. Culture and PCR of \textit{S. equi} in lavage samples collected through the endoscope should always accompany this visual examination as infection and inflammation may persist in the absence of overt, visible pathology.

A recently developed serological \textit{S. equi} specific ELISA has assisted in the screening of groups of animals in which carriers are believed to be present and can be used to inform risk management such as with horses undergoing quarantine or pre-movement screening.

**HOW TO RECOGNISE AND MANAGE AN INFLUENZA OUTBREAK**

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**Clinical suspicion of flu**

Coughing and fever are the most common clinical signs of equine influenza, the cough being dry, harsh and initially non-productive. Coughing is frequent during the first week of infection, and in uncomplicated cases given sufficient rest, will disappear within one to three weeks. The nasal discharge is initially serous but subsequently becomes mucopurulent when secondary bacterial infection of the respiratory tract has occurred.

Increasingly it is recognized that among vaccinated horses that have some but incomplete immunity, outbreaks of clinically mild influenza do occur. In such outbreaks there is frequently mild signs that may not be recognized or diagnosed as influenza and often the first sign noted is poor training and racing performance. Outbreaks have been described in which the infection circulated subclinically for 18 days before recognizable clinical signs were observed.

**Confirming a diagnosis of flu**

The characteristic clinical features of equine influenza in susceptible animals (rapidly spreading disease manifested by a harsh, dry cough, high temperature and nasal discharge) are sufficiently characteristic to permit a tentative diagnosis. However, in animals that have previously experienced the infection or that have waning vaccinal immunity, it is difficult to differentiate influenza from other respiratory infections. In such situations laboratory diagnosis is required involving virus isolation, antigen detection or serology.

Specimens for virus isolation should be collected as soon as possible after the onset of pyrexia and coughing, as the period of virus excretion may be as short as one to two days in primed animals. Virus may be cultured from nasopharyngeal secretions collected into virus transport medium, usually by swabbing of the nasopharynx or by lavage of the trachea through an endoscope.

Influenza virus can be cultured in embryonated hens’ eggs or in susceptible mammalian cells such as Madin-Darby canine kidney (MDCK) cells. Embryonated hens’ eggs are inoculated via the amniotic (six to eight day-old embryos) or allantoic (8 o 12 day-old embryos) routes.

The adoption of more widespread vaccination has made the diagnosis of influenza infection less straightforward, with clinical signs being less severe, blood samples from acute cases already possessing moderate levels of serum antibody, and the quantities of live virus retrievable from the respiratory tract being greatly reduced. The development of a sensitive and rapid influenza antigen ELISA and quantitative PCR methods applied to extracts from nasopharyngeal swabs has greatly improved the ability to diagnose influenza in previously vaccinated horses.

Antibody to influenza virus may be detected by its ability to inhibit the agglutination of chick erythrocytes mediated by the viral haemagglutinin in the haemagglutination inhibition (HI) test. In HI tests a four-fold or greater increase in antibody titre between acute and convalescent-phase sera is regarded as significant and indicative of infection. Serological diagnosis of infection in a vaccinated population is complicated by the presence of vaccine-induced antibody. Most vaccines are trivalent and contain one representative strain of the H7N7 virus and two strains of the H3N8 virus. Vaccination usually stimulates significant increases in antibody to both subtypes, whereas antibody to infection is subtype-specific.
Joint infection is a very common and serious condition in the horse. Preliminary diagnosis of synovial infection is mostly based on the history and clinical symptoms. However, symptoms are often non-specific and a mild synovial infection might resemble an acute non-infectious synovitis. Several techniques can be used to aid in confirming the diagnosis of synovial infection:

1. Medical imaging
   The merit of radiography for the detection of synovial infection in horses is debatable. In adult horses, it may take weeks to months before the first signs of secondary degenerative joint disease become visible. Conversely, damage to the epiphyseal or metaphyseal bone of foals can often be visualised within 1 week after the onset of symptoms.

2. Synoviocentesis and synovial fluid analysis
   Synoviocentesis and collection of synovial fluid for analysis and bacteriological examination certainly has the highest efficacy for confirming synovial infection. The synovial fluid should first be evaluated macroscopically for quantity, colour, turbidity and viscosity. Within a few hours after experimental inoculation of a joint with bacteria, the synovial fluid becomes turbid and less viscous. Routine laboratory analysis of synovial fluid includes a total and differential white blood cell (WBC) count and a total protein (TP) measurement. Normal synovial fluid contains 0.77 ± 0.07 x 10⁹ WBCs/L, predominantly mononuclear cells, and 7.87 ± 0.03 g/L proteins. In a clinical situation, infection should be presumed from the moment the number of synovial fluid white blood cells exceed 30 x 10⁹/L with ≥ 80% neutrophils and the amount of TP increases to ≥ 40 g/L. In chronic cases, WBC counts may be less (5 x 10⁹ to 10 x 10⁹ cells/L) but large amounts of proteins are usually present (> 50 g/L) in the synovial fluid. When infection occurs after the injection of corticosteroids, the early synovial fluid changes may be of little diagnostic value and synoviocentesis should be repeated at 12 hours intervals.

3. Bacteriologic examination
   A positive bacterial culture definitively confirms diagnosis of synovial infection and provides the opportunity for antibiotic susceptibility testing. However, both false positive and negative culture results exist. False positive cultures mostly result from contamination during sampling or inoculation of the sample in culture medium. Because of their high prevalence, false negative culture results are a more important reason for concern. Several reports suggest that enrichment in blood culture medium may significantly reduce the number of false negative cultures. Using enrichment, a low concentration of bacteria in the synovial fluid is of lesser concern since a large inoculum size can be used. Furthermore, natural growth-inhibiting substances and eventually antibiotics present in the synovial fluid are diluted in the large volume of medium. An advantage of blood culture media over non-commercial enrichment broths is that most of them contain specific resins that bind growth-inhibiting substances. Most blood culture media also contain cell-lysing agents such as saponine since fagocytosis of bacteria was considered an important reason for false negative culture results.

Skeletal health is of paramount importance for the future performer. Hence, radiological screening for common orthopaedic disease has become an essential part of the management of young sport horses. Ideally, such a screening is performed at the age of about 18 months. At that moment, the skeleton is sufficiently mature to allow for a reliable screening for the presence of developmental orthopaedic joint disease (e.g. osteochondrosis) as well as juvenile navicular bone abnormalities. Would clinically important osteochondral fragments be detected, timely removal by arthroscopy is possible (secondary joint damage unlikely + excellent healing capacity at 18 months of age).

A standard radiological screening is restricted to the regions commonly involved in lameness:
1. Front feet
   - LM, D55°Pr-PaDiO and D65°Pr-PaDiO
   - Distal border fragments: associated with increased risk for lameness
   - Processus extensorius fragments: risk ~ size of the fragments
   - Subchondral cyst P3: almost always communication with DIP joint

2. Fetlocks
   - LM (Belgium: oblique views not standard)
   - Several types of osteochondral fragments can be detected
   - Roughly: dorsal fragments are important, palmar/plantar fragments not

3. Hocks
   - Minimum: Pl45°L-DMO, D30°L-PlMO
   - Optimum = 4 views = minimum + LM + PID
   - Anatomic variations: flattening medial talar ridge, droplet, enthesiophyte MTIII
   - Screening for osteochondrosis and juvenile bone spavin
   - Clinical relevance of ‘blurring’ of the foramen tarsale not clear

4. Stifles
   - Cd60°L-CrMO
   - Osteochondrosis lateral trochlear ridge of the femur: risk ~ size lesion
   - Medial femoral condyle: screen for subchondral cyst

The report should be complete, summarizing all radiological findings. Judgement on their clinical relevance should be balanced and in line with current scientific evidence. The clinical relevance of a radiological finding may be different for sport horses compared to breeding stallions.

MANAGEMENT OF THE “HIGH RISK” MARE AND FOAL
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The ‘high risk’ pregnant mare can be defined as a mare with clinical signs of an abnormal pregnancy or a systemic disease that is likely to have a significant impact on the foetus. Indications for assessment of foetal wellbeing include premature mammary development and lactation, vulval discharge, excessive abdominal enlargement, a previous maternal history of dystocia and/or premature placental separation, a previous history of a foal suffering from prematurity or sepsis, severe maternal illness (e.g. endotoxaemia) or disease causing marked disability or debilitation (e.g. laminitis/neoplasia/grass sickness), abdominal pain and prolonged gestation. Following assessment of the foetus, a management plan can be formulated, in conjunction with any maternal therapy, for the best care of the pregnancy until full term. Because the foal undergoes critical maturation during the last days of pregnancy, foetal survival at less than 300 days of gestation is rare. Even after this problems of dysmaturity are common. However, in some circumstances, foeto-placental stress (e.g. placentitis), may cause sufficient maturity of the foetal adrenal cortex, so that the foetus is viable even at early gestational ages. For this reason, management regimes are generally designed to enhance foetal viability in utero.

Foetoplacental assessment
Clinical examination should include examination for signs of udder development and lactation as well as a visual inspection of the cervix and culture of any cervical discharge.

Milk electrolyte assessment
Mammary development usually occurs in the last 4-6 weeks of pregnancy, although this does vary significantly between mares, especially maidens. Electrolyte changes in mammary secretions can be used to predict maturity and readiness for birth. Calcium levels rise near term with levels between 20-40 mEq/l in a mature foetus. Sodium concentration will fall and potassium concentration will rise so that levels cross approximately 48 hours from term. These changes are not reliable particularly if there is foeto-placental abnormality.

Ultrasound assessment of the foetus
This is the most useful technique for assessment of foetal wellbeing. Transabdominal ultrasonography (using a 3.5 MHz sector transducer) will allow visualisation of the foetus, whilst transrectal examination (using a 7.5 MHz linear transducer) allows for more detailed examination of the placenta at the cervical pole. Because sedative drugs will affect foetal activity and heart rate, these drugs should be avoided. The mare’s ventral abdomen should be systematically examined from sternum to the mammary glands. The foetus should be lying in anterior presentation, normally in dorsal recumbency. The foetal thorax is usually the most readily identified structure and the foetal heart can be identified beating in the cranial thorax with the major
blood vessels passing caudally, the aorta lying next to the spinal vertebrae. The diaphragm can be recognised separating the abdominal and thoracic cavities and foetal breathing movements may be noted. In the foetal abdomen, the fluid filled stomach, kidneys and liver are all easily identified. The umbilical cord can be seen floating within the foetal fluids. The amnion forms a brightly, echogenic membrane that divides the amniotic cavity from the allantoic cavity. The placenta can be recognised bordering the allantoic cavity. Different measurements of the foetus can be made including the foetal aortic diameter, the combined thickness of the uterus and placenta, foetal activity and foetal heart rate. Transrectal ultrasonography is used to assess the placenta at the cervical pole. The combined thickness of the uterus and placenta (CTUP) can be measured and early placental separation identified.

Management
Therapies for mares with complications in late pregnancy are directed primarily at appropriate treatment of the maternal disease. Placentitis can be treated with systemic antibiotics, depending on the sensitivity of the organism cultured. Trimethoprim/sulphonamide has been shown to cross the placenta and is the most widely used antibiotic. Some clinicians advocate a regime of 5-7 days of trimethoprim/sulphonamide per month when placentitis is suspected during mid pregnancy and continuously when term is near. Exogenous progesterone therapy (e.g. altrenogest) is commonly used to improve cervical tone and increase uterine quiescence. Other therapeutic agents used include pentoxyfylline, vitamin E and aspirin. Nutrition to support the mare should be considered to ensure that the foetus receives adequate nutrition for growth and development.

Regular assessment of the complicated pregnancy as term approaches is helpful to ensure that treatment is effective but also to allow preparation for delivery of a potentially compromised foal.

Management of the Sick Neonate in the Field
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Despite significant advances in the intensive medical care of the neonate there is still need for on-farm assessment and medical care, either because intensive care facilities are not available, are not financially viable or because of risk of spread of disease. Common indications for medical intervention include resuscitation, perinatal asphyxia, sepsis, diarrhoea, colic and neonatal isoerythrolysis.

History and clinical assessment
As with most situations in clinical medicine an accurate history of the sick neonate is vital. This should include gestational length, problems during parturition, placental abnormalities and the mare’s previous foaling/breeding history. Clinicians should be familiar with the normal neonatal foal so that abnormalities can easily be detected. A full clinical examination should be carried out, in particular for evidence of birth trauma.

Fluid therapy
Neonates are particularly prone to hypovolaemia and early identification and treatment markedly improves outcome. In the first instance a collapsed neonate may need a rapid infusion of fluid to support the circulation. For a 50 kg foal 1l of a balanced electrolyte fluid (Hartmanns) can be given as a bolus over less than 20 minutes and then the foal should be reassessed. Up to 3 further boluses can be given, reassessing after each. After correcting hypovolaemia the maintenance rate of fluid can be calculated (4-5 ml/kg/hr). This may have to be adjusted, for example if the foal is losing additional fluid with diarrhoea. It may also be necessary to correct any specific electrolyte or acid base deficits. Plasma is useful to provide colloid support to the circulation as well as providing a source of antibodies. Serial measurements of urine specific gravity are a good indicator of fluid status in foals with normal renal function. Specific gravity rises prior to other signs of insufficient fluid therapy. Normal: 1.001 – 1.008.

Nutritional Support
Neonatal foals have few glycogen reserves and so failure to provide adequate nutrition can quickly lead to
hypoglycaemia (blood glucose < 4mmol/l) as well as dehydration, hypothermia and secondary complications. Therefore, nutritional support should form an essential part of care for the sick foal.

The easiest way to support blood glucose in the short term is to add 50% dextrose solution to the resuscitation fluids (10-20ml to each litre of fluid). It is very important to measure blood glucose and maintain a level between 4-10 mg/ml. However, glucose is not suitable for long-term nutritional support and if enteral feeding is not possible then parenteral nutrition should start after a maximum of 12 hours. Correct nutrition is essential to promote normal development of the foal and poor nutrition will delay recovery.

It is preferable to meet nutritional requirements through the enteral route and this should be encouraged as long as the gut is functioning normally. Foals suck approximately 4-7 times per hour and during the first 24 hours of life will consume approximately 15% body weight (or approximately 8 litres for a 50kg TB foal). From days 2-7, milk consumption will increase to approximately 23% body weight daily. In foals with a weak suck reflex the use of an indwelling naso-gastric tube, that can be taped or sutured to the foal’s muzzle, allows feeding of small volumes of milk at frequent intervals.

Respiratory Support
Oxygen therapy may be required and this can provided with an intranasal oxygen tube, measured to the level of the medial canthus. These are well tolerated and can be sutured or taped to the muzzle. The oxygen should be humidified through sterile water.

Basic Monitoring
There is a basic level of laboratory data that is required when managing a sick foal in the field. This will be tailored to the individual case but should include haematology, biochemistry (including renal enzymes), electrolytes, glucose, lactate and urine specific gravity. Serial measurements should be made and recorded so that trends are easily appreciated.

Equipment
Equipment required to aid effective treatment of sick neonates in the field includes indwelling feeding tubes, IV catheters, extension sets, blood and fluid giving sets, drip counters for fluid lines, a foal muzzle, milk replacer, urinary catheters, a glucometer and a refractometer to measure USG.

General Nursing

Umbilicus
Frequent evaluation and care of the umbilicus is important, as complications such as umbilical infection or patent urachus are common in the sick neonate. Placement of a urinary catheter with a collection bag prevents leaking urine contaminating the ventral abdomen and allows for urine output to be calculated. A 0.5% chlorhexidine solution is a good topical treatment.

Cleanliness
Cleanliness of the box and equipment is essential as sick foals are susceptible to infection, especially via the oral route if receiving anti-ulcer medication, which increases gastric pH. Care should also be taken over the IV and urinary catheters. Foals are also prone to decubitis ulcers and so care should be taken to make sure that the bed is changed frequently.

Physical Therapy
An important part of recovery involves spending time stimulating and mobilising the foals. It is therefore important to encourage them to stand and move as soon as possible.

TRANSPORTATION OF THE MARE AND FOAL TO THE HOSPITAL
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Indications for referral of a neonate to the hospital are numerous but the most common conditions that cause foals to fade soon after birth are perinatal asphyxia, sepsis and dysmaturity. Prompt stabilisation and referral will significantly increase survival rates.

In the most severe cases, it is important to get the foal to the hospital as quickly as possible, even if that means the foal travelling ahead of the mare. The mare should be sedated to minimise the trauma of her foal being removed and then stripped of colostrum, which can then be sent with the foal.
**Abstracts European Veterinary Conference**

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**Scientific proceedings: Equine Programme**

**CHAPTER 6**

**Others considerations include:**

**Antibiotic Therapy**
Prior to giving any antibiotics a blood sample should be collected in a sterile manner for culture. This should be sent with the foal to the referral centre. If sepsis is suspected a broad spectrum antimicrobial can then be administered.

**Fluid Therapy**
Neonates are particularly prone to hypovolaemia and early identification and treatment markedly improves outcome. In the first instance a collapsed neonate may need a rapid infusion of fluid to support the circulation. For a 50 kg foal, 1l of a balanced electrolyte fluid (Hartmanns) can be given as a bolus over less than 20 minutes and then the foal should be reassessed. Up to 3 further boluses can be given, reassessing after each. The foal’s glucose can be measured and if hypoglycaemic, 50% dextrose (10-20ml / litre) can be added to the resuscitation fluid. Plasma is useful to provide colloidal support to the circulation and antibodies, but because it has to be thawed slowly, is not practical to be given in these situations. Placement of a sterile over-the wire polyurethane catheter is preferable.

**Respiratory Support**
If in respiratory distress, an intranasal oxygen tube can be placed to the level of the medial canthus and sutured or taped to the muzzle. A portable oxygen cylinder can be used to provide oxygen whilst the foal is being transported to the hospital. In the short-term it is not essential that the oxygen be humidified. The foal should also be maintained in sternal recumbency during transport, as this will help respiratory function.

**Hypothermia**
This is a common problem in the very sick neonate and the foal should be wrapped in blankets and warmed as much as possible on the way to hospital. Heat packs and hot water bottles are useful for this purpose. Also limb bandages will help to keep the foal warm and prevent injury if it is thrashing.

**Other medications**
Other treatments that may need to be considered include diazepam (5mg IV) for the control of seizures and pain relief if the foal is showing signs of colic.

**UTC, A NOVEL APPROACH FOR INJURY-PREVENTION AND MONITORING OF TENDON LESIONS**
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Lesions of flexor tendons are amongst the most serious orthopaedic disorders in the horse, frequently threatening the animal’s athletic career as these tendons play a vital role in locomotor efficiency. During the first phase of the stance stage, they store elastic energy that is subsequently released as kinetic energy, in fact acting as energy saving springs. This biomechanical function is based on a unique architecture with a collagenous matrix hierarchically organised into tendon bundles. This matrix can deteriorate because of gradual degeneration and/or single (partial) ruptures, eventually leading to an impaired function.

Ultrasonography (US) was introduced as a new imaging modality for tendons and ligaments in the early 1980s. US images have the potential, like no other imaging technique, to provide an inward view because they contain reflections of ultrasound waves against structural entities. However, reproducibility is poor due to instrumental variables and transducer handling.1 Furthermore, conventional US is not reliable for the assessment of stages of integrity because, as a consequence of limits of resolution, every US image is a mixture of structural reflections and interfering echoes: only larger structures (≥ 0.38 mm for 10 MHz and ≥ 0.45 mm for 7.5 MHz transducers), like tendon bundles, generate reflections, while smaller entities, such as fibrils and cells, will result in interference, each with their specific dynamism in real-time US.2 This means that the dynamism of echo-patterns is strongly related to the degree of structural integrity of tissue, but this phenomenon is not captured in still US images.3 Therefore, a method for “computerised ultrasonographic tissue characterization” (UTC) was developed for quantitative evaluation of the structural integrity. Under standardised conditions, with a high-resolution transducer mounted in a motorized tracking-device, transversal images are collected at regular distances of 0.2 mm.4 These images are stored instantaneously in a laptop computer and subsequently the dynamism of
the echo-patterns is quantified and related to the structural integrity. In this way, 4 different echo-types can be discriminated, namely:

- type I, generated by reflections at intact and aligned tendon bundles,
- type II, generated by reflections at discontinuous or waving tendon bundles,
- type III, generated by interfering echoes from mainly fibrillar components,
- type IV, mainly generated by cellular components and fluid.

This ultra-structural information is visualised tomographically in 3 planes of view and in 3-D. The stage of integrity can be quantified by means of the respective ratios of echo-types.\(^4,5\)

UTC has been tested extensively for clinical applications and some relevant observations in these studies are:

A. the ICC (intra-class correlation coefficient) of intra-observer reliability is 0.98 and the inter-observer reliability, both for equine SDF and human Achilles tendon has an ICC of over 0.91.\(^5,6\)

B. normal flexor tendons of young-mature horses (2-5 years of age) are characterized by 85-90% echo-type I, 10-15% type II and scarcely type III plus IV echoes.

C. initial deviations from the normal situation over time (“ageing”?,”degeneration”?), mostly asymptomatic, are characterised by a limited increase of cross-sectional area (<15%) and increasing percentages of echo-types III plus IV. These changes may be reversible within 4-12 weeks, most probably as “training-effects”, if not they can be seen as early signs of “degeneration”.

D. during longitudinal monitoring of tendon repair several stages can be discriminated: (a) till 3 weeks post-injury, “extension and demarcation of the lesion” with a rapid decrease of structure-related echo-types I plus II and a sharp increase of type IV, (b) from week 3 till 8, “fibrillogenesis” with an increase of echo-type III and a decrease of IV, (c) from weeks 9 till 12, “early bundle formation” with a sharp increase of echo-types I and II, and (d) starting from week 13, “organization and remodelling” with continued increase of type I, a gradual decrease of II and with types II and IV tending to normal. These in vivo observations were verified with post-mortem histology and biochemistry.\(^4\)

E. the timetable for non-intervened repair described in D. was subsequently used for the quantitative evaluation of interventions such as intra-tendinous injections with platelet-rich plasma (PRP) and short-term immobilisation of acute tendon lesions. Monitoring by UTC quantified at all stages significant differences between treatment and placebo groups. At end-stage, these UTC observations corresponded precisely with the ultimate quality of repair as shown by post-mortem histology.\(^6\)

It is concluded that UTC is a novel technique that can visualise and quantify the structural integrity of tendons with an excellent reproducibility. As such, UTC is an excellent tool for injury-prevention, monitoring of repair and evaluation of therapies.

References


WEST NILE VIRUS INFECTION: TREAT OR REALITY
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Introduction
International interest in equine vector-borne diseases has increased as result of recent infections such as West Nile Virus outbreaks in Italy and Austria. Competent vectors for West Nile Virus (WNV), such as Culex, Aedes en Ochlerotatus spp., are present in Western Europe. Furthermore, Western Europe has direct contacts with endemic areas through wild bird migrations. The very rapid spread of WNV in the USA makes it clear that the Netherlands have the potential to be similarly affected. The European Committee agrees on this point and has already (in 2004) asked that all member states investigate all unexplained encephalitis cases. The Netherlands takes an active part in this project. However, until the present time all samples have been negative (excepting horses that were imported from endemic areas).

In the Netherlands WNV is not a notifiable disease, and this is based on the fact that the horse is a ‘dead-end host’. However, there is some discussion as to whether it may be considered to be an equine viral encephalopathy. This will become clearer after the revision of the new European Animal Health Regulations.

As WNV infections are also a human problem, it is of great importance that any outbreaks of WNV among horses is detected early.

Epidemiology
People and animals become infected following the bite of certain kinds of mosquitoes that are infected with WNV. Following a bite from an infected mosquito a low-grade viraemia occurs first, then the virus replicates in the lymph nodes and subsequently spreads to the neural tissues either by passing the blood brain barrier or by direct transmission through axons. The virus can replicate in mosquitoes and vertical transmission in mosquitoes is possible. In people vertical transmission from mother to child is also possible. Horses (and people) are ‘dead end hosts’, this means that the replication of the virus in these hosts is so limited that a mosquito that sucks blood from these hosts does not become infected.

Clinical symptoms
Infection with WNV does not always lead to signs of illness in people or horses. Whether a horse develops clinical signs is mainly dependent on differences between virus strains but also on the infection dose and the immune status of the host. In horses systemic signs of WNV consist of low grade fever (38.6-39.4°C), anorexia and dullness. In some cases colic seems to be the first clinical sign. Neurological signs can be very variable. Some horses start with normal gait and others with muscle fasciculation or an abnormal mental status. The muscle fasciculation often starts around the eyes and the muzzle and may be limited to this area. However, in some cases the whole body and all four limbs can be affected. Abnormal mentation means that a normally quiet horse can become very excited and a nervous horse can be very quiet. Incidentally the signs may resemble narcolepsia. Neurological signs often appear abruptly and progress quickly. The spinal cord pathology may induce ataxia and paresis; both fore and hind limbs can be affected and signs may be both unilateral and bilateral.

Diagnosis
The diagnosis WNV infection will probably not be established if the horse only shows limited fever. However, if the case also shows neurological signs, WNV must be considered and a serum sample then should be sent to a laboratory that is able to perform the IgM ELISA or a blocking ELISA (IgG and IgM) for WNV. In the Netherlands the screening tests are performed by the Animal Health Service (Deventer) and the confirmation is made by the National Institute for Public Health and the Environment (RIVM) by means of the plaque reduction neutralisation test (PRNT).

Post mortem examination
In horses that have died of WNV, a polioencephalomyelitis will be found. The macroscopic changes can be very subtle and limited to small haemorrhagic foci in the brain and spinal cord. Whilst performing a post mortem examination on a suspected WNV case, it is important to prevent cross contamination. When a horse is referred for post mortem examination it is important also to warn the pathologists in advance.

Treatment and prevention
Treatment of a WNV infection is only symptomatic as there is no specific therapy. Recently an ‘inactivated vaccine’, Duvaxyn WNV® (Fort Dodge), was licensed in Europe. In the Netherlands a small successful trial was performed at Utrecht University in cooperation with the Animal Health Service (Deventer).
In the USA specific campaigns are undertaken to inform the general public of the risks of mosquito bites and WNV. With relatively simple measures the number of mosquito breeding sites can be reduced by eliminating stagnant water sources (discarded tyres, bird baths, buckets, ceramic pots etc). Mosquitoes may breed in any puddle that lasts for more than four days. Humans are advised to wear long sleeves and use proper insect repellents. The same measures would be effective for horses.

**Conclusion**
At the moment the expectation is that WNV will occur in The Netherlands in the near future. It is important to consider WNV as a differential diagnosis in horses with neurological problems, especially in the summer months when mosquitoes are active.

References available on request.

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**Parasitic skin problems in horses**

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**Introduction**
Parasitic skin infections are common in horses and commonly involve infestations with mites, lice, ticks, warbles and other parasites. In this review only the most common problems in our area will be discussed.

**Mange**
Infections with Chorioptes equi are common, while infections with Sarcoptes scabiei var. equi, Psoroptes equi or Demodex equi are very rare. The clinical symptoms of demodicosis are also not very clear and the author has never diagnosed a clinical demodicosis case in a horse.

Mange infections are contagious and may spread through direct contact or through indirect contact (tack, grooming material etc.).

Equine chorioptic mange is caused by Chorioptes equi, a non-burrowing mite. The mites live on the skin surface and feed on skin debris. Chorioptes equi is recognised by its long pretarsi with short pedicles.

Clinical disease is most often identified in winter in horses with feathered legs. Transmission is by direct and indirect contact. The disease may be transmitted from season to season by carrier horses, which show no sign of disease. There is no interspecies transmission of chorioptic mange, and the mites do not affect humans. Clinical signs are usually limited to the distal limbs and consist of pruritus, scaling and a fine papular eruption. In heavy infestations the whole body can be affected leading to generalised signs of pruritus, irritability and even weight loss. Horses may stamp their feet because of the irritation. In more heavy infestations the horse seeks every opportunity to rub posts and to bite at the limbs even to the point of causing erosions of the skin.

The diagnosis of all types of “mange” and the causative parasite are usually made by microscopic examination of skin scrapings. Skin scrapings or groomings should be taken from the edge of the skin lesions.

Before treatment it is advisable to clip long hair carefully. However, the owner will often not allow this as it may take at least 2 years before feathered fetlocks have grown long enough to show the horse again. In any case, the limbs should be thoroughly washed and
all scabs removed. Then, topical shampoos with ectoparasiticidal chemicals are the treatment of choice. Over the last few years the number of effective skin treatments to treat mite and lice has fallen significantly. However, foxim and diazinon (choline esterase inhibitors) and fipronil are still available, although often not licensed for the horse.

As the eggs hatch after 10-14 days, treatment must be repeated at around 14 day intervals. The whole horse should be washed! It is important to spray or wash also all tack and equipment used by the horse, as well as clean and disinfect the box or other accommodation. Sometimes this is very difficult to accomplish, but since the mites cannot survive away from the host, leaving the box stalls empty for about a week should also suffice. However, under certain circumstances chorioptes mites have been shown to survive up to 69 days. It is very important to note that amitraz (Tactic®) is toxic to horses and will induce symptoms like dullness, ataxia, decreased muscle tone and colonic impaction.

**Pediculosis (infections with lice)**

Lice occur worldwide. In the horse infections involve Haematopinus asini and Werneckiella equi (former Damalinia equi).

Haematopinus asini is a sucking louse (Anoplura) with an ash-grey-blue appearance and a sharp conical head smaller than the thorax. They prefer the mane, tail and fetlocks and are rare in the Netherlands. Werneckiella equi is a biting louse (Mallophagen) with a pale yellow appearance, eating crusts and epidermal cells, and the squareish head is wider than the thorax. These lice prefer the dorsolateral thorax and abdomen and are more common in the Netherlands. The life cycle of both species is 20 to 40 days.

Lice are very host-specific and occur predominantly in young or diseased animals. The infections are particularly severe in winter months and early spring when horses are congregated together; this may be related to higher humidity and the longer hair coat. Spread is by direct contact or indirectly through grooming brushes, rugs and even via the stable environment where they can survive for some weeks off the host animal.

An infestation with lice causes pruritus and the animals start to rub, bite, scratch etc. This produces dermatitis and unthriftiness with secondary patchy alopecia leading to a moth-eaten appearance of the coat. Severe irritation and self-inflicted trauma can produce areas of dermal excoriation and serum exudation. Most obvious locations are the side of the neck, the dorsum and the base of main and tail. As result of a sucking louse infestation anaemia may occur.

The diagnosis is made with the naked eye (‘walking dust’), or after microscopic examination of a skin scrap. Even with the naked eye (or a loupe) the lice and nits can easily be seen. Microscopic examination is necessary for further determination.

Treatment involves bathing, spraying or dusting with ectoparasiticidal compounds. The whole animal must be treated and the treatment repeated within 10-14 days as the eggs (nits) are not killed by the treatment. All in-contact horses should be treated simultaneously. Oral ivermectin has a limited effect. Some clinicians advocate ‘pour-on’ formulations of ivermectin but some cases develop transient urticaria within 12 hours and occasionally some horses react vigorously at the local application sites. Organophosphate or chlorinated hydrocarbons are effective, but regrettably in most countries are not approved for use in horses. Pruritus usually ceases in 24-36 hours following successful treatment.

**Infections with ticks**

In the Netherlands and the UK Ixodes ricinus and Dermacentor reticulatus occur. Mostly it is an accidental finding involving a few individual ticks. Initial skin lesions are papules, pustules, wheals, and, occasionally, nodules centred around a tick. These primary lesions may develop crusts, and alopecia. Pain and pruritus are variable. Ticks may carry other diseases, such as Borrelia burgdorferi, Anaplasma phagocytophilum, Babesia caballi and Theileria equi. As ticks are a rare phenomenon in UK and Netherlands they can be removed by hand using a pair of tweezers with a twisting movement. In the past it was advocated to anaesthetise the ticks with alcohol but it is believed now that during anaesthesia the tick may empty the salivary glands and the gut leading to infection of the host. After removal of the tick the tiny wound should be disinfected with alcohol or povidone-iodine.

**Oxyuriasis**

Oxyuriasis is an infestation with Oxyuris equi, which inhabits the terminal regions of the small colon and rectum. The adult worms migrate out of the anus to lay eggs on the perianal skin and cause anal irritation. The infected horses may show tail rubbing and irritation with self-inflicted trauma to perianal skin. The diagnosis is confirmed by examination of perineal area and identification of characteristic triangular operculate eggs from perineal skin using adhesive tape technique.

Most modern anthelmintics are effective and any deworming program aimed at ascarids and strongyles will treat the adult worms. The condition can be ignored in horses receiving good worming regimens.
involving avermectins in particular. Improved stable hygiene with clean bedding and regular cleaning of water troughs are important. Local treatment consists of washing the perineum with soap and warm water to remove the eggs. The itch may be controlled with tranquillisers until anthelmintics have been given.

**Poultry red mite**
Dermanyssus gallinae is blood-feeding mite of poultry. Sometimes they accidentally infect horses. There is always direct or indirect poultry contact. The mite causes severe, often progressive pruritus over the whole body. Small pruritic skin papules and crusts may be present in areas readily accessible to the mite (legs, face and ventrum). Horses become irritable and stamp and bite at their legs and body. The diagnosis is easily confirmed: groomings will reveal mobile red mites, which are very visible on a black tile. Microscopic identification of mites shows a relatively large mite (1.5-mm) that is somewhat spider-like.

The treatment is to wash or spray the body with an approved (licensed) organophosphate or pyrethroid insecticide and to remove horses from proximity to poultry. All equipment and buildings should be treated with appropriate insecticides; the mites can live up to 4-5 months without feeding and so sporadic infestations occur.

**SOME IMMUNE MEDIATED EQUINE DERMATOSES**
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**Introduction**
Immune-mediated dermatoses can be subdivided into primary (autoimmune) and secondary (immune-mediated) disorders. The latter are believed to be primarily diseases wherein tissue destruction results from an immunologic event that is not directed against self-antigens. In auto-immune disease, antibodies or activated lymphocytes develop against normal body constituents and will induce lesions of the disease by passive transfer. Important autoimmune skin disorders are the pemphigus complex (pemphigus foliaceus, pemphigus vulgaris), systemic lupus erythematosus and alopecia areata.

Secondary immune-mediated dermatoses are erythema multiforme, equine exfoliative eosinophilic dermatitis and stomatitis, vasculitis and sarcoidosis. This review will focus on clinical signs and making a diagnosis of the most common problems.

**Poultry red mite**
Dermanyssus gallinae is blood-feeding mite of poultry. Sometimes they accidentally infect horses. There is always direct or indirect poultry contact. The mite causes severe, often progressive pruritus over the whole body. Small pruritic skin papules and crusts may be present in areas readily accessible to the mite (legs, face and ventrum). Horses become irritable and stamp and bite at their legs and body. The diagnosis is easily confirmed: groomings will reveal mobile red mites, which are very visible on a black tile. Microscopic identification of mites shows a relatively large mite (1.5-mm) that is somewhat spider-like.

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**Pemphigus foliaceus**
Pemphigus foliaceus is an auto-immune disease characterised by an exfoliative dermatitis due to a type II hypersensitivity arising as a result of auto-antibodies directed against the cell membrane of the epidermal cells (desmosomal antigens). The disease often starts with vesicles and pustules forming epidermal collarettes, which starts mostly around the face and/or the limbs. The coronary band, chestnut and ergot are often also inflamed or the disease may be restricted to the coronary bands. Transient, persistent or recurrent urticaria may be the first clinical sign and this can occur weeks before more typical pemphigus lesions are seen. Advanced cases may show severe, diffuse crusting and scaling with extensive alopecia.

**The pemphigus complex**
The ‘pemphigus complex’ includes several autoimmune diseases in which the horse develops antibodies against its own (epi)dermis. Pemphigus foliaceus is the commonest form of pemphigus and occurs in horses of all ages. Pemphigus vulgaris and bulleous pemphigoid are very rare and present with vesicles and ulcerated lesions at muco-cutaneous junctions in the mouth, nose, eyes and vulva. The tentative diagnosis of these diseases is made on the history and clinical appearance and is confirmed by histological examination of biopsy specimens.

**Pemphigus foliaceus**
Pemphigus foliaceus is an auto-immune disease characterised by an exfoliative dermatitis due to a type II hypersensitivity arising as a result of auto-antibodies directed against the cell membrane of the epidermal cells (desmosomal antigens). The disease often starts with vesicles and pustules forming epidermal collarettes, which starts mostly around the face and/or the limbs. The coronary band, chestnut and ergot are often also inflamed or the disease may be restricted to the coronary bands. Transient, persistent or recurrent urticaria may be the first clinical sign and this can occur weeks before more typical pemphigus lesions are seen. Advanced cases may show severe, diffuse crusting and scaling with extensive alopecia.

A pony with pemphigus foliaceus
The disease may be accompanied by mild pruritus, but there may be no pruritus at all. If the disease progresses systemic signs including lethargy or depression, anorexia, ventral oedema, limb oedema, fever and weight loss will occur. Internal organs are, however, not involved. There is no known sex predilection, geographical distribution or seasonality associated with the condition.

The tentative diagnosis is based on history and clinical examination. In some cases the outer epidermis separates easily from the basal layer on exertion of firm sliding pressure (Nikolsky’s sign). A skin biopsy will confirm the diagnosis. Intranuclear or subcorneal acantholysis with cleft and vesicle or pustule formation is typical. Conventional histopathology is far more reliable than immunopathologic testing.

Alopecia areata
Alopecia areata can be considered an autoimmune disease as the hair bulb undergoes a cell-mediated attack. T-lymphocytes, presumably specific for antigens of the hair matrix and root sheath epithelium, damage the growing hair. Often these patients are referred as a case with ‘a non-healing ringworm infection’ that has progressed to also losing its mane and tail hairs (diffuse thinning). The skin appears completely healthy, and the lesions are neither pruritic nor painful. The reasonably circumscribed areas of partial or complete alopecia may wax and wane.

Histopathology of a biopsy reveals lymphocytic bulbitis (‘swarm of bees’). In chronic cases this bulbitis may already have disappeared, and there may be no detectable abnormalities except the small telogen follicles lacking hair shafts.

Erythema multiforme
Erythema multiforme is considered to be an immunemediated or even an auto-immune disease because lymphocytes ‘attack’ the keratinocytes. Drugs, infectious agents or toxins may alter the antigenicity of keratinocytes. However, many cases are idiopathic. In the human two forms are distinguished. In ‘erythema multiforme minor’ only skin lesions are present, while in ‘erythema multiforme major’ the mucous membranes are also involved. In most cases of erythema multiforme in horses the mucous membranes are involved. The most prominent clinical signs are serpigenous raised lesions in the skin. These are also described as ‘doughnut-like rings’. The overlying skin and hair coat are usually normal. The difference between erythema multiforme and gyrate urticaria is often difficult to establish. However, the lesions of erythema multiforme do not pit with digital pressure, unlike the wheals of true urticaria (hives).

The diagnosis is based on history, clinical examination and the histological examination of a punch biopsy. In the biopsy, the major epidermal changes include single necrotic keratinocytes throughout all layers of the epidermis including the adnexal epithelium, lymphocytic exocytosis and satelitosis, vacuolar alteration of the basal cell layer and the basal membrane zone and marked parakeratotic scale and crust. The dermal changes include oedema of the superficial dermis, extravasation of erythrocytes in the superficial dermis and superficial perivascular lymphohistiocytic infiltrate.

Cutaneous vasculitis
Cutaneous vasculitis is a combined type I and type III hypersensitivity reactions, and is most often associated with the deposition of immune complexes and other inflammatory products in the cutaneous vascular endothelium wall. This causes damage to vascular endothelial cells, leakage of serum constituents into the extra vascular space, and consequent oedema. The cascade may progress to cause dermatitis, necrosis and cutaneous ulceration, particularly in the distal limb regions.

The identification of the underlying problem is a diagnostic challenge. This may be an infectious agent such as Streptococcus, Corynebacterium, Equine Viral Arteritis etc. Some of the bacterial pathogens may also cause a lymphangitis. Systemic signs including pyrexia,
weight loss, depression and anorexia may accompany the problem. No age, breed or sex predispositions are known.

Purpura haemorrhagica is commonly associated with the recovery phase of an upper respiratory infection. Affected horses show severe oedema of the limbs and the head with plasma exudation. Partial leucocytoclastic vasculitis is restricted to the non-pigmented areas of the lower limb. Skin biopsies of early lesions will reveal leucocytoclastic vasculitis with vessel wall necrosis and thrombosis. Although sunlight is described as an important factor, contact with grass (by eating or by standing in it) may be more important.

Conclusion

Immune-mediated diseases in the horse with cutaneous manifestations remain an interesting group of conditions. Most clinical signs and diagnostic features are defined in literature, but establishing a definitive diagnosis and choosing an effective therapy or a way to manage the clinical signs are still very challenging.

EQUINE INFECTIOUS ANAEMIA: NUISANCE OR DISASTER?

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Introduction

Many factors influence the epidemiology of exotic (vector borne) diseases among human and animal populations. The mechanisms of transmission are important factors for the successful spread of a new disease in an unprotected, immunologically naïve population. The veterinarian will inevitably play a central role in the early recognition, detection and confirmation of new diseases in the equine population. An important example of the unexpected spread of an infectious disease is provided by the equine infectious anaemia (EIA) epidemics in Ireland in 2006. EIA is caused by an equid-specific lentivirus related to HIV. Although EIA has a worldwide distribution it is not (yet) a problem in Western Europe and the few outbreaks that have occurred have been effectively controlled. In the Netherlands EIA is a notifiable disease, but the government will not combat the disease. This means that in case of an outbreak the equine sector itself has to take its own responsibility.

The infection in Ireland was proven to be the direct result of treatment of a foal with infected imported plasma. Further it was demonstrated that PCR and RT-PCR had potential to detect acutely infected horses earlier than some of the official tests (e.g. Coggins’ Test) and that some horses only became serologically positive after several months.

Epidemiology

Competent vectors for EIA are large blood-feeding insects, such as Tabanidae (horse flies) and incidentally Stomoxys calcitrans (stable flies). These insects are present in Western Europe but transmission is considered to be limited to physical transfer of contaminated blood since EIA virus does not replicate in insects. Man is by far the most efficient ‘vector’ of this virus. Since Romania joined the EU, Western Europe now has direct contacts with a proven endemic area. At present Romania is one of the largest exporters for slaughter horses in Europe. The incidental outbreaks of EIA in Germany and the UK (January 2010) make it clear that the Netherlands also have the potential to be similarly affected.

Clinical symptoms

Infection with EIA does not always lead to signs of illness. The incubation period is generally 1-3 weeks but may be several months. The disease may cause recurrent febrile episodes, anaemia, thrombocytopenia, yellowing of the mucous membranes, oedema, petechiation, emaciation and death, but the signs may be very mild, especially on first exposure. Chronic infections may lead to ill-thrift and predispose to secondary infections, but some horses may live many years without any significant clinical signs.

Diagnosis

Infection with EIA can be easily overlooked unless serologic testing is carried out. Asymptomatic horses will be carriers for life and will be infectious during any febrile periods during which viraemia reaches significant levels. If there is any suspicion of EIA the authorities should be informed and official tests performed (in the Netherlands by the Central Veterinary Institute). PCR had potential to detect acutely infected horses earlier than some of the official tests (e.g. Coggins’ Test) and that some horses only became serologically positive after several months.

Post mortem examination

Horses that have died during the acute phase may show splenomegaly, hepatomegaly, generalised lymphadenopathy, ventral oedema, thrombosis and petechiation. Post mortem examination of Asymptomatic carriers is usually unrewarding. EIA is not a zoonosis and poses no public health risk.
**Therapy and prevention**

There is no therapy for horses with EIA except supportive treatment. There is no vaccine. A degree of prevention may be afforded by insect control and good veterinary hygiene not only for needles but also for dental equipment etc.

Within the present legal framework a farm or riding school can be quarantined until the infected horses have been destroyed and all other horses have tested negative over several months. However, in the Netherlands there is no obligation for an owner of an infected animal to destroy it and it is important for the equine industry to develop a ‘code of conduct’ in cooperation with the government.

**Conclusions**

At the moment the expectation is that EIA may also be identified in the Netherlands in the near future. It is important to consider EIA as a differential diagnosis in horses exhibiting anaemia and thrombocytopenia with fever, especially if these have been imported. Whether an outbreak will be a nuisance or a disaster will be determined by the ability of the Dutch equine industry to prepare and enforce a suitable ‘code of conduct’ in timely fashion.

**Additional reading**


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**ETHICS IN HORSE SPORTS**

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Throughout history humans used horses for a variety of reasons as for combat, hunting, in agriculture and in recent decades more and more for recreation and sports. As a consequence human-horse relationship develops and varies between different horse practices. Equestrian sports like show-jumping, racing, endurance and eventing continues to grow worldwide and contributes considerably to economy. To improve the performances of sports horses housing and feeding practices have been adapted, training methods have been developed and substances will be used. Management practices are driven by human interests and costs limitations. As a consequence the traditional contract between humans and horses has been threatened and animal ethics has emerged.

People involved in horse sports are given freedom and autonomy to pursue their aims in the different disciplines. In return society expects that horses are treated in such a way that the health and welfare of those animals is guaranteed with respect for the intrinsic value of the horse. There is concern that an increasing number of sports horses are merely kept for their extrinsic value and are forced to perform beyond their capacities and the needs of the horses no longer guide management and housing conditions.

It is interesting to learn which moral attitudes of humans towards animals exist in society. In 2007 a national survey has been performed in Dutch society. Most respondents found that humans are superior to animals, that all animals have value, that people are obliged to do good to all animals, and that all animals have a right to life. The most highly valued arguments for that attitude were: animals are living beings, animals are sentient, animals are important for the ecosystem, animals are useful to people and people have a relationship with animals.

Several bio-ethical problems occur in the field of equestrian sports. One of those is the use of veterinary drugs. In my contribution I will introduce the use of the Ethical Matrix (Mepham, 2000). The Ethical Matrix sets a framework to help groups and individuals to work through a debate of e.g. the use of veterinary drugs in equestrian sports. The application of drugs is consid-
ered a bio-ethical problem when good reasons for the use are both supported and opposed. The Ethical Matrix is based on three ethical principles, respect for wellbeing, for autonomy and for justice. The principles reflect the common ethical concerns in society and are based on two large traditional ethical theories and the modern societal contract theory. The Ethical Matrix will help to consider the three principles against the interest of groups. Which interest groups are included depends on the issue that will be addressed. A criterion to include a group is that they should possess ‘ethical standing’. In common morality ethical standing includes people involved and animals involved. In my contribution special attention will be paid to the role of the veterinary profession. The Ethical Matrix enables us to define a checklist of concerns and provokes structured discussion on a specific ethical issue.

Reference:

NORMAL ANATOMY AND ANATOMICAL PITFALLS OF THE DISTAL EXTREMITY OF THE HORSE WITH MULTI DETECTOR COMPUTED TOMOGRAPHY (MDCT)
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Introduction
The recently introduced technique of intra arterial contrast enhancement CT (IA-CE-CT) for diagnosis of Palmar Foot Pain (PFP) need the use of modern Multi Detector Computed Tomography (MDCT) technology [1, 2]. The purpose of this study was to describe normal native MDCT – anatomy of the horse foot.

Material and Methods
10 cadaver fore feet were collected from horses that did not die from orthopaedic-related diseases. Feet were amputated at the carpus, frozen at -18°C and defrosted during 24 hours before scanning with a 64-slice Philips Brilliance™ using an adapted protocol [3]. MultiPlanar reconstructions (MPR) were made using a slice thickness of 2.7 mm and a slice increment of 1.0 mm. The different structures were evaluated using a soft tissue window (W=200; L=100) and bone window (W=2000; L=800). MDCT images were finally compared with macroscopic dissection.

Results
All horses used in this study were of non-professional level. The average age was 10.6 years, with a youngest horse of 2 years and an oldest of 18 years.
Anatomical results:
Normal sagittal, coronal and axial MDCT – images are presented and correlated well with macroscopic dissection. Bone structures and foot balance can be evaluated in detail using the bone window. The different soft tissue structures can be observed using the liver window, they are visualised in different densities and most of them are clearly defined in different planes.
Anatomical pitfalls:
It is easy to have false positives when interpreting the collateral ligaments of the distal interphalangeal joint.

Conclusion
Modern MDCT scanners offer the possibility of visualising both bone and soft tissue structures such as tendons and ligaments. This study presents a complete atlas of normal native MDCT – anatomy with illustration of some potential anatomical pitfalls associated with image interpretation.

References

[3] Table

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Proliferative enteropathy (PE) has been recognised for several decades in the pig rearing industry as a cause of poor growth, anorexia and diarrhea (Biester and Schwarte 1931), and in 1995 the causative organism was identified as *Lawsonia intracellularis* (McOrist et al. 1995). The delay in identifying the pathogen was due to its obligate intracellular nature, and successful cultivation of the organism was not achieved until 1991, when it was cultured by using hamster cell lines (Stills 1991). Since then, it has become clear that *L. intracellularis* can infect several mammalian species (pigs, hamsters, foxes, deer, rhesus monkeys, rabbits, rats and guinea pigs) and birds (ostriches and emus) (Lawson and Gebhart 2000). In Europe, however, substantial natural outbreaks have only been reported in pigs and it is now recognized that PE is a disease of high economic impact in swine worldwide. In most other species the disease occurs as a sporadic infection. Duhamel and Wheelon (1982) described the first case of proliferative enteropathy in a horse. Since then PE is considered to be an important cause of diarrhoea, weight loss and ventral edema in weaned foals in North America, reaching epidemic proportions in certain areas (Lavoie et al. 2000). Although most reports of *L. intracellularis* infection in foals originate from North America, a few cases have been reported in England (Sainty 2002, Cehak et al. 2007) and one case in Australia has been described (McClintock and Collins 2004).

In swine and in foals PE is causing decreased weight gain and increased mortality. In all affected species the disease is characterized by the intense proliferation of intestinal crypt epithelium associated with intracellular argyrophilic bacteria, although the predilection site and the inflammatory reaction can vary among different species. Macroscopically, the mucosa is markedly thickened forming ridge-like folds. Histologically, the thickened mucosa results from proliferation and elongation of crypts with increased mitotic rate and absence of goblet cells, whereas the villi are shortened. An additional feature in rabbits is the expansion of the lamina propria by infiltrating histiocytes (Lawson and Gebhart, 2000).

In foals the diagnosis *L. intracellularis* enteritis is made on basis of clinical symptoms, blood parameters (hypoproteinemia) and confirmed by PCR from fecal samples or serology. First reports on the validity of these tests suggest that PCR on feces appears to be less sensitive than serologic testing. Though there is a need for more investigations, as little is known concerning the time of appearance of the antibodies in the blood and the beginning of fecal excretion of the bacterium in foals. Response to treatment of affected foals with macrolids is reported to be very good and foals recover completely after being treated for several weeks. The appropriate management of sick foals on a breeding farm takes in account the source of infestation, the isolation of affected foals and hygienic measures. As PE is known to affect a large number of animal species, the sources of contamination of foals by *L. intracellularis* are likely to be numerous. An effort should be made in each case to determine the possible cause of infection (neighborhood of a pig farm, transportation in a lorry for mixed uses,...). Affected foals should be isolated in the first days after diagnosis and treatment, as first data indicate that environmental contamination by fecal shedding from infected foals is likely to be minimal once antimicrobial treatment has been initiated.

**References:**

  J Am Vet Med Assoc 215, 511–514
  *Lawsonia intracellularis*-like organism infection in a miniature foal.
  Proliferative enteropathy due to an infection with *Lawsonia intracellularis* in a Warmblood filly.
  Pferdeheilkunde 23, 526-530
  *Lawsonia intracellularis* infection in a 12-monthold colt in Belgium
**Clinical Epidemiology and Management of a Rhodococcus Outbreak**

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*Rhodococcus equi* (*R. equi*) pneumonia and diarrhea cause sporadically or endemically high losses in foals and consequently economic losses in breeding farms because of high morbidity and mortality. *R. equi* is a gram positive bacterium (Actinomycetales, group of *mycolata*, genus *Rhodococcus*) with a lipid-rich cell envelope. This cell envelope forms a permeability barrier to hydrophilic compounds and influences the resistance against many antibiotics. The mucolic acid-rich cell envelope provides furthermore the ability to survive and proliferate in the macrophages of foals up to the age of 5–6 month thereby causing pneumonia and pulmonary abscesses (Martens et al. 1982). Older foals and adult horses with an intact immune system do not develop pulmonary disorders due to *R. equi*..

The gross lesions induced by *R. equi* in the lung of foals are multiple firm nodules that can merge in later course of disease. In some foals miliary pyogranulomatous lesions are observed (Martens et al. 1982). Considering the biology of *R. equi* in the environment on a horse breeding farm, a stunning ability of this bacterium to survive in soil and dust under dry and sunny conditions was already described in the early 20th century (Magnusson 1923, Takai et al 1987, Muscatello et al. 2006).

Several farm management measures and their influence on the disease rates on endemic breeding farms have been evaluated. The effect of separation of sick foals from other foals, high hygiene and medical level in the perinatal period, remove manure from pastures and paddocks, reduction of dust and reduce animal density on the farm was investigated (Chaffin 2003). So far no specific farm management measures have been identified, which could account to a reduced morbidity of foals on *R. equi* endemic farms. The common characteristic of farms with endemic rhodococcosis is the high animal density. A numerous horse population on pastures, the high shedding of *R. equi* by infected foals in their manure increases environmental contamination. This does lead to a high infective challenge especially for foals as adult horses do not develop a clinical rhodococcosis.

Over the last 40 years clinicians and researchers have been trying to develop medical prophylactic methods for foals to prevent them to get the rhodococcosis of....
endemic breeding farms. Attempts were made to vaccinate mares and foals against R. equi without positive results (Becu et al. 1997, Hullmann 2006). Some positive data show the efficacy of repeated administrations of R. equi-hyperimmune serum to neonates and older foals (Cohen 2002, Madigan 1991) but contradictory results have been reported as well (Hurley and Begg 1995, Schulte 2005). Because Plasma has a general beneficial effect on young foals and has a relatively good safety it is widely used. It should be administered in the first days of live and repeated when exposure to R. equi is thought to be high. Further immunostimulatory treatment in foals was evaluated without success (Bau- mann 2006).

Several studies were performed to evaluate the effect of metaphylaxis through administration of macrolid antibiotics within the first weeks of live (Venner et al. 2009, Chaffin 2008). The prophylactic application of azithromycin or tulathromycin in order to prevent pulmonary abscesses in foals was evaluated on a stud with endemic Rhodococcus equi pneumonia (Venner et al. 2009). Results showed that the application of azithromycin (10 mg/kg BW orally) for 28 days postnatally does not reduce the prevalence of pulmonary abscesses in foals on a stud with endemic R. equi pneumonia. Similar result were collected with Tulathromycin. So none of these management related measures and none of medical methods have shown to provide protection to the foals and reduce the morbidity on endemic breeding farms. According to our investigations on a large warmblood breeding farm consisting of about 800 broodmares, the only reliable methods of reducing morbidity and mortality amongst foals are diagnosing the disease in a very early state and initiating the appropriate therapy. Diagnoses are obtained through clinical findings associated with WBC, isolation of R. equi and diagnostic imaging. Rhodococcus equi can be isolated from faeces, tracheobronchial secretions, nasal swabs or in air samples. It was shown recently that diagnostic imaging techniques such as sonography and radiography are more sensitive in diagnosing R. equi pneumonia in foals than culture or PCR (Walter 2006). Furthermore these imaging methods can help to detect abscessing pneumonia in a very early stage of the disease. In daily practice, careful evaluation of the foals through the owner and thorough clinical examination along with sonography of the lungs at first signs of the disease will help to detect foals in an early state of R. equi pneumonia.

References:

- Baumann I. Prophylaxe von Lungenabszessen durch Rhodo-
- Becu T, Polledo G, Gaskin JM (1997): Immunophylaxis of Rh-
dococcus equi pneumonia in foals. Vet Microbiol. 56, 193-204
- Chaffin K 2008
- Giguière S, Jacks S, Roberts GD et al. Retrospective comparison of azithromycin, clarithromycin and erythromycin for the treat-
- Martens RJ, Fiske RA, Renshaw HW. Experimental subacute foal pneumonia induced by aerosol administration of Corynebacte-
- Muscatello G, Gerbaud S, Kennedy C et al. Comparison of con-
centrations of Rhodococcus equi and virulent R. equi in air of stables and paddocks on horse breeding farms in a temperate climate. Equine Vet J. 2006;38:263–265
- Schulte S. Wirksamkeit von Hyperimmunserum zur Prophylaxe der Rhodococcus equi-Pneumonie. Thesis, School of Veterinary Medicine Hannover 2005
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THERAPEUTIC CONSIDERATIONS IN FOALS
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Introduction
The subject of drug therapy in the foal is of great interest because many conditions may occur during the first few weeks of life. General guidelines for therapeutic intervention in the neonatal foal are usually extrapolated from results of drug trials in neonates of other species or adaptation of drug dosages in adult horses. However, the intensity and duration of pharmacological effects of a drug are related to the concentrations attained at the site of action and the rate of elimination of the drug. Differences between neonatal and adult animals in pharmacological effects exist and generally can be attributed to altered disposition processes during the neonatal period.

Drug absorption
During a brief period after birth, the intestinal epithelium is more permeable and hence poorly absorbable drugs will be absorbed during this time span. Furthermore the pH in the stomach and the upper small intestine of the foal is relatively high but decreases with the ingestion of milk and the increase in pepsin secretion. Especially weak organic drugs are better absorbed during this initial period. Another consideration is that drugs administered to the mare that pass into milk will be readily absorbed in the neonatal foal. Drug that undergo extensive “first-pass” metabolism in the liver, such as trimethoprim, will have higher systemic availability in neonatal foals.

Distribution
Changes in the body composition largely accounts for not only species variations, but also age-related differences in the distribution of drugs. For example: a larger percentage of the body weight of foals is taken up by total body water (75% versus 55% in adult horses). Also extracellular fluid volume is larger in neonatal foals compared to older foals. This larger volume of ECF is consistent with an apparent higher volume of distribution and hence plasma concentrations of these drugs will be lower; higher concentrations may be attained at the site of bacterial infections (especially for highly polar drug such as penicillins, cephalosporins and non-steroidal anti-inflammatory drugs).

Hepatic metabolism
Metabolism mainly takes place in the liver and kidney and converts lipid-soluble drugs to metabolites that are more readily excreted by the kidney. In all mammalian species the hepatic metabolising enzyme efficiency is not optimal during the first few days of life and therefore many drugs have prolonged half-lives during the neonatal period.

Renal excretion
The renal excretion mechanisms (glomerular filtration and tubular secretion) are not fully functional at birth. The maturation process of these mechanisms occurs independently from each other at rates that are species specific. The urinary pH of foals (acid) also differs from adult horses and this should favour the reabsorption of weak organic acids. Overall renal function appears to mature within the first 1-2 weeks after birth.

Summary
When treating equine neonates, determining the choice of drug, route of administration and dosage should take the characteristic physiological factors of this period into account. When developing longer-term treatment protocols for critically ill foals, the apparent changes in these physiological factors should also be taken into account. Unfortunately the lack of specific data on developmental physiology and pharmacokinetics hampers establishing adequate dosages and dosing regimes. Therefore the use of drugs in young foals requires greater precision in dosage, more attention to the route of administration and a close monitoring of pharmacological effects.
HOW TO RECOGNIZE A POTENTIAL “WEAK” FOAL
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Probably many horse breeders are keen on getting his mare pregnant, keeping it pregnant and getting the foal delivered. In many instances he or she can sit back and enjoy the newborn. However, a newborn foal can do a good job in concealing a less optimal condition because this is functional for his ambition to stay alive a potential threatening surrounding. The first 24–36 hours it might seem doing reasonably well; even if it is suffering from a pathological condition, it will rely on energy reservoirs until they are empty before obviously crashing.

So in order to anticipate on a worsened situation a horse owner an his veterinarian has to recognize early signs of a pathological situation. In addition he or she needs to be aware of risk situations and in order to prevent further damage, he needs to be prepared. First of all we need to know the criteria of being a normal newborn: It normally will be born within 30 minutes. The sucking reflex should be present in 2–20 minutes. It will sit with elevated head within 4 minutes after birth, stand within at the most 2 hours with a mean of 60 minutes and will drink also within 2 hours average. Within the first 8 hours it will have to have consumed 2 liters of good quality colostrums. After 6–10 hours the first urine should be passing, and after 24 hours the yellow pasty feces should be seen and thus meconium has been eliminated. However, an abnormal foal is slow with basically everything and might stand under the udder a lot as if it drinks but will not actually consume enough milk. It lays down more than a normal foals and is weaker. It might follow the mother not closely when they move around the box, but again, will pretend to be normal. Since normal healthy foals sleep a lot and drink in small potions many times a day, or can be not very straight legged, it can be not that easy to discriminate between a healthy foal and a foal concealing its weakness.

Several risk factors can be pointed out for a potential problematic foal. Not only dysmature or premature foals that are recognized more easily by the low birth weight, silky coat, floppy ears, soft droopy lips, tendon laxity or red mucosal membranes are at risk. Any cause that will lead to diminished colostrum and milk consumption will eventually lead to hypoglycemia, hypovolaemia and hypoproteinaemia. This will result in an even weaker foal and makes the foal even more susceptible for life threatening infections regardless the initial cause.

Risk factors can be divided in insults happening before birth such as dysmaturity caused by maternal problems, during birth such as hypoxia during parturition or fractured ribs as a result of the partus. Directly after birth breaking of the umbilical cord too soon as well as prolonged bleeding from the umbilical cord can anaemia related weakness. Delayed or too little milk consumption can be caused by the previously mentioned conditions but can also be the result of restlessness of an inexperienced mare, too many spectators, or mastitis. In addition premature lactation and therefore loss of colostrum will lead to increased risk for infections in the foals. Unhygienic condition around the partus is also a reason for increased infection risk. We still encounter umbilical cords that are ligated by stable ropes by the owner leading to septicemia at the University clinic in Utrecht.

In many instances the owner does notice that the foals is too slow, but has the idea that by trying to get some milk in the foal, often by using a bottle in a badly swallowing foal, can be tried for quite some hours before calling in the help of the vet. This might result in a situation that in the first 8 hours the foals has not ingested the minimal quantity of glucose, fluid and proteins. If then the veterinarian is trying its very best at home for some hours, in many instances the foals is referred after 24 hours in a hypovolaemic, hypoglycaemic and already infected state.

A mature foal only has a glucose reserve for 18–24 hours, whereas a premature foal has even less reserve. Too many foals are still not making it due to the consequences of malnutrition in the first 24 hours. This results in a viscous circle of getting weaker, drinking less, getting hypoglycaemic and dehydrated and getting infected as a result of failure of passive transfer of immunoglobulin’s.

The veterinarian can play an important role in educating the owner in being prepared. Attention can be paid not only to the optimal health status of the mare, but in preferably not moving the mare to an different stable within 6 weeks of giving birth in order to enable the mare to have colostrum with immunoglobulin’s against pathogens of the surrounding of where the foals is.
born. The mare should be vaccinated around 6 weeks prepartum. Attention should be paid to the hygiene of the stable and clean or sterile materials can be organised that might be needed around the parturitions ch as sterile clamps for the umbilical cord. If the mare has premature lactation, the colostrum collected and frozen and given to the foal during the first day of life at least 500 ml every 2 hours. Attention should be paid to a defrosting temperature of maximum 37 grades Celsius. If this is not possible, the vet can be asked to give hyper immune serum preventively. The owner can take care for good hand hygiene, minimize entering the stable, disinfecting the umbilical stump. If the foal has a slow start it should be guaranteed that it drinks at least 500 ml every 2 hours. Whilst doing this aspiration of milk should be prevented especially with bottle feeding. This might require veterinary assistance at an early stage. If the veterinarian and the owner are aware of the predisposing conditions, and are alert on any indication that the “normal” criteria of development of a newborn foals are not met and do respond to this immediately adequately this would benefit all.

References:

BASIC PRINCIPLES OF FOAL INTENSIVE CARE
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The management of the compromised neonatal foal consists of certain elementary topics that should be addressed. Firstly, a fast and thorough clinical evaluation needs to be made in order to decide what priorities exist. It might need immediate resuscitation. This is evaluated by assessment of respiratory rate, effort in breathing, peripheral pulse, heart rate and peripheral temperature, mucus membranes and fluid balance based on clinical examination.

If respiratory support is required, sternal positioning and nasal oxygen insufflation with or without the use of a nasotracheal tube at a speed of 5 liters/ min. can be sufficient, however cuffing the tube and ventilation with an Ambu bag might be necessary. Hydration state can be assessed by evaluating eyeball position, skin turgor and urine output. Circulatory state can be assessed by pulse quality, capillary refill time and peripheral temperature. Sunken eyes in a 45 kg weighing foals suggest a deficit of 4-5 liter. If hypoglycaemia is suspected initial fluid administration can consist of 2-4 ml/min of a 5-10 percent dextrose solution for a 50 kg weighing foal. Maintenance fluid therapy of 175-200 ml / hr is often sufficient to guarantee adequate urinary output. Secondary entropion can be managed by evertting the lower eyelid, or putting lubricant in the eye.

After administration of a glucose source hypothermia can be treated by using heating pads or blankets. Heating lamps can be used but should be placed far enough away from the skin of the foals to prevent burns. The environment should be clean and draft free. Energy loss due to convulsions should be prevented by diazepam as a first choice but respiratory support might be needed.

Failure of passive transfer of immunity if often already a fact but if not, should be prevented at all times. The foals should receive at least 2 liter of good quality colostrum within the first 8 hours of life. If this is questionable or absent, IgG supplementation should be given iv. Bovine colostrum has been shown to be well absorbed and protective against infection in the foal, but the half life is shorter than normal, so equine colostrum is preferred of possible. As long as the foal is unable to drink milk by the mare, she should be milked every 1-2 hours in order to maintain milk production.

Enteral feeding of a normal foals is as high as 20-30 % of their body weight in milk per day. A sick foals of 50 kg should be given at least 450 ml per 2 hours as a minimum of 10 % BW. If neonatal foals have not maintained an adequate oral intake or are expected to be deprived of any oral feeding for longer than 24-36 hours, they are candidates for parenteral feeding. However it is expensive and not without risk. Especially septicemia due to contamination of the infusion line, metabolic derangement, or thrombophelbitis are complications that can occur.
(Further) Infection of the neonate should be prevented at all times. Not only a good hygiene around the foal is required, but also from caretakers and the veterinarians. The umbilicus should be kept clean and dry. If diarrhoea is present the perineal area should be kept clean and dry. Meconium retention in these foals is and should be anticipated upon. Passive flexion and extension of the legs is necessary to maintain circulation, prevent decubitus and stimulate muscle tone.

Treatment of infection requires broad spectrum antibiotic therapy. In many cases gastroduodenal ulceration will develop and after the time period of colonisation prophylactic antiulcer medication is indicated in many cases.