Proceedings of the European Veterinary Conference Voorjaarsdagen

Amsterdam, the Netherlands
Apr. 23-25, 2009
**How to survive the first two years in practice**
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Dierenhospitaal Visdonk, the clinic I have been working in since graduation, is a referral clinic with three full-time equine veterinarians including myself. I have the privilege to work with both a board certified surgeon and a board certified specialist internal medicine. My work consists of ambulatory clinics, general anesthesia, lameness examination, diagnostic imaging and treating hospitalized patients. In this presentation I will share my experiences as a starting veterinarian and I will describe recognizable situations and problems I have encountered in practice. This way I hope I can help and advice other starting vets.

Working on patients without a professor or teacher looking over your shoulder is the first thing to get used to. There is no one to correct you if necessary and no one to give additional support when needed. Fortunately this is something you get used to very quickly. And of course the only wise thing to do when you are not sure of your diagnoses, is ask your colleague or refer the horse to a clinic or colleague. If you handle it carefully, no client will think you are not capable of treating their horse!

“What?! Who is coming? Martijn? No… I don’t want the new vet. I want one of the partners! They always treat my horse, so I want one of them to do this vaccination!”. What a disappointment, because vaccinating is definitely something I think I am capable of doing. And a vaccination is not really a treatment to impress an owner using my veterinary knowledge and skills. What else can I use to impress? Charms?! I think I missed that class… But after working a longer period of time, I experienced routine visits are very valuable for earning trust of clients en build up credits for other situations.

I thought I was quite good prepared for treating horses in practice. But what was different from treating horses at the University, was that now it did not end with treating the horse. There is an owner attached to each horse and that does not make the job any easier. But how can you prepare a student for difficult clients?

**The prevalence of occlusal lesions in equine cheek teeth from horses with clinical signs of apical pulpitis compared to controls.**
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**Aims**
To assess prevalence and severity of secondary dentinal defects and infundibular caries observed on occlusal examination of teeth from horses showing clinical signs of apical pulpitis compared to normal controls.

**Methods**
Occlusal surfaces of 23 mandibular and 21 maxillary cheek teeth, extracted from horses with clinical signs of apical pulpitis attributed to those teeth, were examined using a fine probe. Occlusal surfaces of 50 mandibular and 40 maxillary cheek teeth from cadavers with no known history of dental disease were assessed as controls. Triadan positions and eruption ages were matched as closely as possible. Occlusal secondary dentinal defects were identified and graded. Maxillary infundibular caries was identified and graded using the modified Honma system. The prevalence of lesions in clinically affected and control teeth was compared using Fisher’s exact test.

**Results**
Secondary dentinal defects were significantly over-represented in diseased teeth (P<0.001). Thirteen out of 23 diseased mandibular teeth had defects compared to none of the controls (0/50). As a test for apical pulpitis in mandibular teeth, assessment of secondary dentinal defects had sensitivity of 56.5% and specificity of 100%. Eleven out of 21 diseased maxillary teeth had defects compared to 1/40 controls (sensitivity 52.3%, specificity 97.5%). There was no significant difference (P=1) in prevalence of infundibular caries in diseased teeth (11/21) compared to controls (21/40). However infundibular caries of Honma grade ≥ 2 was significantly over-represented (P<0.005) in the diseased teeth (6/21) compared to controls (1/40). Such lesions were always in rostral infundibula.

**Conclusions and Practical Significance**
These results confirm that careful examination of occlusal secondary dentine is a vital component in investigation of suspected apical pulpitis in equine cheek teeth. Results suggest that infundibular caries is a poor predictor for apical pulpitis.
indicator of apical disease. However infundibular caries
of Honma grade ≥ 2 may be clinically significant.

Acknowledgements: The Horse Trust

Figure 1: Occlusal anatomy of mandibular cheek tooth (CT) of Triadan 07-10 (a), and also of maxillary CT of Triadan positions 07-10 (b). The locations of the dark-staining secondary dentinal areas are assigned names related to their anatomical location for the purposes of this paper. The endodontic numbering systems of Dacre 2005 (1) and DuToit 2008 (*) are also illustrated. (See appendix figure 3 for occlusal anatomy for teeth of Triadan 06 position.)

Figure 2: Tooth 406 extacted from a 4 year old pony showing clinical signs of apical pulpitis attributed to this tooth. There are severe defects (grade 3) in 4 areas of secondary dentine. The middle lingual area of secondary dentine is also roughened (grade 1).

Figure 3: Tooth 209 extracted from a 14 year old horse showing clinical signs of apical pulpitis attributed to this tooth. The rostral infundibulum (RI) has caries involving cementum and enamel (Honma grade 2). The caudal infundibulum (CdI) has caries involving cementum only (Honma grade 1ii).

EQUINE JOINT TREATMENT: A NEW APPROACH?
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Introduction
In the equine industry, lameness due to joint injury and disease is the most prevalent cause of diminished athletic function and represents a large majority of the equine clinician’s caseload. There is a constant pressure on veterinarians to treat performance-limiting joint problems successfully, preferably with something inexpensive, effective, and risk-free. Ideally, in addition to pain reduction, treatment of joint disease should serve to arrest or slow progression of lesions, which is called chondroprotection or disease modification.

Conventional medical treatments
1. Nonsteroidal anti-inflammatory drugs.
NSAID’s mainly act through the inhibition of the cyclooxygenase enzyme. In order to overcome negative side effects after longterm use, selective COX-2 inhibi-
Polysulfated Glycosaminoglycans (PSGAG).

PSGAG (Adequan®) has been administered both intramuscularly and intrarticularly in intramuscularly with varied success. According to clinical experience PSGAG, given IA, are the most effective chondroprotective medication available. Recent scientific evidence shows positive effects of IA administered PSGAG. PSGAG may reduce the joint’s ability to resist infection, but combined with an aminoglycoside (125 mg amikacin sulphate) the risk of joint infections is reduced. IM administration has been commonplace, but results have been less impressive.

2. Corticosteroids

Corticosteroids remain the most potent anti-inflammatory medications used, but have potential negative effects on cartilage matrix metabolism. Many clinicians currently use lower doses that may be associated with chondroprotective properties. Methylprednisolone acetate (MPA), triamcinolone acetonide (TA) and betamethasone esters are the three commonly used IA corticosteroids. Betamethasone was the first product studied and no deleterious effects to the articular cartilage were demonstrated in rested or exercised horses; however betamethasone has a short lived clinical effect. In contrast, triamcinolone (TA) improved cartilage and synovial membrane parameters suggesting a possible chondroprotective effect. Using MPA, grave deleterious effects on cartilage, but no effect on subchondral bone or cancellous bone was observed. Based on these findings the use of TA is advised, especially in high motion joints. On the other hand, MPA has shown a faster and longer lived clinical response in clinical settings. Anecdotal reports of laminitis suggest a narrower therapeutic index with TA as compared to betamethasone and MPA. In relation to the induction of laminitis it has been suggested that the total body dose of TA should not exceed 18 mg, MPA 200 mg and betamethasone 30 mg. Anecdotal evidence suggests protective effects of hyaluronic acid (HA) when combined with intra-articular corticosteroids; recent evidence suggests the beneficial effects of a combination of glucosamine and IA corticosteroids. IGF-1 in conjunction with TA has the potential to provide beneficial anabolic effects not seen with TA alone.

3. Hyaluronic acid or hyaluronan (HA)

There are conflicting results regarding the use of HA, but this drug (15-30 mg/joint) has been used clinically for years, both alone and in combination with other substances. Reported beneficial effects include anti-inflammatory properties, improved lubrication/ viscosity of joints, improved lameness scores and the potential for inducing endogenous HA production. In a recent study HA reduced cartilage fibrillation, indicating a chondroprotective effect. Intravenously administered HA reduced lameness scores, a decrease in PGE2 and total protein in the synovial fluid and improved synovial membrane histologic scores. Most recently the use of oral HA has been shown to reduce synovial effusion.

4. Polysulfated Glicosaminoglycans (PSGAG).

Polysulfated Glicosaminoglycans (PSGAG). PSGAG (Adequan®) has been administered both intrarticularly en intramuscularly with varied success.

5. Oral Joint supplements

Glucosamine, chondroitin sulfate, and ASU (unsaponified avocado soy) can benefit those suffering from joint disease. They decrease the synthesis of inflammatory mediators, the synthesis and activity of metalloproteases and increase the production of cartilage matrix molecules.

None of the oral supplements or nutriceuticals is licensed, and the proof of efficacy is generally lacking, with the exception of ASU. There is considerable in vitro support for the effectiveness of glucosamine and chondroitin sulfate, in particular when used together. Anecdotally, other joint supplements, like MSM (methylsulfonylmethane), vitamins, minerals and trace elements, herbs and collagen hydrolysate, can be used in prevention or treatment of joint disease and are often ingredients of commercially available equine nutriceuticals.

Novel biologic approaches

1. Autologous Conditioned Serum (ACS, IRAP or Orthokine)

ACS counteracts the deleterious effects of IL-1 by blocking the IL-1 receptor of cartilage cells. ACS is generated by incubating venous blood in the presence of medical grade glass beads hence activating peripheral blood leukocytes to produce elevated amounts IL-1ra. Following centrifugation and extraction, ACS is portioned and either stored until use or injected into the joint. The product is not detected in drug testing and has chondroprotective effects. Frisbie et al. (2007) showed that ACS significantly improved clinical lameness as well as histologic parameters in the synovial membrane of horses. In a long-term study, ACS showed similar effectiveness when compared to corticosteroids, but the effects lasted longer and may only occur after several weeks (personal observation). Good candidates for ACS are horses with synovitis/ capsulitis, unresponsive cases, cases that return in three months with pain and inflammation, horses with severe joints that have a history of poor performance, focal cartilage diseases and bone cysts. In general 2 to 3 treatments of a joint with a 8 – 14 day intervals are performed. No adverse effects have been reported yet.
2. Platelet Rich Plasma (PrP)
PrP is an autologous source of growth factors, like TGF-β, PDGF and IGF-1 and can be obtained by a double centrifuging technique. Platelet concentrates are an important source of growth factors and there is clinical and experimental evidence that supports beneficial anabolic effects on musculoskeletal tissue. Carmona et al. (2005) showed that PrP injected intraarticularly diminished synovial effusion and the degree of lameness in horses affected with severe joint disease. In this study the affected joints were injected 3 times at 2 weeks interval.

3. Insuline-like growth factor- 1 (IGF-1) and Recombinant equine growth hormone (reGH)
IGF-1 has been shown to improve the cartilage repair process. Maintenance of adequate IGF-1 levels after cartilage repair procedures is complicated by the short half-life of IGF-1. To prolong IGF-1 delivery, IGF gene therapy, using adenoviral vectors carrying the genes of IGF-1 (and IL-1Ra), has been explored and IGF-1 gene therapy has been used in cell-based transplantation procedures. Intramuscular administration may be a more efficient means of delivery of IGF-1 to joints for cartilage resurfacing initiatives.

ReGH has the potential to influence articular cartilage by stimulation of IGF-1. These growth factor based therapies have been used experimentally in Australia and the US.

4. Stem cells
Stem cells as a treatment for osteoarthritis or cartilage repair have been used clinically. Subchondral bone cysts, cartilage damage and/ or loss, and torn menisci were treated by use of bone marrow derived mesenchymal stem cells (MSC) mixed in a fibrinogen gel or in PrP. The MSC therapy has yielded favorable results. Cases that did not respond well, had a severely damaged joint surface.

5. Other treatment approaches
Anecdotally, other intra-articular treatments using morphine, ketamine, joint flushing, sarapin, DMSO, tiludronate, bufexamac and botulinum toxin type A can be used by equine clinicians.

In case of interest, a reference list can be obtained by email contact with the author.

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Diagnosis and Treatment Alternatives for Various Causes of Upper Airway Obstruction (in the Horse)
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Diagnosis of upper airway obstruction

History: Abnormality of the patency of the upper airway can be manifested by abnormal upper respiratory noise and/or a decrease in performance. The history is often different for sport horses than for racehorses. In racehorses, a sudden drop in performance in the last half of the race with and without an abnormal respiratory noise is reported. Sport horse riders report a respiratory noise more marked with collection, either without a decrease in performance or with a progressive decrease in performance.

Physical exam: Although endoscopic examination is the most helpful diagnostic aid, an external physical exam should focus on ruling out other causes of airway obstruction or the presence of infection in the upper airways. First, external symmetry of the nasal cavity and sinus would help identify or indicate possible obstruction of the nasal passage. Functional deficits caused by vasodilation of the nasal submucosal vasculature (e.g., Horner syndrome) should be identified. Finally, the presence of nasal discharges and the size of intermandibular lymph nodes should be determined; significant inflammation of the upper airway would dictate gullet pouch endoscopy.

Sound analysis: An objective means to identify and characterize abnormal sounds is spectral analysis. The band of abnormal intensity for horses with DDSP is 18-77 Hz. RLN leads to higher intensity frequencies in three bands centered at 0.3, 1.6, and 3.8 kHz. This technique is quite helpful, yet limited by the absence of criteria for identification of other upper respiratory diseases or for concomitant obstructions.

Endoscopic exam: Videendoscopic examination during exercise is the gold standard. Until recently, the most controlled evaluation method for was high-speed treadmill videendoscopy. The advent of wireless videendoscopy will greatly increase the availability of this diagnostic modality. This is critical for dynamic disease such as some grades of RLN, midline collapse of AE fold, and DDSP. Remember that 80% of horses with intermittent DDSP on the treadmill do not manifest abnormality during resting endoscopy.

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Proceedings of the European Veterinary Conference - Voorjaarsdagen, 2009 - Amsterdam, Netherlands
**Imaging:** Chalmers et al. introduced laryngeal ultrasound. The mid-ventral window is promising for the diagnosis of DDSP, while the lateral window is most helpful in identifying the status of the cricoarytenoid lateralis muscle and lateral surface of the arytenoid cartilage to confirm the presence or absence of chondritis or abscessation.

**Treatment alternatives**

**Dorsal displacement of the soft palate:** Any horses with pharyngeal inflammation should be treated first with systemic, and topical, anti-inflammatory agents. Horses with structural abnormality of the larynx and nasopharynx such as sub-epiglottic cyst, sub-epiglottic granuloma and ulcers, palatal cyst, should have these lesions addressed first by laryngotomy or videoendoscopic laser.

Tack changes to keep the tongue under the bit, decreasing head flexion, and preventing oral breathing are often recommended. Experimental and treadmill data raise questions about the tongue-tie's value. An external device that displaces the larynx forward and dorsally effectively controls DDSP in experimentally induced DDSP.

**Surgical Management**

There are two categories of surgical treatments: those that target intrinsic nasopharyngeal structures (cyst removal) and palatal stiffness (thermal palatoplasty by cautery or laser, chemical palatoplasty by injection of sclerosing agents, and tension palatoplasty) and those that target the position of the larynx and hyoid bone, such as strap muscle resection and laryngeal tie-forward.

There is a clear potential benefit to the removal of granulomas, cysts, and abnormal subepiglottic tissues. The rationale for a targeting procedure that increases palatal stiffness is being questioned given the failure of sustained change in stiffness or morphology of the soft palate. Furthermore, thermal cautery of the soft palate was recently shown to be largely ineffective.

Procedures that affect the position of larynx/basihyoid are 1) strap muscle resection, which is bilateral partial stenothyroidectomy (with a reported success rate of 58% to 70%) and 2) the laryngeal tie-forward, which appears to further increase the success rate by 10-20%.

**Laryngeal obstruction:** The animal’s intended function determines the extent and type of treatment. Some treatments are more effective at reducing abnormal respiratory noise while others reduce airflow obstruction.

**Treatment of abnormal upper respiratory noise:** The bilateral ventriculocordectomy procedure has been shown experimentally to be the most effective treatment in reducing abnormal upper respiratory noise.

**Treatment of poor performance:** To treat poor performance, the following options are available: ventriculocordectomy, partial arytenoidecotomy, laryngoplasty, and laryngeal reinnervation.

- For treatment of early laryngeal hemiparesis or minimally debilitating obstruction, ipsilateral ventriculocordectomy is appropriate.
- For horses experiencing full arytenoid collapse at exercise, laryngoplasty is still the gold treatment for performance horses. Sport horses and draft horses have a success rate around 90% while Thoroughbred racehorses competing at 2400 m or less have a lower success rate of 50-67%.
- Partial arytenoidecotomy should now be recognized as an effective, albeit inferior, alternative to laryngoplasty.
- Laryngeal reinnervation deserves consideration for yearlings affected with laryngeal hemiplegia. This procedure allows horses to return to racing at a rate similar to that of a laryngoplasty, but with a prolonged delay that minimizes the justification for using this procedure on horses in race training.

Laryngeal reanimation through the use of neuroprosthesis is an emerging modality of treatment and clinical results are not available to date.

**References**

TECHNICAL ADVANCES IN UPPER AIRWAY SURGERIES (IN THE HORSE)
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Laryngeal hemiplegia (RLN)
Treatment of abnormal upper respiratory noise: The bilateral ventriculocordectomy procedure has been shown experimentally to be the most effective treatment, but acceptable results are seen with ipsilateral laser-assisted ventriculocordectomy (VC). Therefore, a unilateral laser-assisted ventriculocordectomy or bilateral ventriculocordectomy is the preferred treatment for use in sport and draft horses where abnormal respiratory noise is the main complaint.

Technical update: The VC procedure is more commonly done under videoendoscopic control with the laser in three steps: 1) transection of the ventral aspect of the vocal fold; 2) transection of the caudal aspect of the fold; and 3) eversion of the ventricle. The procedure can be performed standing under sedation or under general anesthesia using nitrox or compressed to lower the inspired oxygen (target 21%). Horses under anesthesia do not do well on 21% inspired oxygen because of venous admixture. The anesthetist needs to monitor SpO2 continuously and take blood gases intermittently. So the surgeon has to be quick! As soon as the laser stage of surgery is over the air should be flushed from the system with oxygen. A suction device is needed to remove the fumes.

Treatment of poor performance
To treat poor performance associated with airflow limitation, the following options are available: ventriculocordectomy, partial arytenoidectomy, laryngoplasty, and laryngeal reinnervation. For treatment of early laryngeal hemiparesis or a minimally debilitating obstruction, ipsilateral ventriculocordectomy alone is a real option. For horses experiencing full arytenoid collapse at exercise, laryngoplasty is still the gold treatment. Partial arytenoidectomy should be recognized as an effective alternative to laryngoplasty.

Technical updates:
Laryngoplasty: In recent years, an attempt at fusion of the crico-arytenoid (CA) joint is used as part of the laryngoplasty procedure. Three techniques are used:
- Transection of the tendon of both compartments of the crico-arytenoid dorsalis and motorising burr (Parente’s technique).
- Transection of the tendon of both compartments of the crico-arytenoid dorsalis and lasering of joint (Hawkin’s technique).
- Application of PMMA into a slit made in the CA joint.

Arytenoid chondritis
The diagnosis of partial arytenoidectomy is now enhanced by laryngeal ultrasound. This modality allows identification of abscesses that are best treated by drainage instead of partial arytenoidectomy.

Technical updates: A partial arytenoidectomy may be performed better by closing the mucosa with a flap from the left aryepiglottic fold.

Dorsal displacement of the soft palate
The results of the treatment of dorsal displacement of the soft palate (DDSP) in horses have been assessed in case-control studies of thermal cautery of the soft palate (TSP) and laryngeal tie-forward surgery. The TSP procedure is apparently not very effective. Therefore, the focus of treatment should be a laryngeal tie-forward with transaction of the ST tendon. A recent evaluation of the outcome of surgery relating to the position of the larynx and hyoid bone after laryngeal tie-forward, established that the laryngeal tie-forward procedure moved the basihyoid bone dorsally and caudally and moves the larynx dorsally and rostrally. Most importantly, in relation to outcome, this recent paper revealed a more dorsal basihyoid bone and larynx position should be targeted. (Note: horses that have DDSP associated with a palatal or subepiglottic cyst/granuloma require targeted treatment for these problems).

Technical update on laryngeal tie-forward
Under general anesthesia in dorsal recumbency, a 15 cm ventral midline incision is made, extending from the rostral aspect of the basihyoid bone 1 cm caudal to the cricoid cartilage. One size 5 polybend suture (Fiberwire® Arthrex Inc., Naples, FL) is inserted at the ventral aspect of the right ST tendon and exited from the lamina of the thyroid cartilage and through the thyrohyoideus muscle 1 cm rostrally and slightly dorsally from its insertion point. The suture is then placed again through the right lamina of the thyroid cartilage slightly more dorsal (0.5 cm) and exited more dorsally than the previous bite, forming a loop in the thyroid lamina. The procedure is repeated on the left side. The ST tendon of insertion are transected. The sutures are passed dorsally to the basihyoid with the most dorsal suture ipsilateral and the ventral suture contralateral. The horse's nose is lifted so the head and neck are angled at approximately 90° and the sutures are tied so the rostral aspect of the
thyroid cartilage is ~1 cm rostral and 4 to 6 cm dorsal to the caudal aspect of the basihyoid bone.

References

THE VALUE OF DIFFERENT CLINICAL AND BIOCHEMICAL MARKERS TO ASSESS OVERTRAINING IN HORSES
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Introduction
The overtraining syndrome is a stress-related syndrome and resembles other stress-related syndromes like burn-out. We usually think of stress as a negative experience. But from a biological point of view stress can be neutral, positive or negative. For an optimal performance a certain level of stress is necessary. Stress causes a temporary decrease in function followed by an adaptation that improves function. This improvement in function (called overcompensation) is seen as positive stress. In the training response, overload is the stress that causes fatigue (temporary decrease in exercise ability), and improved performance (following recovery from fatigue). When the body experiences stressors it responds with secreting a whole array of hormones to reestablish homeostatic balance. The two main stress-related hormonal axes are activated: the sympathetic-adrenal medullary (SAM) axis and the hypothalamic-pituitary-adrenocortical (HPA) axis.

Prevalence
Since exercise stress is a culmination of several different stressors, including physiological, environmental, social and psychological stressors, not only highly trained (race) horses, but also horses involved in daily intensive or monotone exercise and competition might suffer from overreaching (OR), overtraining (OT) or burnout. The occurrence of OR/OT might be severely underestimated in equine practice and every horse referred for complaints of loss of performance without obvious other diseases should be considered as possibly suffering from OR/OT.

Diagnostic criteria
Several longitudinal studies have investigated OR/OT in horses. Not all studies were able to show decrements in performance or used control and test groups, but all contributed positively to the insight in parameters suitable for detecting early OT. From those and other studies the best indicators of overtraining in
horses from a practitioner point of view are unexplained loss of performance, weight loss despite adequate feed intake, concurrence of unspecific subclinical problems, unsuccessful treatments and changes of behaviour. Many parameters have been evaluated for their use as a diagnostic parameter, e.g. plasma lactate during exercise, HR submaximal and maximal during exercise, VO2 max, muscle enzymes, haematological and biochemical blood parameters, enzymes and proteins in muscle biopsies. So far, no parameter has proved to be conclusive. However, detection of hormonal changes seems to be a promising tool for the future. A dysfunction of the HPA axis as well as the GH-IGF-1 axis seems to be related to the development of OR/OT in horses and is worthwhile for further research. One study was able to show changes in mood state in the overtrained horses with objective measurements which is also a promising tool for future diagnosis. Symptoms associated with overtraining like behavioural changes and hormonal dysregulation are indicative of changes in regulatory and coordinative function of the hypothalamus. Overtraining is therefore believed to originate at the level of the hypothalamus and higher brain centers which might develop over time. The development over time in dysfunction of the hypothalamus might explain the many different symptoms of OR/OT described (more than 200) as well as the contradictions found in for instance blood related parameters. More research is needed to detect the time related changes and to specify the hormonal disturbances for the individual.

References

JOINT DISEASE IN HORSES: BIOMARKERS, EXPERIMENTAL MODELS AND THERAPEUTIC INTERVENTION
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Introduction
Joint disease is the most common cause of chronic lameness in equine athletes and pleasure horses alike. (Micro)trauma, inflammation and developmental abnormalities may all predispose to joint degeneration or osteoarthritis (OA). Inflammation and cartilage degradation are central features in the OA process. The in vivo study of disease pathogenesis may be aided by synovial fluid (SF) analysis.

Research objective and methods
To investigate (early) inflammatory and cartilage matrix degrading events in vivo through SF marker analysis in naturally occurring and experimental equine joint disease.

Clinical studies
In the initial phase of this project, two separate cross-sectional studies on patients with joint pain (n=22 horses) or the developmental joint disease osteochondrosis (n=38 joints) showed altered SF levels of substance P, prostaglandin E₂ and leukotriene B₄ in affected vs. control joints, but no differences in cartilage turnover markers. While the value of studying naturally occurring disease is not to be underestimated, these studies also demonstrated some of the inherent downsides of such research, including the cross-sectional nature of sample collection, and the lack of objective information.
with regard to disease progress (inciting cause, duration, previous episodes).

**Experimental models**

To abate some of these concerns and to more specifically address the effects of inflammation on SF marker levels, a longitudinal experimental study was conducted. In a two-period random cross-over design, transient synovitis was induced at post-injection hour (PIH) 0 by injection of 0.5 ng LPS in the left or right intercarpal joint of 6 horses, which were blindly allocated to receive once daily oral meloxicam or placebo, starting at PIH 2. Horses were monitored clinically and synovial fluid was sampled at PIH 0, 8, 24 and 168 hrs post-LPS injection. After a two-week wash-out period, synovitis was induced in the contralateral intercarpal joint and treatments were reversed. Synovial fluid marker levels were compared over time and between meloxicam and placebo treatment. We found that SF inflammatory mediators (prostaglandin E₂, substance P, bradykinin) and MMP activity show an early rise within 8 hours of induction of synovitis. Enhanced proteoglycan turnover in response to joint inflammation proved to be a short-lived phenomenon, evidenced by an early rise at PIH 8, a peak at PIH 24 and a return to baseline by PIH 168 of both glycosaminoglycan (GAG) release and CS846 epitope, a marker of novel aggrecan synthesis. Type II collagen degradation and synthesis markers (C2C and CPII, respectively) also showed a parallel time course in response to joint inflammation, but changes in marker levels were slower to occur and were sustained: collagen markers peaked later than proteoglycan markers (at PIH 24), and changes in collagen turnover markers persisted at one week after induction of inflammation.

**Therapeutic intervention**

With regard to treatment effects, meloxicam caused a significant reduction in lameness at PIH 8 and 24 and tended to reduce joint effusion. In addition, meloxicam significantly suppressed SF prostaglandin E₂ and tended to reduce joint effusion. In addition, meloxicam caused a significant reduction in lameness at PIH 8 and 24 and 168 hrs post-LPS injection. After a two-week wash-out period, synovitis was induced in the contralateral intercarpal joint and treatments were reversed. Synovial fluid marker levels were compared over time and between meloxicam and placebo treatment. We found that SF inflammatory mediators (prostaglandin E₂, substance P, bradykinin) and MMP activity show an early rise within 8 hours of induction of synovitis. Enhanced proteoglycan turnover in response to joint inflammation proved to be a short-lived phenomenon, evidenced by an early rise at PIH 8, a peak at PIH 24 and a return to baseline by PIH 168 of both glycosaminoglycan (GAG) release and CS846 epitope, a marker of novel aggrecan synthesis. Type II collagen degradation and synthesis markers (C2C and CPII, respectively) also showed a parallel time course in response to joint inflammation, but changes in marker levels were slower to occur and were sustained: collagen markers peaked later than proteoglycan markers (at PIH 24), and changes in collagen turnover markers persisted at one week after induction of inflammation.

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**Therapeutic intervention**

With regard to treatment effects, meloxicam caused a significant reduction in lameness at PIH 8 and 24 and tended to reduce joint effusion. In addition, meloxicam significantly suppressed SF prostaglandin E₂ and substance P release at PIH 8 and bradykinin at PIH 24 compared to placebo treatment. General MMP activity at PIH 8 and 24 was significantly lower in meloxicam- vs. placebo-treated joints, as were GAG, C2C and CPII concentrations at PIH 24. Interestingly, the clinical efficacy of meloxicam was paralleled by changes in SF mediators and markers, but not by routine parameters like WBC counts and total protein. These remained unaltered, indicating that SF WBC counts and total protein levels bear little relation to clinical symptoms in this model of acute severe synovitis, and underlining the added value of SF markers to monitor effects of therapy. It was concluded that early oral treatment with meloxicam ameliorates not only clinical symptoms and joint inflammation in acute synovitis, but may also limit inflammation-induced cartilage catabolism. Future work will focus on improvement of experimental models to more closely reflect the chronic, low-grade inflammation in OA, and on *in vitro* mechanistic studies to further unravel the links between pro-and anti-inflammatory mediators and cartilage turnover.

**Conclusions**

Synovial fluid biochemical analysis has thus far proven useful in detecting pathological changes in joint homeostasis that conventional parameters fail to identify. Analysis of mediators and cartilage markers in SF may better define the need for and effects of therapeutic strategies, and may alter timing and selection of anti-inflammatory (or disease-modifying) therapy.

**References**


**SEDATION OF COMPETITION HORSES**

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**General introduction**

Being able to perform surgical and non-surgical procedures in horses a reliable sedation and analgesia is a basic necessity in equine medical practice. This sedative and analgesic medication is necessary for both the benefit of the equine patient and the...
Butorphanol is a short acting opioid with minimal cardiovascular effects. In high doses it may cause restlessness and may induce walking behaviour. While butorphanol has probably more potency for visceral analgesia it is often used to relief mild colic.

**Anti-cholinergics**
In the past but also to day there is some controversy regarding the benefit or even the necessity of anti-cholinergic drugs in routine premedication in horses. The use of anaesthetic drugs in horses may cause unwanted effects which can be counteracted with an anti-cholinergic. But it is questionable if this means that anti-cholinergics must be included in a premedication as a standard procedure. Among others the most obvious effects of anti-cholinergics in horses are blocking the vagal mechanism, mydriasis and reducing gut motility. Blocking the vagal mechanism can be valuable when certain drugs are used. Specially drugs that cause vagal mediated as high dosed opioids. But also some surgery of the head or in the neck region may trigger vagal reflexes which may result in bradycardia or sudden cardiac arrest. In these circumstances pre-emptive administration of an anti-cholinergic is preferable because that seems to be more effective than reverse these effects after their occurrence. At the other hand a combination of an anti-cholinergic with some drugs have to be avoided. For example the alpha2-agonist drugs with their initial hypertensive phase and a marked bradycardia due to central stimulation and a vagal reflex mechanism. Mydriasis is an unwanted effect of anti-cholinergics. This ocular effect also results in visual disturbances which can cause problems with horses treated with an anti-cholinergic. Reduced gut motility may cause colic. Atropine and glycopyrrolate are both drugs with an anti-cholinergic effect. The little central action of glycopyrrolate cause less ocular effect and less visual disturbance than atropine. So pointing at this specific aspect glycopyrrolate could be the anti-cholinergic of choice in horses.

**Sedative and analgesic combinations**
In equine medical practice different combinations of sedative and analgesic drugs are used. Most drugs are combined to obtain the best effect with the lowest dose rate of each drug. The real option however is to minimize the unwanted or less wanted effects of the drugs used. An equi sedative/analgesic effect can be obtained with a high dose of detomidine or a combination of a low dose detomidine combined with butorphanol. The combination causes no or less hind limb incoordination than a high

**Drugs used for sedation and analgesia in equines**

**Alpha2-agonists**
The first alpha2-agonist to be used as a sedative (and maybe also as an analgesic) was xylazine, synthesized in 1962. Next to the sedative and analgesic effect the major side-effects were bradycardia and a biphasic blood pressure response with an initial hypertensive period followed by a moderate hypotension. In the 1980s new alpha2-agonists for horses with a much higher potency and a greater specificity at central alpha2-adrenoceptor sites were introduced: detomidine and romifidine. These drugs showed side-effects similar to those of xylazine but had a longer sedative and analgesic effect.

**Phenothiazines**
Acepromazine blocks post-synaptic dopamine receptors in the CNS and may also inhibit the release of dopamine and increase the turnover rate of dopamine. The primary desired effect for the use of ACP in equine medicine is the tranquilizing action. ACP may decrease respiratory rate but little or no effect occurs with regard to the blood gasses, pH or haemoglobin saturation. Besides a lowering of the arterial blood pressure ACP causes a vagally induced bradycardia which may be negated by a reflex tachycardic effect secondary to decrease in blood pressure. ACP has also antidyssrhythmic effects. Another effect is depression of the control of body temperature leading to a slight hypothermia.

**Opioids**
Different opioid drug have been used in equine anaesthesia and medical practice. Due to legislation rules in The Netherlands only butorphanol can be used in horses. Other opioids used successfully for many years are due to the official rules no longer the first choice when opioids are indicated. Butorphanol is a short acting opioid with minimal cardiac effects.
Another often used combination is a sedation with the oral administration of acepromazine followed by a pre-medication with an alpha2-agonist and an opioid. This procedure is used in case of patients that violently react on injections. This procedure is also suited to obtain a sufficient sedation and analgesia with a low dose of the alpha2-agonist again to prevent the incoordination caused by the alpha2-agonist.

Not only combinations of drugs but also combination of different ways of administration may be used for optimal results. For example the epidural administration of an alpha2-agonist. This procedure results in sedation and analgesia but with less influence on motor nerve activity. As xylazine has also a marked local anaesthetic effect when administered in the epidural space this drug is preferred. When epidural xylazine is combined with a local anaesthetic a sufficient sedation and analgesia is obtained to facilitate surgery in the tail and perineal region on standing horses.

Some veterinarians that use the combination alpha2-agonist/opioid add a very low dose of ketamine to the combination. Aim is to improve the analgesic effect. But this is a tricky way to handle special when the veterinarian has less experience. To much ketamine can produce a high level of instability in the patient.

The use of sedative and analgesic drugs alone and in combination always brings up the question: How long do we have to wait until a treated horses can be used in competition?

Although there are official rules to answer this question it should be taken in mind that different horses can react in a different way on medication. So to be sure a horse can be used in competition or any other work consulting a veterinarian is advisable even when official rules allow participating in competition.

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**DOPING AND THE COMPETITION HORSE: CURRENT AND HISTORICAL PERSPECTIVES.**

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**Introduction**

Throughout history humans have used a variety of substances and methods in an attempt to improve performance and so their chances in combat, mating or hunting. The Ancient Greeks had sport as part of their lifestyle by 800 BC, and within 400 years it had achieved huge importance with mass spectator events and the emergence of a hero class of athletes.1 Horse sport also emerged with chariot races prominent in the early Olympic Games. To increase endurance, the Ancient Romans fed supplements to their chariot horses, and in the following centuries war horses from the Andes to China as well as the steeds of medieval knights were given ‘remedies’ and ‘treatments’ to boost alertness, strength and staying power. In the 16th century, stimulants were known to have been given to racing horses to help them win, and the first rules prohibiting the use of ‘exciting substances’ in horse races are thought to have been introduced in England in 1666.

**Anti-doping and Medication Control in 2009**

The Fédération Equestre Internationale (FEI) is the world governing body for equestrian sports.2 Its primary mission is to advance equestrian sport by promoting, regulating and administering humane and sportsmanlike international competition. The FEI has a Code of Conduct specifically formulated to protect the welfare of the horse as well as strict rules on anti-doping and medication control; each year the FEI tests some 3000 horses at about 500 events worldwide.

The control of drug use in competition horses is more than ensuring fairness, providing a ‘level playing field’, or reassuring sponsors or public opinion, although all of these are important. Human athletes decide for themselves if they wish to take drugs, horses do not. As a result, there is a moral and ethical dimension when medicating animals. The FEI states that no horse requiring bona fide veterinary attention must be denied it.
and distinguishes between medication, i.e. veterinary treatment justifiably provided to safeguard the animal’s health and welfare, and doping, i.e. the deliberate intent to affect the performance of a horse or to mask an underlying health problem. But the dividing line between the use of medication to treat injury and disease as opposed to preparing horses for competition is often narrow and difficult to define.3

The End of ‘zero tolerance’
In recent years, the FEI has had a major overhaul of its medication policy and this is continuing. It became apparent that the so-called ‘zero tolerance’ means of doping control was ‘a fading illusion’ and unsound both philosophically and pragmatically.4 To call a positive based on traces of a drug given to a horse for legitimate clinical reasons days or even weeks before a competition when it could not possibly still influence performance was irrational. European Racing pioneered a new approach with the goal of establishing ‘reporting levels’ for therapeutic substances and defining accurate detection times for major veterinary drugs through standardised and rigorous excretion studies. Recommended limits of detection were established for a number of commonly used therapeutics using a pharmacokinetic/pharmacodynamic (PK/PD) model based on irrelevant drug concentrations in plasma and urine5 and a risk management review of factors that could be significant in administering the drug to a racehorse. The FEI also provides data on a group of medicines that might reasonably be used clinically close to an event2; specific forms and procedures must be completed and the horse examined by the official Veterinary Commission/Delegate who will advise whether it may compete under an emergency equine therapeutic use exemption.

Doping and Medication Control in the FEI
The FEI’s Equine Anti-Doping and Medication Control Rules2 are based on the general guidelines used by the World Anti-Doping Agency for human athletes and severe sanctions are available for serious abuses. There are presently three classes of offences: Doping (e.g. mixtures of drugs, masking agents, substances with no generally accepted medical use in competition horses, methods used to hypersensitise or desensitise); Medication Class A (therapeutic agents that could influence performance e.g. relieving pain, sedating, stimulating, or producing/modifying physiological or behavioural effects); Medication Class B (substances that have limited performance enhancing potential or to which horses may have been accidentally exposed, e.g. certain dietary contaminants).

The Future
There will always be some who will try to get more out of a competition animal using increasingly sophisticated exogenous aids. The FEI has sent out a clear message that it will not tolerate doping abuse; a Medication Code has been issued2 and a review is currently underway chaired by Dr. Arne Ljungqvist, Chairman of the IOC Medical Commission to study the way policies can be further harmonised to WADA to ensure horse welfare remains at the heart of the system and is fully protected. But the battle against doping is far from won, and although sport may be fairer than ever before, the ‘winning’ horse may still not be the ‘best’. The FEI will continue to fight to preserve the welfare of the horse and the spirit of the sport, and its Code of Conduct remains unequivocal: ‘…at all times the welfare of the horse must be paramount and must never be subordinated to competitive or commercial influences’.

References
2. See FEI web site: www.horsesport.org

UPPER AIRWAY ANATOMY AND PHYSIOLOGY IN HORSES
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The physiologically distinct functions of breathing, deglutition and vocalization in the horse rely on the carefully orchestrated action and anatomic arrangement of the nasopharynx, the common conduit through which both air and ingesta pass. Simply conceived, it is a muscular tube suspended rostrally from the pterygoid and palatine bones, and anchored caudally...
on portions of the hyoid apparatus and the laryngeal cartilages. During strenuous exercise, airflow velocities and airway pressures fluctuate tremendously, and appropriate sensory and motor activity must occur to maintain airway patency. Failure of these anatomic structures or neuromuscular activities results in a constellation of clinical disorders, ranging from exercise intolerance to dysphagia.

While the nasopharynx may be simply conceptualized as a muscular tube, it is an anatomically and functionally complicated structure composed of multiple muscle groups including the dorsal pharyngeal constrictors and dilators, the hyoid muscles, soft palate muscles and muscle of the tongue, and innervated by several cranial nerves, including branches of C.N. V, IX, X, and XII. The action of the dorsal pharyngeal constricting muscles and the stylopharyngeus muscle is responsible for stiffening and dilating the nasopharynx. (Figure 1; SH = stylohyoid bone, Pt.p.=pterygopharyngeus; Pp. = palatopharyngeus; Sp. = stylopharyngeus; Cp. = cricopharyngeus; Th.p.= thyropharyngeus). Contraction and shortening of these muscles form a sphincter, moved the food bolus caudally into the esophagus during swallowing. These same muscles must remain tense during breathing to stabilize the nasopharynx. The major dilating muscle of the dorsal nasopharynx is the stylopharyngeus muscle. This muscle originates on the distal portion of the stylohyoid bone and rami-fies in the wall of the dorsal nasopharynx. Contraction of the stylopharyngeus muscles pulls the pharyngeal wall dorsally, to receive a food bolus during swallowing. In a similar manner, during breathing, contraction of the stylopharyngeus muscle pulls the nasopharyngeal wall dorsally thereby supporting the dorsal wall of the nasopharynx and preventing dynamic collapse of this area during inspiration. Evidence of contraction of the stylopharyngeus muscle can be seen as “dimpling (arrows)” of the dorsopharyngeal wall, especially after swallowing (Figure 2a). Experimentally creating stylopharyngeus muscle dysfunction by anesthetizing the glossopharyngeal nerves caused collapse of the dorsal nasopharynx (DP) and inspiratory obstruction in exercising horses (Figure 2b). However, glossopharyngeal anesthesia did not result in dysphagia or any quantifiable swallowing dysfunction in horses.

The nasopharynx is demarcated by the soft palate, which completely divides the pharynx into nasal and oral compartments in the horse. Because the horse is an obligate nasal breather, it is critically important that the soft palate remains ventral to the epiglottis, except during swallowing, to allow unimpeded nasal breathing. The position of the soft palate is determined by the coordinated activity of groups of antagonistic muscles, which include the levator veli palatini, tensor veli palatini, palatinus, and palatopharyngeus muscles. The action of the levator veli palatini muscle to “elevate” the palate can be seen when the gag reflex is stimulated. A “sling (arrow)” forms within the nasopharynx as the nasopharynx contracts into a sphincter (Figure 3). Contraction of the tensor veli palatini muscle “tenses” the palatine aponeurosis and, therefore, the rostral portion of the soft palate, and depresses this portion of the soft palate toward the tongue. Contraction of the tensor veli palatini muscle also aides in opening the pharyn-
The thyrohyoideus is a flat rectangular muscle attached to the lateral surface of the thyroid cartilage lamina that inserts on the caudal part of the thyrohyoid bone. It moves the hyoid bone caudally or the larynx rostrally and dorsally. In studies evaluating he electromyographic activity of some “extrinsic” nasopharyngeal muscles during exercise, Ducharme et al., observed decreased thyrohyoideus muscle activity prior to soft palate displacement in one horse. Investigations by Tsukroff et al. reveal that transection of a combination of the following muscles results in dorsal displacement of the soft palate in horses: thyrohyoideus, omohyoideus, sternohyoideus and hyoepiglotticus muscles. The displacement observed was associated with a more caudal positioning of the basihyoid bone. In subsequent studies thyrohyoideus muscle resection caused intermittent dorsal displacement of the soft palate in exercising horses. As well, thyrohyoideus muscle prosthesis created by placing a suture through the basihyoid bone and the thyroid cartilage returned airway function to normal such that dorsal displacement of the soft palate no longer occurred in any of these horse and the “tie-forward” procedure was created.

Reference


UPPER AIRWAY CAUSES OF POOR PERFORMANCE IN HORSES

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At rest, the average 450 kg horse breathes 12 times per minute with a tidal volume of 5 liters and peak inspiratory flow of 5.09 ± 0.34 L/s, making its resting minute ventilation approximately 60 liter/min. During intense exercise, respiratory frequency increases to 120 breaths per minute, peak inspiratory airflow increases to 75 ± 9.35 L/s, tidal volume increases to 12 – 15 liters, and minute ventilation increases to approximately 1400 – 1800 liters per minute. All the upper airway segments are exposed to varying degrees of negative pressure as the diaphragm contracts during inhalation, creating negative driving pressure for airflow to the lungs. These pressures range from – 1.9 ± 0.2 cm H2O during normal tidal breathing at rest to –38.6 ± 3.9 cm H2O while running at speeds that result in maximal heart rate (HRmax). Inspiratory pressures are more negative in horses with upper airway obstructive disease. The ratio of the peak pressure that occurs to produce a given peak airflow is the airflow impedance and impedance is a measure of how the airflow is opposed by the airway. The determinants of airflow impedance include resistance, which is dependant on the airway geometry, and the elastance and inertance of the tissues. The most important component of airflow impedance is resistance, which is principally determined by the radius of curvature of the airway or its diameter. Resistance to airflow changes with head and neck flexion and also with alterations in lumen diameter, classically caused by collapse of different regions with in the upper airway. In the resting horse, two thirds of the total resistance to airflow resides in the upper airway. As the horse inhales the largest pressure deflections occur at the nares and larynx due to narrowing in these areas. During exercise, upper airway resistance increases to 80% of total airway resistance, because the tissues of the upper airway tend to collapse dynamically as airway pressures become more negative. Positive pressure tends to dilate the upper airway during exhalation and therefore upper airway resistance to airflow during exhalation is only 50% of total expiratory airway resistance. Static or dynamic obstructive airway disease can result in large changes in airway resistance. Throughout the respiratory cycle, the horse relies on neuromuscular mechanisms to dilate and stabilize the airway during intense exercise to expand and stabilize the airway in order to accommodate such high flow rates and pressure changes while minimizing re-
sistance to airflow. Dynamic airway obstruction results when these neuromuscular mechanisms fail, resulting in poor performance and at times, abnormal respiratory noise. Examples of such conditions include idiopathic laryngeal neuropathy or “roaring”, dorsal displacement of the soft palate, nasopharyngeal collapse, and dynamic laryngeal collapse.3-6

Upper airway obstructive diseases can limit athletic performance by decreasing minute ventilation, exacerbating exercise-induced hypoxemia, decreasing maximal oxygen consumption and increasing airway resistance.1-5 Such lesions can be dynamic and only apparent during exercise, or static and evident at rest. Diagnosing and treating these diseases can be challenging because definite etiologies of many obstructive upper airway problems are yet unknown. Indeed, much of the current information that has been generated about these conditions comes from experimentally created disease models.2-4 In addition, measurements are made of horses with experimentally created disease exercising on treadmills at speeds that correlate with maximum heart rate rather than the speeds at which horses race i.e. (12 –13 m/s vs. 16 – 18 m/s).2-4 Videendoscopy during treadmill exercise has been the gold standard for diagnosing upper airway problems. However, the treadmill does not mimic the “racing circumstance” perhaps because of different tack, lack of competition, or different running surface and comfort level with the surface. This may explain the disconnect between evaluation and treatment of upper airway obstructive disease in the laboratory and retrospective performance evaluations of these same conditions. Telemetric endoscopy is the newest method of upper airway evaluation and will likely be the new “gold standard” for diagnosis of dynamic airway disease.7

References

VIRAL DISEASES OF HORSES
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Viral diseases of horses, in particular Equine influenza (EI) and Equine herpesvirus myeloencephalitis (EHM) continue to have a major impact on the global equine industry. The international movement of horses is now almost commonplace, and this can increase the risk of dissemination of viruses throughout the world.

Equine Influenza
- Highly contagious respiratory disease caused by the EI virus (EIV).
- Clinical signs: fever, coughing, tachypnoea, weight loss, +/- more severe clinical signs eg secondary bacterial pneumonia
- Up to 100% morbidity in susceptible populations
- Two subtypes of EIV:
  - A/equine 1 (H7N7): not isolated for more than two decades
  - A/equine 2 (H3N8): associated with all recent outbreaks
- H3N8 evolved into two subfamilies:
  - American lineage
  - European lineage
- Both lineages currently co-circulate in Europe and the USA

Equine Herpesvirus
- Two predominant types: EHV-1 and EHV-4
- Most horses infected around the time of weaning
- Latent infection established in lymph nodes, circulat
- Ubiquitous throughout the global equine population
CHAPTER 6

Scientific Proceedings: Equine Programme

Abstracts European Veterinary Conference Voorjaarsdagen 2009

PROCEEDINGS OF THE EUROPEAN VETERINARY CONFERENCE voorjaarsdagen

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• Up to 80% of adult horses likely to be latently infected
• Reactivation of latent virus can follow transport, handling, re-housing and weaning
• Disease syndromes:
  - Upper respiratory disease
  - Abortion
  - Neonatal disease
  - Neurologic disease (EHM)

EHM
• Primarily caused by EHV-1
• Recent increase in the frequency of reported EHM outbreaks in the UK, Europe and the USA
• Majority of EHV isolates associated with EHM caused by a genetic variant resulting from a single point mutation of the DNA polymerase gene (involved in viral replication)
• Viraemia after infection (new infection or reactivation of latent infection)
• Virus infects endothelial cells within CNS – vasculitis, thrombosis and ischaemic degeneration
• Clinical signs - hind limb paresis/paralysis, recumbency, urinary and faecal retention
• Remain contagious - isolate

The Impact of viral infections
Whilst time lost in training, as well as the expenses associated with diagnosis and treatment are relevant to the individual horse, the real impact of EI and EHM result from their contagious nature and subsequent need for quarantine restrictions in an outbreak situation. The recent outbreak of EI in Australia has highlighted the effect a viral disease outbreak can have on a previously naïve equine population. During the outbreak, approximately 100,000 horses were infected, and severe restrictions were placed on the movement of horses within the country, effectively decimating the 2007 breeding season and resulting in the cancellation of countless equine competitions. The cost to the equine industry in Australia has been estimated at over $AU300million. Many outbreaks of EI occur as a result of the international movement of horses for breeding, competition or sale. In the past 20 or so years six countries that had not previously been exposed to the virus suffered major outbreaks—South Africa, India, Hong Kong, Dubai, the Philippines and, most recently, Australia. Each of the outbreaks was associated with the importation by air of subclinically infected horses and inadequate post-arrival quarantine procedures. Like EI outbreaks, EHM infections are often associated with recent transport of index cases. The outbreak of EHM reported in Florida in 2006/7 was traced to horses imported from Germany. The mortality associated with EHM is often high, and recovered horses may also be left with long term deficits.

Minimizing the impact
Minimizing the impact of viral diseases involves a combination of enhancing immunity through vaccination, as well as implementing good management practices.

Vaccination
Vaccination against EI and EHV can play an important role in control of these diseases, however there are no vaccines which will provide 100% protection.

EI
• Three types: inactivated, modified live and recombinant vaccines
• Immune response to vaccination different to response induced by natural infection
• Inactivated vaccines
  - Immunity dependent on high levels of circulating antibody
  - Immunity generally short-lived, in comparison to the long term immunity afforded by natural infection
• Modified live intranasal vaccine
  - Longer lasting immunity compared to traditional vaccines
  - Induces immunoglobulin profile similar to natural infection
• Live recombinant vaccine derived from a canarypox virus vector and expressing haemagglutinin genes of EIV has also been shown to provide protection from clinical disease in experimentally challenged ponies, inducing both a humoral and cellular immune response
• Vaccines need to be updated to account for antigenic drift
• OIE recommendations state that influenza vaccines should contain both H3N8 lineages (ie European and American) of the virus

EHV
• No vaccines provide complete protection against EHV
• Available vaccines:
  - Provide some protection against respiratory disease
  - Help minimize the spread of infection by decreasing nasal shedding of the virus after infection
  - Minimal effect on the duration of viraemia
• No vaccines licensed for the prevention of EHM

Quarantine
International trade is considered the single most important factor in the global spread of equine infectious disease. Imported animals can be incubating a disease, can be subclinically infected, or may be asymptomatic carriers. Quarantine protocols, involving pre- and post-
import isolation, examination and laboratory testing are implemented in an attempt to identify potential viral infections. A breakdown in quarantine procedures, such as occurred in Australia, can have devastating consequences. Despite adherence to all protocols, especially for viruses which can exist in a latent state, it is possible, indeed probable, that outbreaks of viral infections will continue to occur. The impact of these outbreaks can then be minimized by establishing protocols aimed at minimizing subsequent spread of the virus. (For example, the Voluntary Code of Practice for EHV in the UK)

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OVERTRAINING AND OVERREACHING IN HUMAN ATHLETES: AN OVERVIEW

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From an operational standpoint, overtraining can be defined as the total (training plus non-training) amount of stress (allostatic load) surpasses the regeneration processes. Consequently regeneration is hampered and athletic performance is decreased. Overtraining appears to be a severe condition that involves a long recovery period (up to 6 months). The predecessor of the overtraining syndrome, overreach or functional overtraining, is a less severe condition, where the athletes are able to recover within 2-6 weeks. Overtraining key findings are performance incompetence, alteration in mood state, persistent high fatigue rates, depressed reproductive function, alterations in neuro-endocrine function, and alterations in immune function. Traditionally, dissociation is made between sympathetic and para-sympathetic overtraining, however, the symptoms are far from unambiguous among the published scientific research papers. This is not unexpected, since there are only a few prospective studies available. The underlying mechanisms of an imbalance between allostatic load and regeneration processes can be found on the level of the overloaded target organs, including changes in the brain involving for example the neuroendocrine system, decrease in sympathetic activity, decreased beta-adrenoceptor density, decreased excitability of muscle fibres. In this review I will show results of the most recent prospective studies involving human athletes after a period of very intensive training.

THE PHYSIOLOGY OF EQUINE SPORT

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Horse sport continues to grow on a worldwide basis. In 2008 there were just over 2400 competitions held under Federation Equestre Internationale (FEI) rules; a four fold increase in the past 10 years. The highest number of competitions is for show-jumping (~900 per annum), followed by endurance and eventing which have similar numbers of competitions annually (~500 per annum). An understanding of the metabolic and physical demands of sport and how physiology adapts to exercise demands can help us make judgements on the health and welfare impact of sport, it may provide insights into the causes of injury, allow us to set appropriate levels of competitive demand and to manage competitions in difficult situations, such as the 2008 Beijing Olympic Games.

There are three special issues when considering the horse during exercise. Firstly, apart from the racing camel, none of the other athletic species carry a human whilst performing their athletic feats. In most cases the performing horses is required to carry around 10-20% of its own bodyweight in the form of a rider and associated tack. Secondly, the horse is the most athletic of the larger animals. And although the horse is not the fastest in absolute terms across the animal Kingdom as a whole, within the main athletic species the horse is faster than man, camel and dog. Finally, the horse is incredibly versatile. Here is a species which can run fast,
run for a long time, jump, perform intricate movements, anticipate riders’ movements and react explosively.

A number of different aspects of the horses’ physiology in relation to exercise are apparent. The horse by nature is a prey animal rather than a predator. It has an extremely well developed “fight or flight response”. This response is geared to explosive effort to put as much distance between the horse and the predator as quickly possible. However, this type of maximum effort is clearly time limited, and thus we can consider the horse to essentially be a sprinter. This is born out when we examine the muscles of foals, which are well adapted for fast explosive bouts of exercise with large fibres, few capillaries per fibre and a high glycolytic (anaerobic) and relatively low aerobic enzyme content. Perhaps therefore somewhat surprisingly, most training studies on horses produce a shift away from anaerobic to improved aerobic capacity.

Catecholamines play a major role on the physiology of the horse, particularly during exercise. In particular, as well as the influencing the level of cardiac output and its distribution towards locomotory muscle and away from “non-essential” (at least with respect to short-term exercise) vascular beds, adrenaline also has important roles in thermoregulation and energy metabolism. High levels of circulating adrenaline contribute to control of sweating and also to a shift towards glycogen metabolism. Being large is an advantage if you live in a cold climate. It is not necessarily beneficial if you are exercising. This is especially true when it comes to heat production. Athletic horses have a very high proportion of their bodyweight made up by muscle – perhaps over 50%. For every unit of energy that generates movement, 4 times as much is released as heat. Heat is primarily lost at the body surface. Thus although a 500kg horse is around 6 times heavier than an 80kg man, it will only have around 2.5 times as much surface area. This means a horse has to squeeze out more heat through each metre-squared of skin than a person would. The horse relies heavily on sweating to dissipate heat and no other animal can sweat at a higher rate than the horse – up to a formidable 50 ml of sweat through each metre-squared of skin surface each minute and around 3 times higher than maximum sweating rates in man.

It is not uncommon to measure rectal temperatures of horses after eventing, polo or racing as high as 42°C, with blood temperatures of 45°C and muscle temperatures as high as 48°C. These are temperatures that generally cannot be tolerated by humans or dogs. Thus, horses also possess a high degree of thermotolerance. There has also been speculation that horses have some capacity for selective brain cooling, which is the ability to keep the brain at a lower temperature than would normally be predicted from blood temperature entering an organ’s vascular bed.

The role of adrenaline in redistributing blood flow during exercise is also highly refined in the horse. The majority of species, especially larger animals, balance the demand for increased muscle blood flow with the demands of thermoregulation. The horse is somewhat unusual in that at the intensity of exercise where significant heat has already been stored and body temperature is already elevated, the horse is able to reduce skin blood flow and redirect more blood flow to the working muscles. This accelerates the rate of heat storage but allows greater and more prolonged intense muscular effort.

The horse is by no means “perfectly designed” for exercise and there are a number of peculiarities that can limit performance. The first disadvantage the horse has is being a herbivore. The horses’ GI tract comprises around 12% of its bodyweight, due to a combination of the size of the GI tract and its water content. Contrast this with the weight of the GI tract of a person at around 6% of bodyweight. Another peculiarity of the horse concerns the respiratory system. The horse is an obligate nasal breather and at canter and gallop healthy horses take one breath in perfect time with each stride. The horse adopts this strategy due to the fact that it has a large and stiff rib-cage and coupling the breathing with the stride is a mechanism to save energy. Finally, the respiratory system of the horse does not respond to training. The outcome is that the respiratory system is a weak point in the chain in getting oxygen from the atmosphere down to the mitochondria in the working muscles.

The physiological responses of horses in a variety of different equestrian disciplines have been described in the literature and these will be reviewed in the context of what we can learn from a clearer understanding of the physiology of exercise in sport.
MANAGEMENT, TRAINING AND OTHER LESS COMMON CAUSES OF POOR AND LOSS OF PERFORMANCE IN SPORTS HORSES

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In the absence of obvious clinical signs of injury or disease, the investigation of poor performance or loss of performance in the performance horse can be challenging. Any investigation would of course involve taking an accurate history and a detailed clinical examination of the animal concerned followed by appropriate diagnostic investigation. However, when this fails what other steps can a veterinary surgeon take in order to try and identify the underlying problem or problems?

The first step in a more detailed investigation is to try and ascertain if the situation involves a true loss of performance or poor performance. In the case of poor performance, a horse is reported by the owner, trainer or rider to be performing below their expectations, based primarily on breeding and confirmation. Previously less common but now beginning to be encountered more frequently, the classification of poor performance may also be made based on objective measurements of gait, cardiac or locomotory muscle characteristics or measurements of heart rate and blood lactate during standardised exercise tests. Unfortunately, in many cases poor performance due to lack of ability is the correct but an unpopular diagnosis. Studies of the heritability of performance in horses in a variety of disciplines most commonly show a value of around 0.1 to 0.2. If the performance of the offspring could be perfectly predicted from that of its parents then the value would be 1, and 0 if there was no relationship. In one recent study of sport horses in Germany a heritability value of 0.61 was reported. This appears to be the highest value reported to date.

For a horse to be classified with “loss of performance”, it should previously have been performing more successfully than at the time of referral or investigation. For a sport horse with several years competition records this may be relatively straightforward to confirm. However, in the case of horses that have only been competing for less than a year, it is possible that the previous high performance is not truly representative. This may especially be the case if the horse in question had been competing against poor class horses.

Another important step to take is to try and ascertain if the problem affects only the horse presented or whether other horses may also be affected, but simply not be presented as they appear healthy. Furthermore, horses not being competed are unlikely to be presented for loss of performance.

Routine haematology and biochemistry can be extremely helpful in these investigations, but even more so when good baseline values are available when the horse was considered healthy and performing well. Good practice for obtaining reliable results from blood samples is to collect pre-feed and pre-exercise, usually early in the morning. In addition, care should also be taken to try and standardise for the previous days exercise as this can result in differences in a number of measurements, particularly CK/AST.

Where the immediate cause of loss of performance in multiple horses is not apparent, if time permits a visit to the yard to watch normal husbandry, training and to visit the feed room can be valuable. Points that the owner, trainer or rider do not consider of importance and therefore worth relating may turn out to be of significance.

It is important to attempt to ascertain what recent changes there may have been in the yard. The introduction of new horses for example may point to infectious disease. New staff may mean that there have been changes in management. The delivery of new batches of hay or feed may point to a problem of a nutritional origin, for example contamination of feed with mycotoxins.

Supplement use can also be another area worth investigating. The range of supplements now available is immense and whilst many are innocuous, there is the potential for trainers to use ones which can have negative effects on health. Sometimes the supplements are fed in excess or it may be a combination of different supplements that can produce specific problems. Therefore it is always worth asking to see the supplements a horse is being fed.

In some cases problems can arise from failure in translation of instructions from the owner, trainer, rider to staff looking after the horses on a daily basis. Thus, what the trainer tells you they believe is being fed may be significantly different from what is actually being fed by staff.
MUSCLES AND POOR PERFORMANCE
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Muscular disorders make up an important subset of diseases that cause poor performance. Commonest are the exertional myopathies which are often subclinical, and easily overlooked. A variety of acquired factors underlying the development of exertional rhabdomyolysis are proposed. Unfortunately, for some causes, the evidence remains speculative. Current and historical acquired causes include overexertion or exhaustion, oxidative injury, eccentric contraction, hormonal influences and electrolyte abnormalities.

We currently recognise two common forms of exercise-related myopathy in horses with underlying genetic causes. These include a condition examined extensively in a small group of Thoroughbreds in the USA that has been termed 'recurrent exertional rhabdomyolysis' (RER)\(^1\) and another condition, termed 'polysaccharide storage myopathy' (PSSM or EPSM).\(^2\) They have clinical similarities and are managed similarly, though they also have key differences and breed susceptibilities.\(^1\) The word ‘recurrent’ in Thoroughbreds with exercise-related myopathy has been used variably to describe certain Thoroughbreds with documented abnormalities in muscle calcium regulation,\(^1\) a wider group of Thoroughbreds with an apparent inherited form\(^3\) and all Thoroughbreds with the syndrome.

Estimates of the prevalence of exercise-associated rhabdomyolysis in Thoroughbreds suggests that 5-7% are affected.\(^4,5\) Pedigree analysis in the USA supports autosomal dominant inheritance.\(^5,6\) The abnormality in muscle calcium regulation identified in some Thoroughbreds shares certain experimental similarities with malignant hyperthermia (MH). In particular, muscle from affected horses and other species with MH is hypersensitive to agents (such as caffeine and halothane) that stimulate release of calcium from the muscle calcium store (sarcoplasmic reticulum) through a calcium release channel known as the ryanodine receptor (Ryr1).\(^1,9\) However, though MH has been reported in some horses following halothane anaesthesia, and indeed, though an Ryr1 receptor mutation has been identified in MH-susceptible Quarter horses\(^10\), Thoroughbreds with abnormal calcium regulation do not share the same mutation and there is evidence suggesting that the Ryr1 receptor is not mutated in Thoroughbred RER.\(^11\) Despite this, the basic research similarities between RER and MH do still suggest the possibility of involvement of another protein or proteins that regulate intracellular calcium concentration in muscle. Indeed mutations in other proteins are known to cause or are implicated in human MH. Novel research methods may prove useful in the future investigation of the disease.\(^12\)

Polysaccharide storage myopathy can be definitively diagnosed by muscle biopsy. Pathognomonic changes include diastase-resistant inclusions detected by Periodic Acid Schiff staining. This disease was first reported in detail in Quarter Horses with exertional rhabdomyolysis in the USA\(^2\) and has since been reported in a
References

A PITFALL........................A POTENTIAL HAZARD OR DANGER THAT IS EASILY ENCOUNTERED BUT NOT ALWAYS IMMEDIATELY OBVIOUS

G N Potts, BVetMed, MRCVS

The pitfalls in equine practice is a boundless subject that could alone provide enough material for a whole conference. One would like to think that with advancing years and a wealth of experience, the pitfalls might all be avoided but sadly they still exist and all too frequently we find ourselves dealing with situations that go wrong despite our best intentions and efforts.

I have been in equine practice as a general practitioner for 25 years but still have vivid memories of my early days when, unfortunately I was oblivious to many of the pitfalls ahead and consequently encountered most of them at some point. In retrospect, perhaps ignorance was bliss but in the modern world there is little forgiveness when things go wrong and thankfully I believe today’s graduates are better prepared for the problems that lie ahead.

This presentation will hopefully contain something for equine practitioners of all ages, with tips for new graduates on how to avoid problems to discussions of more advanced pitfalls that we might encounter even when we think we have ‘seen it all’!

I would like to think that I am not too proud to deny ownership of some of my less auspicious moments in practice and I hope the audience will emphasise or at the very least sympathise!
A REVIEW OF SELECTED NEUROLOGICAL DISEASES AFFECTING HORSES
NEUROLOGY IS NOT A EUPHEMISM FOR NECROPSY

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Introduction
Disorders of the nervous system are serious and often debilitating problems affecting horses. Our knowledge of these disorders as well as how to diagnose and treat the conditions remains incomplete. My goals for equine practitioners are to be able to perform a neurological examination, to arrive at a neuroanatomic localization and to use diagnostic tests to help determine the cause of the clinical signs in the affected horse. It is important to recognize that treating horses with neurological conditions, even serious ones, can often result in successful outcomes, including useful athletic lives.

Possible causes of neurological diseases in horses include developmental problems, trauma and infectious diseases. Conditions such as cervical vertebral stenotic myelopathy, equine protozoal myeloencephalitis, Equine Herpesvirus myeloencephalopathy, rabies, west nile virus and botulism are some examples of neurological disorders affecting horses.

Cervical Vertebral Stenotic Myelopathy
Cervical vertebral malformation is often referred to by many names including cervical vertebral stenotic myelopathy, spinal ataxia, cervical stenotic myelopathy and wobbler’s syndrome. Cervical vertebral malformation is one of the most common causes of neurologic disease in horses worldwide. The most important feature of this condition is a narrowed or stenotic vertebral canal as a result of abnormal vertebral development resulting in compression of the spinal cord. Typically the narrowing is identified between the 3rd and 7th cervical vertebra, although it can occur at any site from C1 to T2. The disorder appears to be a multifactorial disease although the underlying cause is not fully known. At this time the problem is thought to be a developmental disorder that begins very early in life, perhaps even in utero. Many factors have been suspected to play a role in the development of this condition in the appendicular as well as the axial skeleton of the horse. Included among these factors are genetic predisposition, endocrine dysfunction, dietary or nutritional imbalances, biomechanical stress and rate of growth. The underlying abnormalities associated with osteochondrosis result in abnormal bone metabolism as well as damage to cartilage.

Many investigators have suggested osteochondrosis of the cervical vertebrae as contributing to or as the sole underlying problem in this disease process. Osteochondrosis is described as a disturbance in endochondral ossification in rapidly-growing animal species and humans. Many factors have been suspected to play a role in the development of this condition in the appendicular as well as the axial skeleton of the horse. Included among these factors are genetic predisposition, endocrine dysfunction, dietary or nutritional imbalances, biomechanical stress and rate of growth. The underlying abnormalities associated with osteochondrosis result in abnormal bone metabolism as well as damage to cartilage.

One dietary feature of interest is the effect of copper supplementation on the prevalence of cartilage lesions in foals. Several investigations compared farms with apparent high incidence of developmental orthopedic disease in foals to farms that did not have similar problems. From this survey of farms in central Kentucky, a difference in the dietary levels of copper was identified. This information was used as the basis of several prospective research projects on the role of dietary copper on cartilage lesions in foals. In this experiment, 21 mares were divided into two groups and fed rations containing 13 parts per million (ppm) of copper (control) or 32 ppm of copper (supplemented) in the diet during the last three to six months of gestation and the first three months of lactation. The foals were fed pelleted concentrate containing 15 ppm (control) or 55 ppm (supplemented) copper. At 90 days of life, five control and five supplemented foals were destroyed and at 180 days six control and five supplemented foals were destroyed. In the post-mortem examinations of the foals destroyed at 90 days more than twice as many osteochondrosis lesions and more than four times as many articular lesions of osteophyte formation or thinning were identified in the copper control versus the copper supplemented group. Interestingly, in the foals euthanized at 90 days the predominant cause for the difference was the presence of many lesions in one copper control foal.

In the foals euthanized at 180 days of life, there were seven times more articular lesions of osteophyte formation or thinning, twice as many osteochondrosis lesions in the physis and more than five times as many osteo-
chondrosis lesions in the articular-epiphyseal complex in the six foals on the low copper diet. In many of the foals with osteochondrosis of the articular–epiphyseal complex, there was separation of the thickened cartilage from the subchondral bone, including nine foals showing subchondral fibrosis. The work from this study was very significant because it resulted in further investigation regarding the role of nutrition in developmental problems of cartilage and bone in foals. The work was examined and critiqued by many veterinarians, nutritionists and biochemists and led to changes in the way horses are fed today.

Following the nutritional investigations of Knight et al, application of this information to the investigation of examples of developmental bone disease was performed. The first study at Ohio State examined the frequency and severity of osteochondrosis in horses with cervical vertebral stenotic myelopathy. The most significant findings of the work by Stewart in 1991 were the identification of a generalized narrowing of the vertebral canal of horses with cervical vertebral stenotic myelopathy compared to clinically normal horses. This finding corroborated the work of several other investigators. The finding of a smaller minimum sagittal diameter throughout the vertebral canal rather than only at the site of compression indicates that this problem is related to a general failure of vertebral canal development, and not simply a focal malformation due to arthritis or some biomechanical disorder. It follows that in any horse with a narrow vertebral canal, development of arthritic changes of the articular processes or abnormalities of the growth plates of the vertebral bodies may lead to further stenosis and thereby contribute to the onset of clinical signs. Subsequent to the work of Mayhew and Stewart, many investigators have sought to understand the underlying pathology associated with this.

Stewart found that both osteochondrosis and non-osteochondrosis lesions were more severe at sites of compression than at sites of non-compression, although compression was also identified at sites with no lesions of the articular processes. In this study an increased frequency and severity of osteochondrosis was noted in the appendicular skeleton of horses affected with cervical vertebral stenotic myelopathy. An increased severity of osteochondrosis of the cervical vertebrae was noted over the control group, yet the incidence of non-osteochondrosis lesions was equal between control and affected groups. These findings support the notion that the underlying problem in horses affected with cervical vertebral stenotic myelopathy is a failure of normal bone and cartilage development and maturation.

Horses with CVM will present with symmetric ataxia, paresis, and spasticity that is delegated almost entirely to the hindlimbs. Toe dragging, stumbling and circumduction of the hindlimbs will also be apparent. These signs tend to be exaggerated when the horse moves with its head elevated. The horse may also stand base wide when at rest. Clinical signs in horses are often observed in the first year of life and may sometimes be identified as early as before three months of age. The onset of clinical signs often appears quite acute, despite the fact that the signs may have been present and slowly progressive for several months. In some horses the onset may follow a traumatic episode or a day of very hard playing while turned out. CVM manifests itself as both a loss of awareness of the limbs along with weakness and ataxia while walking.

The most valuable test to diagnose cervical vertebral malformation is to identify stenosis of the vertebral canal. Lateral views of the cervical vertebral column are often an informative ancillary diagnostic test when searching for a compressive site or lesion. Lateral views can be performed without the need for anesthetics, unless one has a very unruly animal.

The horse should be positioned with the head and neck in its natural position with the cassette on one side and the x-ray tube centered on the cassette on the other side of the horse. For recumbent horses, pads and wedges should be placed under the animal’s neck and head in order for the cervical spine to be parallel to the ground. Placing the cassettes under the recumbent horse’s neck may be difficult unless a bucky tray is used. A three-image series is generally adequate to image the entire cervical spine. The most cranial radiograph should be made with the tube centered on C4–C5, centered on C4 for the middle radiograph and on C6 for the most caudal radiograph. These views will provide some overlapping. Lead markers on the neck can be useful to assist the viewer with the exact location of the radiograph. With experience the viewer will become familiar with the anatomy and will readily identify the location based on anatomic features such as the size of dorsal spine, the size and shape of the facets or the length of the vertebral body.

When viewing the radiographic images, changes in vertebral body appearances should be closely examined. The normal equine vertebral canal in the cervical region should have a gentle curve. The spinal canal should be smooth with only the slightest changes at each intervertebral space. Normal articular processes (facets) should have joint spaces with smooth, rounded articular surfaces. One should look for irregular joint spaces, a lack of joint space, irregular surfaces and decreased...
widths of the vertebral canal when searching for steno-
sis or other abnormalities of the vertebral canal. Flaring
of the caudal epiphyses, dorsal laminar extension, sub-
luxation, degenerative joint disease and abnormal os-
sification of the articular processes are all characteristic
of CVM. However, identification of these findings alone
may not be adequate to diagnose the exact location of
spinal cord compression and a myelogram may be
required. However, the most important factor in the di-
agnosis of cervical vertebral stenotic myelopathy is the
identification of cervical vertebral canal stenosis. Ob-
jective testing is much more dependable than qualita-
tive evaluation and the sagittal ratio method should be
utilized. Myelography is required to confirm the exact
site for surgical intervention in horses diagnosed with
cervical vertebral stenotic myelopathy.

Collection of CSF can sometimes be a useful diagnostic
aid to determine the cause of ataxia in horses. Cerebro-
spinal fluid analysis is particularly helpful if the disease
is suspected to be either inflammatory or infectious.
However, it is important to remember that identifica-
tion of normal cerebrospinal fluid does not necessar-
ily negate any possibility of neurological disease, and
that it should be used as supportive evidence along
with other findings and the history. CSF is produced at
a constant rate that is independent of the absorption
rate by the venous sinuses. Since the production rate is
constant and independent of intracranial pressure, the
rate of absorption is the primary regulator of intracra-
nial pressure.

Myelography is useful to confirm the diagnoses of
cervical stenotic myelopathy and to determine the lo-
cation of the lesion(s). This procedure is especially im-
portant when surgical intervention is anticipated. In my
opinion, the performance of a myelogram on a horse
is a safe procedure that can be utilized on even the
most severely ataxic horses. Although this procedure
is typically performed with the horse under general
anesthesia, there have been at least two descriptions
of the procedure in awake, sedated horses. Although
some horses may transiently worsen following a my-
elogram due to manipulation of the cervical vertebral
column while performing the study, in my experience
(> 1,500 myelograms) it is rare for a horse to be unable
to recover following this procedure. Whenever possible
it is best to avoid performing this procedure on horses
with inflammatory or infectious causes of neurologic
disease and if a severe compression is suspected based
on the findings observed on the standing radiographs,
then minimal manipulation of the vertebral column
and spinal cord are indicated. Radiographs should be
taken of the cervical vertebrae in the normal, flexed
and extended positions with care not to overflex or overex-
tend, as this can cause further damage to the cord. The
x-ray tube should be positioned centrally on the verte-
bra as described earlier.

Interpretation of cervical myelography is the subject of
much debate and several publications have appeared
during the past 30 years. To begin, it is the opinion of
this author that no single criteria can be used to deter-
mine whether a horse is a candidate for surgical correc-
tion using a ventral stabilization. Rather it is important
to consider all information that is known including the
history, the severity of the clinical signs, the findings on
the standing radiographs (perhaps most important af-
after the neuroanatomic localization) and the results of
ancillary testing for other diseases such as EPM, EHM or
meningitis.

Interpretation of the height of the contrast column in
cervical myelography has been considered the best
antemortem test for diagnosing extradural spinal cord
compression in horses suspected to suffer from cervical
vertebral stenotic myelopathy. In our hospital, interpr-
etation is generally based on reduction of the dorsal con-
trast column compared the space ahead or behind this
site, with a 50% attenuation considered abnormal. As
means of allowing independent evaluation, the myelo-
gram is generally performed by medicine clinicians and
interpreted by those completing the study. The study
is then examined independently by the responsible
surgeon. Following independent review, those cli-
nicians that will perform the surgical correction as a team
then review and interpret the films together, prior to
contacting the owner and/or agent. This helps to insure
that we have discussed all aspect of the clinical signs as
well as the diagnostic testing, prior to deciding whether
surgical intervention is to be recommended. If the my-
elogram is especially difficult to interpret, for example if
the compression is very mild or there is suspicion that
the spinal cord is swollen, then final recommendations
will be reserved until additional results are obtained (eg
western blot for EPM).

Interpretation of the myelogram has been based at
least in part on the subjective opinion of the individuals
performing the studies. However, a recent review of the
evaluation criteria used for interpretation of myelogra-
phy indicated that the use of a reduction in height of
the myelographic contrast column showed a low sen-
sitivity and moderate specificity for diagnosing spinal
cord compression due to CVM. These authors indicated
that interpretation of myelography should be done
with caution. This advice is important and it is useful to
make a decision for surgical intervention with the fol-
lowing questions: could the clinical signs in this horse
be explained by a compressive lesion at that site and
are there other test results that indicate the presence of another disease process that could explain the clinical signs in this horse? The individuals performing the study read the myelogram, followed by an independent reading by the surgeon and finally a combined review of the myelogram. At this time we use all published guidelines and criteria available from the literature and have chosen to err on the side of early surgery when a site only marginally meets the published criteria for surgical correction. This technique provides the best for the horse and the owner in our opinion and because the horse is most important, we feel comfortable with this decision tree. In some cases the myelogram will be referred to two or more surgeons or radiologists for additional opinions.

The aim of medical management of horses with cervical vertebral stenotic myelopathy is to reduce swelling and inflammation of the spinal cord. When this problem is diagnosed in weanlings or horses less than 12 months of age then a conservative approach directed at management changes can be instituted. These changes include a reduced level of exercise, careful attention to the diet including avoidance of excess weight gain along with close observation for balance of macro minerals (calcium and phosphorous) as well as trace minerals such as copper, zinc and manganese. We also often supplement young horses with vitamin E and selenium, especially when raised in a selenium deficient area of the country. In mature horses with compressive lesions that are not candidates for surgical intervention, use of anti-inflammatory agents such as non-steroidal medications as well as short term use of corticosteroids, combined with exercise limitations, can sometimes result in a successful return to athletic use. In horses with arthritis of the articular process joints, intraarticular use of corticosteroids combined with medications to promote cartilage healing can be useful.

If surgical correction is desired, the best treatment is ventral interbody fusion. This treatment has been in use since 1979 but continues to be somewhat controversial. Questions surrounding the multiple factors responsible for the development of this condition are a part of the reason for this controversy. The role of genetics in the development of cervical vertebral stenotic myelopathy continues to be one important question. Investigations of the published literature beginning as far back as 1937 have failed to convincingly demonstrate this condition to be inherited. Work by Dimock, published in 1950 in the Journal of Heredity, suggested a recessive nature of the defect although breeding of affected to affected failed to produce an affected horse. This was also the first work to demonstrate the occurrence of cervical vertebral stenosis to be three times more frequent in males over females. A study involving Thoroughbreds in Great Britain provided no evidence that affected TB horses are suffering from a genetically determined disease. Additional concerns about surgical correction of horses with spinal cord compression include safety, as the horses must recover sufficiently to return to some athletic or breeding use; although some owners are content to allow the horse to live out its life as a pasture pet.

In man, it has been shown that 80% of patients with vertebral canal stenosis similar to what is observed in horses improve following a laminectomy and posterior fusion and 80-90% of patient’s damage due to discogenic injury to the cord improve following ventral interbody fusion. Similar studies in dogs with cervical arthropathy affecting the caudal vertebral bodies showed an 89% improvement and in a previous study in horses 77% improved and 46% returned to athletic performance. In a study by Nixon and Stashak comparing use of ventral stabilization to dorsal laminectomy, there was a 56% return to normal in the ventral stabilization (N=27) group with 37% showing only grade 1 deficits. Following dorsal laminectomy (N=30) there was a 57% returned to normal and 27% showed only grade one deficits at one year post-surgery. The technique used to perform ventral stabilization in horses has been described extensively.

Patient selection is very important and many factors are a part of the selection criteria, including severity of clinical signs, the duration of clinical signs, the number of sites affected, the age of the horse, the commitment of the client and cost. In the experience of this author, horses with the best chance for a successful outcome have mild to moderate clinical signs and relatively short time period from diagnosis to surgical correction. However, exceptions do exist and several recent publications describing successful outcomes following surgical correction in horses with three sites of cervical cord compression. While all of these criteria are important, a successful outcome is also dependent on the skill of the surgeon and the commitment of the owner to a reasonably long term period of rehabilitation, often taking as long as one year.

A technique that has been used on some horse with mild signs of spinal ataxia caused by cervical vertebral osteoarthropathy is injection of the articular process joints (often referred to as facets). This procedure is most indicated when the horses are showing signs of neck pain and stiffness and very little or no ataxia. Further, the procedure can be performed in awake sedated horses. Arthrocentesis is performed for patients suffering from osteoarthritis and osteochondrosis of the cer-
vical vertebral articulations. Many of these horses will often present for obscure lameness rather than neurological disease.

Ultrasoundography is utilized throughout the entire procedure, first for location of the facet and then for accurate passage of the needle into the facets. The ultrasound beam is directed parallel the joint space to show the space between the cranial facet of the caudal vertebra and the caudal facet of the cranial vertebra. The angles of entry and the depths for complete insertion of the needle will vary for each horse depending on the placement of the head, the age of the horse, and the size of the lesion. Some veterinarians prefer to use the biopsy guide while others are more comfortable using a free-hand technique. A 6 in. 18 gauge spinal needle can be used for the facets of C3-7, although occasionally a longer needle may be needed for C6-7.

Equine Protozoal Myeloencephalitis

Equine protozoal myeloencephalitis (EPM) is an important neurological disease of horses in the Americas. The disease was first reported in 1970 in 52 horses from Kentucky and Pennsylvania although some literature describing clinical cases dates to the late 1960’s. Within a few years similar clinical findings were identified in horses from other regions. Confirmation that the disease was caused by a Sarcocystis organism was obtained by Simpson and Mayhew in 1980. Based on the organism’s structure, reproductive characteristics of the organism and association with neurons Dubey proposed the name Sarcocystis neurona for this organism.53

The most common agent recognized to cause EPM is Sarcocystis neurona, an apicomplexan protozoan, but clinical signs in some horses are a result of Neospora hughesi, which causes a rare sporadic form of EPM. Less is known about the life cycle of N. hughesi. Most Sarcocystis spp affect a single host while S. neurona has the ability to affect a wide host range, similar to Toxoplasma gondii and Neospora caninum. S. neurona has been shown to have considerable antigenic diversity among its surface antigens (SAG) proteins. Recognition of the major surface antigens has been utilized in the development of diagnostic testing modalities in order to evaluate the equine antibody response.

Equine Herpesvirus 1 Myeloencephalopathy

Equine Herpesvirus 1 (EHV-1), a member of the Alpha-herpesvirus subfamily in the Varicellovirus genus, has a worldwide distribution and results in economic losses as well as various forms of infectious disease including mare abortions, respiratory disease, neonatal deaths and myeloencephalopathy. Infection typically occurs early in life and is followed by the establishment of latent infection. The virus can then be reactivated after a stressful episode resulting in onset of fever, respiratory disease or neurological disease. Many horse, viral and environmental factors are likely to have an impact on the incidence of recrudescence of EHV-1 in horses. Investigations to examine shedding in stressed horses have recently been published and it appears unclear what conditions are stressful enough to result in viral shedding. In young horses, most typically horses less than two years of age, the clinical signs include fever, loss of appetite, serous nasal discharge and loss of training days. With this manifestation the horses are usually out of work for four to seven days and then return to work with little or no apparent long term consequenc- es, though the horse may now be latently infected with EHV-1 virus. When the virus affects pregnant mares abortions can occur, most commonly during the last trimester, though they can occur at any time. The third manifestation is the neurological form of the disease in which many horses on a premise can become infected. Some of the affected horses develop equine herpesvi- rus myeloencephalopathy (EHM) with significant neuro- logical signs of weakness and ataxia, often involving the pelvic limbs but any part of the nervous system may be affected.

Virus transmission is usually from horse to horse by aerosol transmission through close contact, although fomite transmission should also be considered. Virus enters the respiratory epithelial cells and is transported to the regional lymph nodes, usually within one to two days. Once the virus has established itself in the respiratory epithelium it undergoes replication and may be infective for other horses for as long as 14 days. The recently recognized strain associated with EHM appears to have a replicative aggressiveness that results in higher numbers and a longer period of replication, with the consequence of a greater concentration of virus for longer periods on nasal mucosal surfaces thus prolonging the period of infectivity. The virus rapidly enters peripheral blood mononuclear cells (lymphocytes and monocytes) and circulates in the blood in what is described as a cell associated viremia that can persist for up to 21 days. Once the virus is within the cell it appears to be able to circulate without destruction even in the face of high circulating antibody titers. In this location the virus can disseminate to other tissues including the CNS. It is thought that the mononuclear cells contact the endothelial cell lining of the central nervous system and the virus enters into the endothelial cells through this close contact. The damage that occurs as a result of this infection with EHV-1 is vasculitis followed by thrombosis and ischemia in the nervous tissue without direct viral infection, thus resulting in a myelopathy rather than myelitis. This cell associated viremia is an
important feature in the development of abortions as well as myeloencephalopathy and is an important target for stopping or blocking outbreaks of these problems in the horse.

The first definitive association between EHV-1 and myeloencephalopathy was made following isolation of the virus from the brain and spinal cord of a horse in Norway in 1966. Since that time, examples of similar cases have been demonstrated to have a worldwide distribution. In most instances the myeloencephalopathy occurs as an outbreak of cases although sporadic disease can be observed. In most situations EHM occurs as an outbreak often during the winter or spring. The outbreaks are typically associated with congregation of horses in a somewhat closed environment with common air space as well as common management system.

There is often a history of recent introduction of new horses or a return home of horses that have been on the travel circuit. Horses that are most often involved are greater than three years of age and engaged in frequent travel for racing, showing or sales activity. The initial signs will usually be fever in one or more horses followed in seven to ten days by horses showing signs of neurological disease, such as poor tail tone, urinary incontinence and pelvic limb weakness.

During the past seven years several outbreaks of the neurologic manifestation of EHV-1 have been observed and/or described in the literature from around the world. In the United States, the USDA: APHIS with the Centers for Epidemiology and Animal Health determined in 2007 that this viral problem met the criteria for an emerging infectious disease. Careful examination of the outbreaks and particularly of the virus isolated from horses infected in these outbreaks indicated an apparent increase in virulence of the virus along with increased morbidity and increased mortality than previously described. Outbreaks of neurological disease associated with EHM have been described. Beginning in 2000 there have been an increase in the number of epizootics of EHV-1 myeloencephalopathy in which the number of horses affected, the severity of the clinical signs and the rapidity with which the virus spreads through a group of horses appears dramatic when compared to previously described outbreaks. A recent report from central Kentucky demonstrated a number of horses harboring a particularly neuropathogenic strain of this virus.

The study of outbreaks from around the world along with development of newer more sophisticated diagnostic testing has helped us learn a great deal about this infection. Still many questions remain unanswered about the virus, the pathogenesis of EHM, the mechanism of infection of endothelial cells, the recognition of a single nucleotide polymorphism in the DNA polymerase gene and how the role of host and environmental factors affect the risk of development of this disease syndrome. Recognition of apparent lateral transmission of an infectious disease resulting in signs of ataxia suggestive of EHM should signal any veterinary hospital or stable to immediately initiate an intensive investigation to determine the cause for the clinical signs, as well as any potential relationship between animals presented for this disease and new cases identified among patients in ones hospital. Following the recognition of apparent lateral transmission of an infectious disease it is very important to protect horses remaining in one's hospital as well as new patients presenting to the hospital. To do so may require a period of closure of the hospital in order to accomplish thorough cleaning and disinfection. This should be followed by segregation of all remaining horses into a separate portion of the hospital with no new horses allowed to be admitted for a period of 21 to 28 days. Whenever there is adequate space to allow separation of the hospital stalls as well as all hospital staff, the hospital may be divided and, following appropriate disinfection, at least one portion of the hospital may be re-opened for use. Prior to re-opening, the hospital designated as clean should be closed, steam cleaned, disinfected and allowed to dry for a period of five to seven days. Once re-opened, the horses, grooms, veterinarians, veterinary technicians and all other personnel and owners should remain separated from those working around horses that were in the hospital during the time of the outbreak. No cross traffic of personnel, horses or equipment should be allowed.

Following these guidelines an epizootic can usually be quickly brought under control and most importantly no additional horses become infected. On a more general level, the outbreaks have stimulated further investigation regarding the virus and risk factors associated with the development of clinical signs in naive horses, better diagnostic testing for use on horses involved in a suspected outbreak of EHM and development of biosecurity strategies for prevention and control of outbreaks. Additional research is being focused on development of new vaccines and the study of medications useful to treat affected horses. Finally, several workshops on this virus have been held and a consensus statement is being developed by the ACVIM regarding this disease.

Information regarding the virus has been significantly impacted by the notion that strains of EHV-1 with variation in pathogenic capacity exist. This hypothesis was investigated by Nugent et al who recognized that variation of a single amino acid of the DNA polymerase was strongly associated with differences in clinical signs.
(neurological versus non-neurological disease). The recognition of this amino acid variation in a highly conserved region of the herpes virus DNA polymerase increased the likelihood of this having an important function in the pathogenicity of the virus. Investigation regarding the virus is ongoing and has generated some controversy amongst major researchers in the field, in an effort to determine more precisely when the mutation occurred. Some of what has been learned about this strain of EHV-1 is that the strain has enhanced replicative capacity resulting in greater likelihood of neurologic morbidity and mortality. This increased replication of the mutant strain results in increased levels of viral particles in the nasal mucosa of horses, which then increases the efficiency of spread from horse to horse. Recently, recognition of this virus in certain populations of horses has been demonstrated. The finding of this strain of the virus as latent EHV-1 DNA in the submandibular lymph nodes of Thoroughbred broodmares in central Kentucky provides evidence that the virus has a presence in certain equine populations. These findings suggest that attention should be focused on development of vaccines capable of providing protection against this strain of the virus and that horses with this strain of the virus do not need to be treated differently than other EHV-1 survivors.

The importance of this virus to the equine industry is in part a result of the economic impact from either multiple abortions, multiple cases of equine respiratory disease and outbreaks of myeloencephalopathy. The respiratory and abortion outbreaks have resulted in a detailed tracking of this virus worldwide; through this surveillance, variations in the composition of the nucleic acid have been detected. The Alphaherpesviruses have a wide host range; short reproductive cycle and most importantly have the capacity to establish latent infections. Use of genetic and biologic markers has helped to track the viruses and explain the changes in virulence of the virus and may partially explain the inability to predict when and why cases of neurological disease occur. In addition the protective immune response following either natural infection or vaccination is quite short.

The criteria which have been utilized for the diagnosis of the neurologic form have been described, but in general a prospective diagnosis can be made based on clinical signs alone. To confirm the diagnosis, a history of antecedent or concurrent upper respiratory tract disease, xanthochromic cerebrospinal fluid (containing increased quantity of protein), or a 3 to 4 fold rise in anti-EHV-1 neutralizing antibody titers are useful. In addition, histological evaluation of nervous tissue from an affected horse will show the classic vasculitis changes and in some cases virus isolation from theuffy coat, nasal swabs or post-mortem samples are helpful.

When an outbreak of EHM is suspected it is very important to have available rapid testing that has both good sensitivity and specificity in order to guide the management to control the outbreak. The testing that yields the most rapid result is polymerase chain reaction (PCR). The samples of choice are nasal swabs and buffy coat samples of blood collected from horses suspected to have this disease. A PCR test that distinguishes the mutation associated with EHM has been described.

The natural spread of this disease is through inhalation and ingestion, primarily by nasal aerosols from infected horses. Infections first occur on the mucosal surface of the respiratory tract, although direct contact with infected aborted fetuses or placental tissues may also serve as a source of infection. Spread of infection may occur by direct cell-to-cell spread and hematogenously through infected peripheral blood monocytes. The virus is considered to be endotheiotropic and results in vasculitis.

Clinical signs include fever, inappetence and depression, combined with serous nasal discharge and cough. Neurological signs are often preceded by a fever or upper respiratory disease in the few days to weeks prior to onset of neurological signs. The clinical signs observed as a result of EHV-1 myelitis can be quite variable. In most affected horses, symmetric ataxia and weakness of the pelvic limbs, urinary incontinence, and loss of sensation and motor deficits around the tail and perineal area are typical.

The management of horses with suspected EHV-1 myeloencephalopathy should be directed at achieving a safe environment and providing excellent nursing care. A horse with obvious bladder dysfunction should quickly and as frequently as possible have aseptic evacuation of the bladder. Prophylactic antibiotics are essential to combat the problems associated with the development of cystitis along with nonsteroidal anti-inflammatory agents such as flunixin meglumine, phenylbutazone, ketoprofen or Equioxx. The daily water needs for an affected horse should be 60 to 80 ml/kg daily. Along with the water, it is important to feed a gruel or if the horse can eat to provide a highly palatable source of energy and protein daily.

Treatment with antiviral drugs is now becoming more commonplace. Initial investigation looked at Acyclovir, however results using this drug were very inconsistent and often despite good dosing, blood levels failed to be recognized. Most recently use of valacyclovir (Valtrex)
has been investigated in the horse. This compound is a prodrug of acyclovir and has much better absorption and good bioavailability form the gastrointestinal tract.

Despite the fact that many available vaccines contain high titers of inactivated virus and are able to stimulate high titers of virus neutralizing antibodies in horses, none are able to provide protection against the most severe forms of the disease, abortion and myeloencephalopathy. Still, the risk of viral shedding may be decreased in herds of vaccinated horses. Therefore regular vaccination is still important. The ideal frequency of revaccination is unknown but may be two to three times per year.

At this time it is unclear whether any of the current or new vaccines will be effective against the neurological form of EHV-1. Preliminary work by researchers at Cornell University suggested a benefit using a modified live vaccine, however the data is not yet compelling enough to recommend changes in choice of vaccine. Understanding the role of the various arms of the immune system against this agent, particularly with regard to abortions and development of neurological disease, is important as is an understanding of the role of the various vaccines and adjuvants used in the production of herpesvirus vaccines.

A critical part in the prevention of both abortion and herpesvirus myeloencephalopathy is to reduce the duration of the cell associated viremia. Reduction of the cell associated viremia appears to be important in preventing abortion following challenge with live virus in previously vaccinated horses. Although the specific role of increased numbers of cytotoxic lymphocytes in prevention of myeloencephalopathy is unknown, it is likely this will play a role in protection from EHM in horses. In any case, having both a humoral and cell mediated response against EHV-1 appears to be important in prevention of this disease. Currently much work is being devoted to the investigation of the role of cell mediated immunity in EHV-1, as well as the level of protection afforded by currently available vaccines.

Investigation regarding EHV-1 is currently focused on three areas; the development of reliable and quick diagnostic testing, identification of the role of specific parts of the equine immune system active against this virus, especially the mutant strain that has “replicative aggressiveness”, and the development of a live virus vaccine which is hoped will be fully protective against the neurological form of this disease.

What Does the Future Hold
Outcomes for horses with neurological diseases are improving as a result of the recognition by many practitioners that including the neurological examination as a part of the routine physical examination leads to earlier diagnosis and treatment. In addition, newer rapid and quantitative testing for diseases that affect the nervous system of horses means early recognition and accurate diagnosis and ultimately leads to significant economic savings for owners and trainers. Over time many veterinarians are going to have access to horse side testing for infectious diseases as readily as they now have access to radiography.

Regardless of what neurological examination technique one chooses to employ, a difficulty faced by the examiner is elimination of the subjectivity associated with assigning a severity grade to the degree of weakness, ataxia, spasticity and/or dysmetria. In some texts a lack of joint flexion is described as a hypometria which could also indicate weakness5, 23, 185, 186 while in others a lack of joint flexion is used to describe “spasticity”131, 187. In the future with the benefit of technology, practitioners may be able to better evaluate subtle gait changes including weight bearing though the use of force plates and videography188-190. Techniques such as fuzzy clustering and kinetic gait analysis will likely become more commonplace8, 9, 191. Using kinetic gait analysis one is able to detect significantly higher lateral force peaks and greater variation in vertical force peaks in ataxic horse over lame horses8, while use of fuzzy clustering demonstrates a greater amount of uncertainty of movement in ataxic horses over lame horses8.

Following the identification of an ataxic horse as well as the neuroanatomic localization of the lesion, use of more sophisticated techniques for ante-mortem evaluation of the lesion are now available.192 Techniques such as magnetic resonance imaging, computed tomography and ultrasonography are currently becoming more popular. With continued publications regarding the benefit of these techniques it is my belief that modification of existing equipment will occur such that these techniques will become applicable to all parts of the equine anatomy, including the entire vertebral canal of an adult horse.

References are available from the author upon request
EARLY PREGNANCY LOSS IN THE MARE
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Abstract
In well-managed breeding farms, per cycle and per season pregnancy rates have improved significantly during the last 20 years. However, early pregnancy loss (EPL) remains a significant source of financial loss, with approximately 10-15% of pregnancies detected at day 15 after ovulation failing to survive to term. Interestingly, 60% of these losses occur in the period before day 40 after ovulation when pregnancy maintenance is critically dependent on progesterone produced by the primary corpus luteum (CL), and when a number of essential developmental events take place, including maternal recognition of pregnancy, development of the embryo proper, dissolution of the blastocyst capsule, the endometrial cup reaction and the onset of definitive placental formation. Despite its economic impact, surprisingly little is known about the most common causes of EPL and, therefore, whether, how and to what extent EPL can be prevented or averted. This is partly because EPL is usually diagnosed retrospectively (i.e. the conceptus has already disappeared), when it is no longer possible to determine the initiating cause. Moreover, there has been a tendency to assume that the primary problem is ‘progesterone insufficiency’ because this can be addressed pharmacologically, even though available evidence suggests that EPL more commonly occurs despite maintenance of a functional CL.

The possible causes of EPL can be broadly divided into embryonic abnormalities, inadequacy of the maternal environment and ‘external’ factors. Moreover, it is increasingly clear that abnormalities of the embryo per se account for a sizeable proportion of EPLs. In some cases there may be ultrasonographic evidence that conceptus development is not progressing normally, such as a ‘small for dates’ vesicle or failure of an embryo proper to develop. In addition, it was recently shown that a high proportion of equine embryos contain chromosomally abnormal cells, and sometimes in proportions guaranteed to compromise embryonic survival. While little is known about the true contribution of chromosomal aberrations to EPL in horses, it is likely that chromosomal abnormalities contribute to the increased incidence of EPL in older mares and in mares inseminated ‘too long’ after ovulation. It is also likely that for a proportion of the stallions associated with high rates of EPL the underlying problem is embryonic chromosome abnormalities arising from karyotypic abnormalities of the stallion himself or instability of the DNA in his sperm.

‘Deficiencies of the maternal environment’ is a broad concept encompassing diverse factors such as insufficient maternal progesterone, inadequate provision of nutrients from an aged or damaged endometrium and infection/inflammation as a result of unresolved post-breeding or subsequently acquired (i.e. ascending) endometritis. EPL due to endometritis is sometimes ultrasonographically evident as accumulation of uterine fluid despite the presence of a conceptus; subsequent conceptus death may result from either direct infection of the embryo or via luteolysis due to PGF2α release from the irritated endometrium. And while it is tempting to assume that inadequate maternal progesterone is a common cause of EPL, there is little evidence to support this assumption. Indeed, even when maternal recognition of pregnancy and CL maintenance have been shown to fail, the failure is often paired with abnormal pregnancy development (e.g. ‘small for dates’ vesicle) such that it is impossible to determine cause and effect. Nevertheless, luteal failure certainly does occur and can be induced by PGF2α release by organs other than the uterus, for example in the case of severe systemic illness accompanied by pyrexia and/or endotoxaemia. In addition, there is a growing body of evidence that the day 18-35 pregnant mare is particularly vulnerable to luteolysis because the inhibited endometrial PGF2α release capacity that underlies maternal recognition of pregnancy wanes after conceptus vesicle fixation on day 17. Indeed, a number of manipulations (e.g. twin crushing) and hormone fluctuations (e.g. oestradiol, oxytocin) have been shown to stimulate uterine PGF2α secretion during the day 18-35 period. While this PGF2α release rarely results in complete destruction of the primary CL, it is possible that there is an individual mare threshold for progesterone below which pregnancy may be endangered, presumably due to a compromised ability of the uterus to provide the nutrients required for conceptus development. Reduced endometrial quality as a result of advancing maternal age can also markedly affect the supply of nutrients to a conceptus, and while this classically leads to fetal compromise later in gestation, EPL can result if endometrial inadequacy is severe. Finally there are numerous anecdotal reports that stress in the form of pain, systemic disease, weaning, transport, changes in group structure, poor nutrition and extremes of temperature can predispose to EPL; although the exact role of stress in EPL is not clear, it is therefore prudent to minimise stress during early pregnancy. Preventing EPL is currently only realistic when a mare shows clear signs of returning to oestrus despite the presence of an apparently normal conceptus in her uterus, has a history of repeated EPL associated with...
a return to oestrus, is systemically sick or has recently undergone severe stress likely to compromise CL maintenance. In these cases, the logical approach is supplementation with a suitable progestagen (e.g. altrenogest) from as soon as CL failure is suspected, or in cases of repeated EPL from before day 7 after ovulation, and continuing until adequate maternal progesterone production is certain. While progesterone supplementation clearly has a role in protecting pregnancies threatened by maternal progesterone deficiency, its large-scale use is questionable because it will do no more than delay or mask EPL due to embryonic abnormalities and may exacerbate uterine inflammation in those caused by endometritis. Indeed, in cases of (suspected) abnormalities of conceptus development, frequent ultrasonographic monitoring is likely to be more useful and informative than progesterone supplementation.

In summary, although EPL is a significant cause of economic loss to the horse breeding industry, the development of strategies to combat these losses is currently hampered by inadequate understanding of the underlying causes.

Time management
Research showed that the average workweek of an equine veterinarian contains 53.5 working hours. You can try to decrease the hours by being more efficient with your time. This is straightforward and can be achieved by:
• Educate your clients. They have to adapt to your schedule as much as possible.
• Route planning that will minimize travel time
• Do not accept any appointments after a certain hour that you decide yourself (for example 19:00 or 20:00 pm) unless you want to. We also offer the owners the option to visit our clinic in the evening instead of going out on call.
• Try to increase the workload at your clinic and decrease the work in the field. It saves time and travel expenses. In the clinic you have better medical equipment and professional assistance. For less fortunate owners this could be a way of cutting down in veterinary bills by reducing travel expenses. Making your own schedule is another advantage of working at the clinic.
• Do not forget to stay flexible for your clients when you try to achieve a more efficient time management.

Knowledge and practical experience
You will get both by the years but you can do more to increase your level.
• You need good coaching from an experienced veterinarian
• Concentrate on general practice first before specialising
• Specialise by studying, visiting conferences, specific education, internships and last but not least hard and dedicated work. This will ask a lot of discipline and I believe you can only achieve this when you enjoy your job.
• Refer a case when you need to.

Administration
Most veterinary surgeons lack management skills and lose money by poor practice management. Getting a professional part- or full-time administrative assistant will quickly save money.

Investments
Research did not show any correlation between better accommodation and equipment with a better financial situation.
What questions have to be answered to decide to make an investment?
• How will the investment be financed?
• What is on the market?
• Does the investment result in direct increase of turnover or will it attract a new group of clients.
• Is the investment a tool to achieve a future goal?
• Is the investment necessary to avoid losing clients?

**Staff management**
When there are more people working there has to be more time for staff management. We started with 2 veterinarians only and now our team consists of 4 veterinarians 3 assistances (in the clinic and the stable) one bookkeeper and one fulltime trainee. Subjects we try to manage in our team are:
• How to control the working ability and are the given instructions being carried out.
• Keep everybody motivated
• Coaching your new young veterinarians
• Appreciate the work everybody does. You can tell them you are very pleased and you can give them a nice working environment.

**Contacts outside the veterinary world**
It is very important to have good contacts outside the veterinary world. This group of people can increase your customer file. What people do I mean in particularly?
• Farriers
• Physiotherapists
• Riding school owners and private stable owners
• Instructors
• Saddle fitters
• Tack shop owners

**Expanding your clinic with non-conventional medicine**
In the (top)sport horse business there is a lot of interest for non-conventional medicine. This is called integrative medicine, acupuncture, chiropraxis, osteopathy, laser-therapy, herbology and so on. The advantage of these particularly methods is that the treatments are most of the time doping negative and can be used (if done by a trained professional) to enhance the performance and wellbeing of the top athletes shortly before the competition. The combination of the non-conventional and conventional medicine can achieve better results then both methods used separately.

**HOW TO MANAGE EQUINE RHODOCCOSIS ON AN ENDEMIC BREEDING FARM**
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*Rhodococcus equi* (*R. equi*) pneumonia and diarrhoea can cause high losses in foals and consequently economic losses in breeding farms because of high morbidity and mortality. *R. equi* is a gram positive bacterium with 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*. This has lead clinicians and researchers to evaluate the efficacy of medical prophylactic methods in foals of endemic breeding farms such as vaccinating mares, vaccinating foals, repeated administrations of hyperimmune serum to neonates and older foals, immunostimmulatory treatment in foals and metaphylaxis through administration of macrolid antibiotics within the first weeks of live. None of these measures have shown to provide protection to the foals and reduce the morbidity on endemic breeding farms (Hurley and Begg 1995, Becú et al. 1997, Chaffin 2003, Schulte 2005, Baumann 2006, Hullmann 2006, Venner et al. 2007c).

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 have been evaluated whether they influence the disease rates on endemic breeding farms. The effect of separation of sick foals, high hygiene and medical level in the perinatal period, reduction of dust and reduce animal density on the farm was investigated (Chaffin 2008). So far no specific farm management measures have been identified, which could account to a reduced morbidity of foals on *R. equi* endemic 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 an 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 an 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. Treatment should be started early in order to reduce shedding of *R. equi* into the environment. Adequate treatment is considered to be the combination of a macrolid antibiotic (azithromycin, clarithromycin or tulathromycin) and rifampicin over a period of 4 to 8 weeks.

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HOW TO EXAMINE AND TREAT COMMON EQUINE DENTAL PROBLEMS

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Examination of the incisors, the bars or the premolars in the horse’s mouth is fairly easy and can be accomplished using a minimum of manipulation of the lips and lip commissures. Treatment of encountered pathologies in these regions is therefore mostly straightforward. Detecting dental problems in the back of the mouth on the other hand will require more skills and the use of specialized instruments to allow their proper identification and treatment. If performed with the correct intentions, examining a horse for possible dental problems should be done systematically and thoroughly. A dental exam should be preferentially performed in a quiet and indoor surrounding to avoid unnecessary disturbance of the patient. This ‘examination room’ should be free of any obstacles, have a rough surface and provide the necessary utilities for water and electricity. The exam starts with a brief history of symptoms and other relevant information. After having observed the animal, careful palpation of head and neck may already attract our attention to one or more points of interest. These may include muscle atrophy, swollen and/or painful areas, and fistulous tracts. If possible, detailed inspection of the animal’s manure will allow detection of long fibres (> 1cm) and/or undigested grains indicating insufficient masticatory preparation of food. For the next steps of our examination the horse should receive a sedative (α2-agonist ± opioid) which greatly facilitates the cooperation of the patient while manipulating its head and using different instruments. After thoroughly flushing the mouth to remove any food remnants, we’ll determine the horse’s age based on its

Proceedings of the European Veterinary Conference - Voorjaarsdagen, 2009 - Amsterdam, Netherlands

Abstracts European Veterinary Conference Voorjaarsdagen 2009
mandibular incisors to help direct our attention to age-related tooth problems and further inspect all incisors for any pathology. The range of motion of the lower jaw in relation to the upper jaw is determined both in the latero-lateral (lateral excursion) and in rostro-caudal (antero-posterior movement) direction. The first is done by moving the mandible sideways while looking for (a)symmetry between left and right and measuring lateral excursion until separation of incisor teeth. Antero-posterior movement is observed while flexing and extending the head and checking occlusion of the incisors. The bars are carefully palpated (eruption of canines, (blind) wolf teeth) before application of an appropriately sized full-mouth speculum to examine the deeper parts of the mouth.

Some specialized instruments/aids will greatly facilitate easy recognition of common dental abnormalities. Good illumination is a prerequisite to accomplish a good dental exam. It includes the use of a head lamp that preferentially positions the light source between the eyes. A dental mirror, cheek retractors and long dental picks or probes further complete the vet’s possibilities to fully examine the mouth. Inspection of the cheek teeth should be done from front to back looking for asymmetries, overgrowths, fractures, missing parts, discolorations of the occlusal surface and presence/absence of interproximal spaces residing food remnants. The soft tissues (tongue, cheek, gingiva) are carefully checked for the presence of lesions caused by the neighboring teeth. Then, careful palpation of the different teeth may further reveal/confirm the presence of suspected dental problems. Any diagnosed problem can be recorded on a dental record sheet (paper or computer-based). This will greatly enhance communication to the horse owner and allows evaluation of the evolution of a tooth problem on later occasions.

Commonly encountered tooth problems in horses can be classified into developmental problems and abnormalities of wear. They are the kind of problems every equine practitioner should be aware of and should be able to treat in proper manner.

Abnormalities of eruption are the most commonly encountered developmental problems and can be seen both in the incisor as well as in the premolar region. Loose, displaced or rotated deciduous caps (premolars) can cause oral discomfort and should be removed. Persistent incisor and premolar caps can become displaced and cause irritation or disturb the normal congruency of the dental arcades with subsequent abnormalities of wear later in life. Diagnosing this problem requires knowledge of age determination, eruption sequences and identification of deciduous versus permanent dentition. Removal of a persistent deciduous tooth is easily performed in the standing animal. A root elevator (incisors) or cap extractor (eg. Reynold’s type for premolars) are suitable for this purpose. Delayed eruption of canine teeth is often accompanied by the formation of an inflammatory reaction around the unerupted crown which can be painful or cause abnormal bit behavior. Making longitudinal release incisions greatly facilitates the eruption process and reduces any oral discomfort. The same can be seen in case of an impacted wolf tooth which is treated by tooth extraction. Impaction of cheek teeth is most commonly encountered in the 8’s and may result in periapical disease or displaced eruption of the permanent tooth with subsequent periodontal disease later in life. Careful evaluation of existing ‘eruption cysts’ (symmetrical swelling especially visible on the mandible) and examination of the dentition for delayed shedding of caps should direct the practitioner to deciding on whether to prematurely remove milk teeth to avoid more serious problems.

The ‘modern’ way our horses are fed (commercial soft food, lush pastures, feeding from a height) predisposes them to the development of sharp enamel points on the outside of the upper and the inside of the lower cheek teeth. In up to 80-90% of our horses they will be accompanied by the presence of lesions in the soft tissues of the cheek and tongue. A lot of these lesions can only be recognized if careful illuminated examination of the mouth is performed. They’ll mostly have a fibrous and chronic aspect and are yellowish-brown colored in comparison to the surrounding pink healthy mucosa. Their removal is the most frequently performed dental procedure but also one of the most debated topics as to the degree of correction that should be achieved. This author advocates a more conservative approach to dental floating where the appreciation of the sharpness of the teeth should be done with the fingers. My golden rule has been: ‘if I firmly palpate the teeth and my fingers cannot be wounded by any remaining roughness’s on the specific predilection places of the cheek teeth, than my dental correction has met with the requirements to prevent further lacerations to the soft tissues of the mouth and I can stop at that point’. Corrections are preferentially made with hand instruments held at an angle of 45° to the occlusal surface. Motorized equipment is evenly possible to execute these routine corrections although in the hands of an inexperienced person, overcorrections are easily made causing the patient to exhibit more complaints after the procedure than before.

Focal overgrowths on the rostral aspect of the upper second premolars and the caudal aspect of the lower third molars are sometimes denoted with the terms fronthooks and backhooks respectively. They will be encountered frequently in all different kinds and shapes
and should definitely be removed to allow normal ranges of motion of the jaws. Diagnosis of fronthooks is very straightforward but identification of backhooks requires proper sedation and meticulous examination of the caudal teeth. Their treatment is as so. Motorized equipment is very handy in these cases and will allow quick and thorough removal of the hooks although careful manipulation of the instruments is required in the back of the mouth to avoid inadvertent damage to the surrounding soft tissues. When using motorized equipment to remove more important overgrowths it is extremely important to avoid floating the same surface for longer than 30’ in a time without cooling the tooth with cold water. Otherwise thermal damage to pulp horns may lead to insufficient secondary dentine production during subsequent tooth wear which will cause the pulp cavity to become exposed to the oral environment and provoke tooth infection (mostly after 3-5 years).

Wave, step and shear mouth conformation are less frequently encountered. Depending on the degree of wear abnormality, these conditions require step-wise corrections at 3-4 months intervals.

OUTCOMES OF STIFLE ARTHROSCOPIES IN COMPETITION HORSES

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The indications for arthroscopy of the stifle fall broadly into two categories. 1) Horses in which no clinical, radiographic, ultrasonographic or scintigraphic abnormalities in the stifle have been detected; their lameness has been abolished by intra-articular analgesia of the stifle, and conservative treatment has been unsuccessful. 2) Horses with specific stifle lesions for which arthroscopic investigation or treatment is indicated.

From a series of 631 stifle arthroscopies at the Liphook Equine Hospital the following were the most frequently diagnosed lesions deemed to be the main cause of lameness:
Articular cartilage disease (not including cases where this was secondary to another injury) 152; meniscal injuries 126; osteochondrosis dissecans (OCD) 106; cruciate injuries 75; subchondral bone cysts 45; patellar fractures 15, other fractures 16.

Lesions in the articular cartilage of the medial and lateral femoral condyles (MFC and LFC) were grouped as follows: softening and fissuring of the articular cartilage with localised areas of fibrillation (Outerbridge grades 1 to 2); localised areas of cartilage fibrillation; extensive areas of cartilage fibrillation (grade 3); erosion of the articular cartilage to subchondral bone (grade 4); chondral flaps.¹

Lesions were treated by careful removal of loose, projecting cartilage flaps and superficial fibrillations followed by thorough joint lavage. Postoperatively the affected joint was medicated and the horse kept in controlled exercise for 6 months. Of the horses with Outerbridge grades 1 and 2 lesions, 77% returned to full work and this figure was 63% for those with grades 3 and 4 lesions.

Meniscal tears were graded by the amount of damage visible at arthroscopy rather than by morphology.² Damaged tissue was debried. Of 126 meniscal tears, 101 occurred in the medial meniscus; overall 50% of horses returned to full work; 60-65% of horses with mild to moderate and 10% of those with severe lesions were successful. The prognosis for return to full work was significantly related to the severity of the injury, the presence of articular cartilage lesions and the presence of radiographic changes.²

OCD lesions of the femoropatellar joint occur most frequently on the lateral trochlear ridge of the femur but are also encountered on the patella and medial trochlear ridge.³ In acute cases in foals where large areas of cartilage may be loose surgery may be delayed in the hope that some cartilage may reattach and reduce the size of the lesion. The use of polydioxanone pins to fix these lesions can be successful in some cases.⁴ In older horses complete debridement of lesions and thorough lavage of the joint is performed. The prognosis is reasonable and has been reported as 64% of 161 horses returning to athletic use; best results were seen in older horses and those with mild lesions. From a series of 106 horses treated arthroscopically for OCD at the author’s practice, 83% returned to full use; 91% of horses under 3 years and 87% of horses 3 years and over became athletes. The presence of associated degenerative change on the articular cartilage, which was seen in 47% of cases and more often in the older horse, did not affect the prognosis.

Cranial cruciate ligament (CrCL) injuries are often difficult to evaluate because the CrCL lies beneath the fascia of the median septum. Small avulsion fractures or fracture fragments off the tibial intercondylar eminence may also be present. Removal of loose dam-

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Aged tissue is the only available treatment for cruciate ligament tears. In a series of 75 cases of cruciate injuries diagnosed arthroscopically at the author’s hospital the prognosis appeared to be related to severity. Only four involved the caudal cruciate ligament. Overall 56% of horses returned to athletic use; 62% with mild injuries and 33% with severe injuries were successful. However, many severe injuries are euthanased without an arthroscopic examination.

Subchondral bone cysts occur most frequently in the distal MFC but they are also seen in the proximal tibia, and cyst like lesions occasionally develop in the caudal MFC or LFC in foals. Debridement of the lesion has been shown to be less successful in horses older than 3 years than those under 3 years (Walmsley unpublished data). Multiple injections of corticosteroid into the lining of the cyst under arthroscopic control have given more promising results in older horses, 13 out of 18 being successful in one report.

Medial sagittal and basilar fractures of the patella, fragmentation of the patellar apex, fracture fragmentation of the trochlear ridges and condyles of the femur and fracture of the medial intercondylar eminence of the tibia (MICET) can be treated arthroscopically. Fifteen patellar fractures were treated arthroscopically in the author’s series and of those followed up 64% returned to work. Arthroscopic treatment of fragmentation of the patellar apex carried a similar prognosis. Excision of fractures of the MICET was performed in 20 of 22 cases and 13 out of 18 horses followed up returned to use (Roneus and Walmsley unpublished data 2008).

References

WHAT CAN SUPPLEMENTS DO FOR YOUR HORSE?
C.M. Westermann

If one searches the internet for “supplement + horse” the Dutch Google system finds 17.800 hits, and the English version even more: 4.080.000. If, on the other hand, Pub Med is used, the number of hits is only 250. In equine practice the same occurs: every horse owner hears a lot about food supplements and everyone has an opinion on a few supplements but nobody knows exactly what is true or not. Producers claim that products have many beneficial properties, but most claims are unsubstantiated. In contrast to medications, supplements don’t have to undergo many tests to be allowed on the market. So often the scientific base is missing.

To think that this problem can be solved in 45 minutes is not realistic. But to give it a go the author and a group of final year students analysed literature and books to obtain evidence based information regarding some supplements that are popular and have a relationship with poor performance or enhancing performance. We realize that literature is not the only aspect that one should take into account when advising a client about their horse, and that “good results in the past”, “trust”, “placebo-effect” and many other factors are also important, but we want to share our findings with you to broaden your vision about them.

Causes for poor performance are numerous. The most common are disorders of locomotion, respiration, circulation, muscle system and metabolism. We have chosen to evaluate some supplements based on popularity, discussion between veterinarians and personal interest of the group. A short explanation and conclusion for some supplements will be given in this abstract.

In the case of locomotion disorders a popular supplement given for joints is glucosamine, and for the hooves biotin. In cases where poor performance is suspected to originate from muscular disorders vitamin E, selenium and carnitine are important supplements. Metabolic causes for poor performance are often thought to be due to an electrolyte disbalance. Iron is claimed to enhance performance and power.

Glucosamine is one of the supplements that can be given to horses with osteoarthritis. It is a water soluble amino monosaccharide. The economic turnover is more than 50 million dollar per year. Analysis of the literature revealed that:

- there was no correlation between product quality and price.

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-many products advise subtherapeutic dosages
-glucosamine seems to be a safe product
-the oral biological availability is only 5.9%
-experiments have different results: some show positive results with glucosamine, others show no improvement, and sponsored trials show better results than non-sponsored trials…

Our department has started a double blind experiment to determine if glucosamine improves the locomotion of the geriatric horse.

**Biotin** is a sulphur-containing vitamin that is part of the vitamin B complex. Absorption from food occurs in the small intestine, and horses also have intestinal production in the colon. Deficiency is rare but could arise if the horse has gastrointestinal problems. Several experiments describe a positive effect of biotin on the repair of poor hoof quality. One should, of course, not forget that hoof quality depends on many other factors too, including management, housing and grooming.

**Vitamin E** and **selenium** are antioxidants that are claimed, among other things, to reduce acidification of muscles. Several myopathies have been described that are considered to arise (in part) from a deficiency of vitamin E and sometimes selenium. Normally, supplementation of these antioxidants is not necessary, and in the case of selenium, may sometimes even be toxic, but in active sport horses, myopathic horses and horses that are fed a fat-rich diet, supplementation of vitamin E can be useful. A problem concerning vitamin E is that a single determination in blood is not reliable, because the concentration fluctuates during the day and blood contains only about 1% of the vitamin E in the body. Multiple analyses or analysis of fat tissue is more reliable, but also more expensive.

**Magnesium** has the same problem as vitamin E: the analysis is not reliable. Magnesium concentrations in one horse differ between (pseudo) hypo- and hypermagnesemia in one day. If a horse really suffers from a persistent hypo- or hypermagnesemia many symptoms can be seen. Muscle biopsy or collection of 24-hour urine can produce more reliable results if problems are suspected. Horses that are fed normally will not develop hypomagnesemia.

**Carnitine** has several functions, including assisting the entry of long-chain fatty acids into the mitochondrion to be converted into energy via beta-oxidation, and buffering toxic by-products of metabolism by binding to them and being excreted in the urine. Currently, research is being performed at our department to study the role of carnitine in glucose-insulin metabolism.

**Electrolytes**
Horses lose electrolytes by sweating. Normally, these losses are well compensated. In case of long-term heavy work in a warm, humid atmosphere it is advisable to supplement horses with electrolytes. This needs to be done several hours before starting to work, because the horse needs time to drink enough water

**Iron**
Iron is an essential part of cellular respiration and oxygen transport. In normal feed enough iron is available. Supplementing healthy (sport)horses might even be toxic. In case of chronic blood loss, periods of rapid growth of foals or in some cases of chronic illness supplementing iron could be beneficial.

## The Heart and Poor Performance: What Role Does It Play

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**General**
The main causes of poor performance in horses are lack of ability and/or motivation and inappropriate training. Cardiovascular causes of poor performance are less common than respiratory and orthopaedic diseases, although many horses have cardiovascular dysfunction, especially cardiac murmurs, and as a consequence, the significance of cardiovascular abnormalities can often be difficult to determine in individuals. The end result, of course, is that the heart is often blamed for loss of performance when it is completely innocent and, conversely, on the rare occasion cardiac disease is significant, it is not uncommon for it to be totally overlooked. Today’s presentation will attempt to introduce some perspective into the bewildering array of cardiovascular abnormalities commonly encountered in Thoroughbreds and their effects on performance.

**Cardiovascular causes of poor performance**

**Dysrhythmias**
Dysrhythmias occur commonly in horses and with the notable exception of atrial fibrillation (AF), usually do not affect performance.

**Permanent and paroxysmal atrial fibrillation**
This dysrhythmia is the commonest cardiovascular cause of poor performance in horses. The approximate
incidence of the sustained form of the dysrhythmia is 1% in National Hunt Thoroughbreds. When present, the sustained form always affects race performance. The prevalence of the sustained form of AF is lower in flat racing Thoroughbreds, but it is likely that paroxysmal AF occurs more commonly than we appreciate. Recent data (Ohmura and colleagues, 2003) have suggested the incidence of paroxysmal fibrillation is 1.4% in poorly performing flat race horses c.f. to 0.3% in the population without a history of poor race performance.

At rest, when diastole is long and cardiac output is low, the loss of atrial contraction has little effect. However, during exercise, when diastole is short, peak cardiac output is reduced despite the increased heart rates during exercise. As result, the performance history of these horses reflects compromise at maximum effort. Depending on the training regime, the problem may not be apparent at home. If fibrillation occurs suddenly during a race, the horse may pull up in distress, recovering after a few minutes. In hunters, performance problems are seen during intense exercise usually up hills. Dressage horses, showjumpers and hacks rarely show signs of poor performance despite the presence of sustained AF.

Successful treatment of atrial fibrillation should result in return to previous exercise tolerance. However if the dysrhythmia is not affecting performance, in most cases treatment should not be attempted. In some horses, recurrence of paroxysmal or sustained atrial fibrillation is a problem, although some owners and trainers become aware of the trigger factors and learn to manage them accordingly. In other cases when frequent recurrence, or poor tolerance of treatment occur, an alternative career can often be found for the horse. In such cases, and before treatment is attempted, it extremely important to establish that the rhythm is present in isolation and has not occurred secondary to atrial enlargement and heart failure.

**Murmurs**

*Mitrals and tricuspid valve regurgitation*

<table>
<thead>
<tr>
<th>Regurgitation</th>
<th>Prevalence by auscultation</th>
<th>Prevalence by Doppler</th>
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<tbody>
<tr>
<td>Mitral</td>
<td>21%</td>
<td>67%</td>
</tr>
<tr>
<td>Tricuspid</td>
<td>46%</td>
<td>88%</td>
</tr>
<tr>
<td>Aortic</td>
<td>5%</td>
<td>65%</td>
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</tbody>
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In our recent epidemiological study of 700 flat and NH racehorses (Young et al 2008), we were unable to detect a significant effect of valve murmurs or regurgitation assessed by colour flow mapping on BHB or Time-form rating, regardless of whether presence or absence of murmur, or only murmurs >grade 3/6, or regurgitation >6/9, were considered.

Although uncommon, severe AV valve disease that has progressed to cause heart failure is always associated with poor performance, yet despite this, performance decrements may not be evident to the horse owner, as it will still depend upon the level of work being done by the horse.

Most commonly such cardiac failure is associated with catastrophic mitral regurgitation, often in conjunction with secondary atrial fibrillation. Progression of either mitral, or tricuspid valve disease to cause cardiac failure is rare. To differentiate between compensated and decompensated disease, check resting heart rate and look for the presence of a widely radiating murmur, associated with a palpable precordial thrill.

**Aortic insufficiency**

Aortic insufficiency is most commonly detected in middle aged to older horses and is the least common of the murmurs encountered in performance horse practice. The condition is progressive and is associated with thickening and fibrosis of the valve leaflets. It is usually initially detected as a low-grade murmur (grade 1-2/6) in horses over 10 years of age, and then progresses very slowly. In older horses, it rarely affects performance, as progression occurs over many years and work expectations usually decrease with age. Aortic insufficiency causes volume overload of the left ventricle resulting in dilatation. Pleasure horses can continue to perform adequately even with severe aortic insufficiency and marked volume overload. However a dilated ventricle works against increased afterload and has an increased oxygen requirement. In aortic insufficiency, there is decreased coronary perfusion due to reduced diastolic aortic pressure and oxygen delivery to the myocardium is compromised. Increased oxygen demand and reduced coronary reserve increases susceptibility to ventricular arrhythmias that can result in sudden death. Affected horses must be monitored regularly so they can be retired from work before severe ventricular dilatation develops.

Aortic insufficiency also occurs sporadically in younger racehorses. The condition may be associated with the presence of a small ventricular septal defect or there may be no obvious pathology other than valve leaflet prolapse +/- dilatation of the aortic root. Severe aortic insufficiency in such animals can progress to heart failure, although in some cases progression is very slow and the
racing career is not noticeably compromised. Progression of this condition is impossible to predict in individuals, so all affected cases should be carefully monitored.

**Ventricular Septal Defect**
The size of a ventricular septal defect will determine its affects on performance. If the defect is small and restricts blood flow between the two ventricles, performance will not be affected. If however the defect is large, horses perform below their genetic potential. The intensity of the expected exercise will also influence whether performance decrements are observed. Show and pleasure horses perform with relatively large ventricular septal defects that would probably be incompatible with a successful racing career. Echocardiography is valuable for determining the likely significance of a ventricular septal defect. Murmur loudness is not necessarily associated with a poor prognosis

**Vascular causes**
**Aortoiliac thromboembolism**
This is an occasional cause of reduced performance in horses. It appears to be more common in male horses and can occur in any age group. Symptoms vary from mild hind limb stiffness that reduces performance to more severe unilateral or bilateral hindlimb lameness. Although one would generally expect to recognise the characteristic clinical signs associated with this condition, this is not invariably the case, and we have diagnosed this disorder in horses referred for poor performance investigation with no previous history of exercise-induced lameness.

**Less common cardiac causes of diminished performance**
**Bradydysrhythmias**
In the resting horse many bradydysrhythmias are of no clinical significance, as within limits, cardiac output can be maintained by an increase in stroke volume. The trained horse has an increased resting stroke volume and therefore the resting heart rate is normally low. During exercise however, there is an increased requirement for muscle perfusion and cardiac output must increase to maintain arterial blood pressure and blood flow. The major part of this increase in cardiac output is caused by an increase in heart rate. Therefore if a low heart rate is maintained during exercise, cardiac output will be inadequate and performance will be limited. Occasionally these conditions are associated with collapse at rest and during exercise. Examples include 3rd AV block and advanced 2nd degree AV block. Pathological sinus bradycardia is found most commonly in elderly ponies and donkeys. Occasionally horses affected with profound 2nd degree AV block and sinus bradycardia respond favourably to daily oral administration of β1 adrenoceptor agonist drugs e.g. clenbuterol. In general for bradydysrhythmias, the prognosis is guarded to poor.

**Tachydysrhythmias**
Tachydysrhythmias reduce cardiac output by reducing the time available for ventricular filling. In addition ventricular depolarisations reduce cardiac output further by decreasing stroke volume due to incoordinate contractions.

**Atrial premature systoles** are detected fairly frequently in horses in training and affect performance only if they produce excessive heart rates during exercise, or when they predispose to paroxysmal atrial fibrillation. More commonly the premature excitatory focus is over-ridden as sinus nodal rate increases, and the extrasystoles disappear, thus poor performance is not usually a feature.

**Ventricular premature systoles** will also affect performance if they produce a tachycardia during exercise. This dysrhythmia is of more concern due to the risk of sustained ventricular tachycardia predisposing to ventricular fibrillation. This situation is likely to be exacerbated during exercise when sympathetic tone increases. Ventricular extrasystoles are very common in normal horses during the recovery period after fast exercise. A hypothesis for their role in sudden cardiac death will be discussed in the lecture that follows.

**Useful references**


Ohmura H, Hiraga A, Takahashi T, Kai M and Jones JH. Risk factors...


HEART SIZE AND PERFORMANCE: ARE THEY RELATED?

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Introduction

The role of the heart in defining athletic performance has been the subject of speculation and interest in human and equine sports medicine for many years. Post mortem examination of superlative athletes - both equine and human - have revealed substantially larger hearts than would have been predicted from lean body mass. The Fick principle and the dependence of cardiac output and thence VO2 on stroke volume suggest a relationship between relative heart size and athletic performance is likely; particularly as success in endurance competitions is inextricably linked to the ability of an individual to support a high VO2 over prolonged periods. Nevertheless, despite a body of evidence supporting the concept that heart size and athletic performance are related, this hypothesis had never been satisfactorily proven in a population of athletes with a wide range of abilities.

Experimental design

Cardiac morphology in human athletes is known to differ depending upon the sports-specific endurance component of their primary event, whilst anecdotes abound about superlative athletes with large hearts. As the heart is a key determinant of stroke volume and $VO_{2\text{max}}$, in mammals, we undertook a study to test the hypothesis that the morphology of the equine heart would also differ between trained equine athletes, depending upon their race type, and that left ventricular size would be greatest in the horses racing successfully.

Methods

Echocardiography was performed in 482 conditioned and race fit Thoroughbred racehorses engaged in either flat (race distance 1000-2500m) or jump racing (3200-6400m). Associations between different indices of left ventricular size and function and objective measures of race performance were determined using a standard regression approach.

Results

Body weight and gender adjusted measures of left ventricular size were largest in horses engaged in jump racing over fixed steeplechase fences compared to horses running shorter distances on the flat (range 8 – 16%). The observed differences in cardiac morphologies between horses suggested that subtle differences in training and competition also results in differing cardiac adaptations that are appropriate to the endurance component of the event. Derived left ventricular mass was strongly associated with published rating (quality) in horses racing over longer distances in jump races (P≤0.001), but less so for horses in flat races. Rather, left ventricular ejection fraction and left ventricular mass combined were positively associated with race rating in older flat race horses running over sprint (<1408m) and longer distances (>1408 m), explaining 25-35% of overall variation in performance, as well as being closely associated with performance in longer races over jumps (23%).

Conclusions

Predicted differences between otherwise equivalent horses with small and large hearts explained a significant proportion of the difference between elite and non-elite racehorse performance, providing the first direct evidence that cardiac size influences athletic performance in a group of mammalian running athletes.

Useful References


Young LE, Marlin DJ, Deaton C, Brown-Feltner H, Roberts CA and Wood JLN. Heart size estimated by echocardiography correlates
Young LE, Rogers K and Wood JLN. Left ventricular size and systolic function in Thoroughbred racehorses and their relationships to race performance *J Appl Physiol* 99 1278 -1285, 2005