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FOR ANIMALS. FOR HEALTH. FOR YOU.
There have been remarkable technological advances in diagnostic imaging modalities over the past two decades, and, perhaps, equally significant improvements in ‘user friendliness’. Collectively, this progress has improved our abilities as equine practitioners to make very specific diagnoses of musculoskeletal conditions, often at point of service.

Digital Radiography.

Radiography has served as the primary musculoskeletal imaging modality for many decades. Self-evidently, radiography is primarily indicated for skeletal imaging but is not particularly sensitive to soft tissue pathologies, or to early or subtle osseous changes.

The most impressive advances in radiography relate to substantial improvements in the image quality from portable units and the digitization of the ‘output’ images. Digital images are available immediately for viewing and can be altered via many post-acquisition processes; magnification, altered contrast and positioning, black/white inversion, image annotation and measurement, quantitative analyses.

Further, digital radiography has largely eliminated the problem of image storage and also made image sharing for remote consultations and referrals extremely easy. Your consultant is only an e-mail away.

Ultrasonography.

U-S has been the imaging modality of choice for soft tissue pathology in equine limbs for the past 4-5 decades. In this respect, U-S conveniently complements the strengths of radiography for the assessment of both skeletal and soft tissue lesions. As with radiography, most of the advances in recent years relate to improved quality of image quality, particularly with portable units, and the markedly improved ‘image handling and storage’ options that digital imaging provides.

Although the flexor structures palmar/plantar to the cannon and fetlock are the primary targets for U-S imaging in most equine practices, U-S is also extremely valuable for identifying less common peri-
articular soft tissue, musculotendinous junction and muscular injuries. Doppler “flow” functions also allow for the assessment of vascular flow in and around sites of injury.

**Nuclear Scintigraphy.**

Scintigraphy provides very poor anatomical resolution, in comparison to radiography, CT and MRI, but, unlike other modalities, scintigraphy provides direct and highly sensitive information on skeletal patho-physiology. There are clear liabilities associated with this modality. The facilities required to house a gamma camera are expensive to construct and maintain, the isotope-phosphate complex is not easy to access, the imaging process itself is labour-intensive, imaging times with older horses can be prolonged and strict bio-security measures are mandated to protect against excessive radiation exposure (this, despite the very short half-life of Tc⁹⁹). Accepting these logistic issues, scintigraphy is an invaluable imaging modality in several contexts, as follow:

- multi-limb/multi-level lameness cases
- poorly defined and/or intermittent lameness, where nerve and joint block responses are equivocal
- suspected vertebral problems
- lameness cases localized to a specific skeletal ‘site’ but with no evident radiological changes
- severe ‘proximal limb’ lameness cases, such as pelvic fractures, where general anesthesia (required for diagnostic radiologic images) is an unacceptable risk

Commonly, scintigraphy provides an ‘embarrassment of riches’ with potential diagnoses and additional blocks and imaging are usually necessary to exclude clinically irrelevant ‘hot spots’ and confirm specific pathological diagnoses. Although scintigraphic imaging is usually limited to ‘bone scanning’, 2-3 hours after isotope administration, vascular and soft tissue pool phase images can also be acquired within the 1-15 minutes following the isotope injection, to assess differences in vascular supply to specific sites. While acknowledging the logistic issues with this modality, scintigraphy provides highly sensitive ‘whole body’ diagnostic information that cannot be accessed using other modalities.

**Computed Tomography.**

‘CAT scans’ are generated by an X-ray emitter/receiver that rotates around the patient’s target organ(s) to produce a number of cross-sectional image ‘slices’. These slices can be combined via post-acquisition computation into 3D reconstructions of the target. CT is able to discriminate between different tissue densities with far greater resolution than conventional radiology and avoids the inherent problem of superimposition. CT facilities capable of accommodating equine patients are expensive to
set up and maintain, and the target structure size that can be scanned is limited by the CT gantry diameter; restricting imaging to the equine limbs and head for the most part. CT imaging is far quicker and less expensive than MRI (see below) and so has obvious advantages where both modalities are availbale.

Although CT provides high-resolution information on soft tissue and vascular structures as well as bone, the modality’s primary application in horses is the assessment of complex joint pathology and immediate pre-surgical determination of comminuted fracture configurations; challenging cases that are likely to remain the ‘property’ of tertiary referral centers for the foreseeable future.

**Magnetic Resonance Imaging.**

MRI essentially records signals derived from free water molecules. It provides extremely high-resolution images of soft tissue structures, along with ‘water-poor’ tissues such as tendon and bone, when water molecules accumulate through edema or inflammation.

The ‘target’ is limited by the size of the MRI coil that needs to surround target region, so the site of the lameness needs to be localized to a 10-15 cm ‘zone’ before MRI becomes relevant. Imaging times are prolonged, in comparison to other modalities; 2-3 hours under general anesthesia is not uncommon in cases that require multiple, bilateral sequence acquisitions. A comprehensive MRI scan generates several hundred images and a wealth of high-resolution information on the target structures. Analyzing an MRI study requires a high degree of expertise, a systematic approach and a considerable amount of time. MRI is now the gold standard for imaging the equine foot, given that many distal limb lameness cases involve soft tissue injuries within the hoof capsule that are not accessible with ultrasonography. MRI is also very informative for joint problems (fetlock, knee and hock). Contrast-enhanced MRI (dGEMRIC) requires high-field magnets to be accurate but can be used to quantitatively assess cartilage thickness and matrix glycosaminoglycan content.

There are major financial and infrastructural constraints to MRI, although low-field standing units such as the Hallmarq Limbscanner mitigate these restrictions somewhat. Several Equine Veterinary Diagnostic Imaging Centres have been established in ‘horse dense’ regions of the US in recent years, designed to serve the surrounding primary equine practices. This business model effectively spreads the burden of the equipment purchase and maintenance across a large caseload base.
Yearling Radiography – how to optimise your images

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The quality of a radiographic image directly affects its usefulness as a diagnostic tool. Each yearling’s 36 radiographic views submitted to the Sales Repository should allow adequate visualisation of the joint or area being evaluated, that is most likely to be affected by pathological changes. For example the dorsal palmar/plantar view of the fore and hind fetlocks are required for examination of the fetlock joint space, the distal aspect of the third metacarpal/metatarsal bones, the proximal first phalanx and the distal aspect of the proximal sesamoid bones. In the instance of yearling thoroughbreds; primarily veterinarians are looking to identify osteochondritis dissecans (OCD), subchondral bone cysts or osseous cyst like lesions, osteophytes and entheseophytes, sesamoiditis, fractures and fragments and club feet. Poor radiographic quality may result in artifacts, conceal abnormalities or lead to misdiagnosis.

A study was published in 2011 by M Jackson et al that examined 167 sets of radiographs from 8 major Australian yearling sales in 2003. The aim of the study was to quantify common errors when radiographing horses. Such errors included movement, under and over exposure, positioning, labels or markers and processing errors. These were identified by 4 radiologists. 3.8% of films were found to be non-diagnostic with 78.2% of these due to positioning errors. The images that were most commonly affected were the flexed lateral to medial of the fetlock, the lateral of the hind fetlock and the dorsomedial oblique of the carpus. 30.2% of radiographs were less than ideal. Overexposure, particularly the lateral stifle and the front fetlock oblique views contributed to these less than ideal radiographs. 39% of radiographs were less than ideal due again to positioning, particularly the medial oblique carpus and lateromedial tarsus views. The lateromedial view of the foot was the least likely view to be taken incorrectly. Only 3.4 percent of the 167 sets had no errors. Radiographic quality has improved dramatically with the introduction of Digital Radiography; however it is NO substitute for poor radiographic technique and positioning.

PREPARATION

Equipment

Direct digital radiographic unit, x-ray generator, handle, lead gowns including thyroid protector, lead gloves or arm shields, wooden sleeper foot blocks (dimensions: 8cm high x 20cm wide x 30cm long), radiation badge.
A UPS (uninterruptable power supply) or battery backup is also something that can be useful particularly on properties which have unreliable power supply.

The Sales catalogue or a list of yearlings being x-rayed is needed so that all necessary information can be placed into the DR unit prior to commencing x-raying.

Microchip scanner is also needed so as to confirm the identity of each yearling prior to it being radiographed.

**Radiographic area**

The chosen radiographic area should be level and should not have bedding, shavings or loose dirt on its surface. This will allow the x-ray plate to be placed in a more relatively distal position, enabling inclusion of the pastern joint as well when acquiring the views of the fetlock.

**Horse cleaning**

Horse limbs need to be completely clean and dry, as both dirt and wet hair will cause artefacts on the radiographs. Front feet need to be picked out and the hoof wall cleared of dirt (a metal brush is useful to remove built up grit).

**Sedation**

The yearling is ALWAYS sedated prior to the radiographic exam. A combination of an alpha 2 agonist and an opioid appears to be the best combination. For example 0.4ml detomidine and 0.4ml butophanol is usually adequate for a 400kg thoroughbred yearling. Over sedation of a yearling can cause as much difficulty as under sedation as increased swaying can lead to poor radiographic quality due to motion.

**Order of radiographs**

All direct digital radiography machines can create a personal list of radiographs in a program to suit each individual veterinary. The program list and continuous mode i.e. ‘rapid fire’ can be selected which will allow the following radiograph to be selected automatically once the previous image has been acquired. Once this order is created it will allow the radiographs to be taken more quickly as apposed to having to individually choose the next view.
ORDER OF REPOSITORY RADIOGRAPHS AT SCONE EQUINE HOSPITAL

1. Right front foot
2. Left front foot
3. Left front lateral oblique fetlock
4. Left front DP fetlock
5. Left front medial oblique fetlock
6. Left front flexed lateral fetlock
7. Left flexed lateral carpus
8. Left lateral oblique carpus
9. Left medial oblique carpus
10. Right front lateral oblique fetlock
11. Right front DP fetlock
12. Right front medial oblique fetlock
13. Right front flexed lateral fetlock
14. Right flexed lateral carpus
15. Right lateral oblique carpus
16. Right medial oblique carpus
17. Right hind lateral fetlock
18. Right hind lateral oblique fetlock
19. Right hind DP fetlock
20. Right hind medial oblique fetlock
21. Right medial oblique hock
22. Right DP hock
23. Right lateral hock
24. Right lateral stifle
25. Right caudal lateral cranial medial oblique
26. Right caudal cranial stifle
27. Left caudal cranial stifle
28. Left caudal lateral cranial medial oblique
29. Left lateral stifle
30. Left lateral hock
31. Left DP hock
32. Left medial oblique hock
33. Left hind medial oblique fetlock
34. Left hind DP fetlock
35. Left hind lateral oblique fetlock
36. Left hind lateral fetlock

Labelling/data and display of images

With direct digital radiography units this can all be preset and will be displayed by the computer automatically. They need to include the dam, year, microchip number, lot number, sex, brands, date of birth, vendor and clinic/vet. It is worth while taking the time to set up your machine with labels as it will save you time in the long run. Labels should be placed on the lateral or dorsal/cranial aspect of the limb. Radiographs should also be submitted to the repository abiding by the standard international hanging protocols i.e. cranial or lateral to the left. Again if the imaging plate is always placed in the same way for a particular radiographic view, then the acquisition software for that particular view can be programmed so the orientation is correct for standard hanging protocols so as images don’t have to be re-orientated each time.

POSITIONING TIPS

General

For all views the horse should be standing square with weight evenly distributed through all four limbs. Attempts to adjust the angulation or elevation of the radiographic primary beam to compensate for incorrectly positioned limbs will result in inconsistent radiographs. The plate should be held against the limb or on the ground so as to reduce the likelihood of motion artefact. The radiographic beam needs to be perpendicular to the plate to avoid distortion or stretching of the image. It is important to not only adjust the position of the primary beam but also the position of the plate as well.

Exposure factors will vary between machines so these again will have to be adjusted to the individual and are worthwhile assessing prior to embarking on numerous yearling radiographs.

Film focal distance is generally approximately 70 cm. This can be standardized by the use of the laser lights on the x-ray generator. There is a small level on most of the x-ray generators but I recommend you get a larger one from the hardware store and stick to the top of the machine to help maintaining a horizontal x-ray beam.

Collimation is important and should still be carried out when using a direct digital radiography machine. Generally collimation occurs laterally and medially but not proximal and dorsally as this allows for more to be evaluated on the image.
**Foot**

Both front feet should be placed on wooden blocks (dimensions: 8cm high x 20cm wide x 30cm long). The hoof is placed on the block so as the medial wall is flush to the inside edge of the block. The primary x-ray beam is horizontal and directed at the caudal heel bulbs. All of the dorsal hoof wall and toe should be included in the view.

**Fetlock**

Plate positioning is important when radiographing the fetlock. Placing the plate on the ground parallel to the pastern axis and tucking it underneath the heel, allows both the fetlock and pastern joint to be evaluated. On the oblique and dorsopalmar/plantar views the primary beam is tilted down (proximal to distal) to allow elevation of the sesamoids so as the fetlock joint can be visualised. For hind limbs and yearlings that have very upright conformation less proximal to distal elevation is required. This is the opposite for yearlings with sloping pasterns where more elevation is required to lift the sesamoids up to allow visualization of the fetlock joint space. When taking lateromedial views of the hind fetlocks it should be noted that horses naturally stand slightly toe out behind and so often the radiographer needs to go more caudal to be perpendicular to the fetlock. In the flexed lateromedial view of the fetlock the plate holder is very important. The limb is held at the foot and the fetlock needs to be sufficiently flexed by pushing the limb cranially. It is useful if there is a second person to push on the dorsal aspect of the cannon to block forward motion and to encourage flexion.

**Carpus**

In the flexed lateromedial view of the carpus again the plate holder is very important. The carpus should be approximately two thirds flexed and the limb should be held directly underneath the horse. If the limb is held out or further underneath the horse you will generally oblique the view (proximal to distal). The medial oblique view of the carpus is taken from the lateral aspect of the horse. It is quicker to take the view this way as you do not need to move the limb prior to taking the shot. I line up perpendicular to the heel bulbs and then move one step towards the palmar or caudal aspect of the horse to ensure the right medial to lateral obliquity.

**Tarsus**

Horses naturally stand slightly toe out behind and so again just like the lateromedial view of the hind fetlock, the lateromedial view of the hock requires the radiographer to go more caudal to be perpendicular to the hock. The hock joints slope from proximal to distal from lateral to medial so if you aim lower down at the small tarsal bones then you will need to compensate by angling the beam DOWN slightly (2-3⁰) from proximal to distal.
**Stifle**

When radiographing the stifle we use a second person to hold the horse’s tail and push on the hip so as to stabilize the horse and reduce motion artifact. These images need to be magnified so as to evaluate the radiograph for movement – look for trabecular pattern and sharpness of bone edges. When taking the caudo cranial view the focal distance is reduced to 10-20cm. Plate position is also important for this view. The plate is placed on the cranial aspect of the stifle and held vertically with the distal aspect of the plate off the tibia. This allows a semi ‘air gap’ effect reducing the amount of scatter reaching the plate.

**POST PROCESSING PROCEDURES**

Check that there are 36 radiographs and discard those that are non-diagnostic and repeat.

QA (quality assurance) images to improve factors such as brightness and contrast this will avoid sending in radiographs to the repository that are overexposed i.e. too white (in regards to digital radiography images)

Crop images to remove other limb etc from image

Within the foot view increase the cropped image to show part of block so all of the sole can be evaluated

Send images to sales company no less than 4 days prior to the first day of Sales

**References**

1. Bladon BM and Main JPM. Clinical evidence in the evaluation of presale radiography: are we a desert on a horse with no name? Equine Vet J. 2003;35(4):341-342
3. Garret, KS and Berk, JT. How to properly position thoroughbred repository radiographs. AAEP Proc 2006 52:600-609
Pre-purchase radiography – a surgeon’s and vendor’s perspective

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Pre-purchase examinations make up a significant proportion of many equine practitioners’ work load. As part of these examinations it has become increasingly common to be asked to acquire and interpret radiographic images of horses being sold. A specialised type of pre-purchase radiography which has become common in this country is the radiograph repository system associated with thoroughbred sales.

In a clinical setting a veterinarian examines a horse and then makes a judgement whether radiographs will aid in making a diagnosis. In the pre-purchase situation, the practitioner will often be called upon to radiograph or examine radiographs of regions of horses which are asymptomatic or which they have not examined. Interpreting these radiographs becomes challenging because the purchaser wishes to know whether the variations from a perceived normal are likely to affect the horse over time.

There are many studies which have attempted to correlate radiographic findings with performance and lameness in the sport horse world. For example one study of 91 horses with lameness which blocked to the small tarsal joints showed a poor relationship between radiographic findings and lameness (Byam-Cook and Singer 2009). This poor association of radiographic signs in the small tarsal joints and lameness has been reported in another study (van Hoogmoed, Snyder et al. 2003). This study did report better correlation of higher grade radiographic signs and lameness in the navicular and distal phalanx bones however. There are few large scale studies which prospectively have followed sport horses reporting the correlation of pre-purchase radiology findings and performance or soundness. This presents the clinician considerable challenges and ultimately in practice decisions are made based in part on evidence in the published literature and partly on each clinicians own experiences.

Since 2003 when a repository system was introduced into Australia, it has become common for weanling, yearling and some 2 year old thoroughbred horses to be subjected radiography prior to sale. Most thoroughbred sales in Australia now have a repository system and vendors may submit a standard series of 36 views which must be taken within 6 weeks of the sale. Purchasers can then engage a registered veterinarian to view the radiographs either at the repository at the sale grounds or often via an online system.

There are many challenges in obtaining fair outcomes for all parties in this system. Often, clinicians are requested to view a set of radiographs without having the opportunity to examine the horse. The purchaser in these situations generally wishes to have a judgement on what the significant radiographic
abnormalities are and the likelihood these will impact on resale, soundness and performance. There have been many studies published which attempt to provide evidence upon which these decisions can be made. (Kane, McIlwraith et al. 2003; Kane, Park et al. 2003; SPIKE-PIERCE and Bramlage 2003; Jackson, Whitton et al. 2009; Preston, Brown et al. 2012). A striking feature of many of these and other studies is the poor correlation of pre-purchase radiographic findings and performance in most cases. Therefore it is likely many decisions which will have large implications for the sale price of these horses are based largely on the experience of clinicians and have little evidence based support.

From a vendors perspective there are many hurdles when selling horses. Pre-purchase radiography, while common, has become potential confounder introducing uncertainty into the selling process. There remains a lack of evidence upon which to make decisions and while this remains there will considerable variation in the opinions of clinicians on the significance of lesions detected. While a clinician is generally engaged to provide an opinion on a horse or radiographs of a horse for one purchaser, the reality is that these opinions often leak out into the wider market. This can adversely affect sale prices even though there may be no consensus amongst clinicians regarding the significance of findings.

From a surgeons perspective there has become a strong trend to screen horses before they are subjected to pre-purchase radiography. Decisions are then commonly made based not just on whether radiographic findings are likely to affect a horse’s welfare, performance or long term soundness but also on the appearance of radiographs and whether this will likely affect sale value. To further confound the issue, it is known many lesions may heal or improve spontaneously (Jacquet, Robert et al. 2013) yet often there is insufficient time available under industry conditions to allow lesions to spontaneously heal. This may result in surgeries which are purely for cosmetic reasons and the industry should question the validity of this practice.

Imaging Panel

Matthew Stewart, Tias Muurlink and Ben Mason

A collection of cases will be presented by the members of the imaging panel to generate case discussion among conference attendees and members of the imaging panel.
RESEARCH SESSION

Recent thoughts on Shivers.
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ABSTRACT

Shivering is a chronic, often gradually progressive movement disorder that usually begins before 7 years of age and has a higher prevalence in tall male horses.

Based on similarity of stride characteristics, horses with repetitive, dysmetric but non-ataxic limb movement disorders may be divided into groups as follows:

• Standing-HF: exaggerated hyperflexion with manual lifting of one or both hindlimbs. These horses walked forwards and backwards normally.
• Shivering-HF: hyperextension of hindlimbs when walking backwards, with normal forward-walking or intermittent mild hyperflexion.
• Shivering-HF: hyperflexion of both hindlimbs when walking backwards, with normal forward walking.
• Shivering-FHF: hyperflexion of both hindlimbs when walking backwards, with intermittent severe hyperflexion of both hindlimbs with forward walking.
• Stringhalt: hyperflexion of both hindlimbs occurring regularly during forward and backward walking.

Shivering affects backward walking, with either hyperflexion or hyperextension of hindlimbs, and can gradually progress to involve intermittent abducted hyperflexion during forward walking. Shivering-HF and Shivering-FHF can look remarkably similar to acquired bilateral stringhalt during backward walking; however, Stringhalt can be distinguished from Shivering-HF by hyperflexion during forward walking and from Shivering-FHF by an acute onset of a more consistent, rapidly ascending, hyperflexed, adducted hindlimb gait at a walk.

Detailed post-mortem examinations performed on 5 [WB & TB] Shivers and 3 [WB & TB] control horses revealed calretinin-negative, calbindin-positive, and glutamic acid decarboxylase-positive spheroids increased 80-fold in Purkinje cell axons within the deep cerebellar nuclei of horses with shivers.

The immunohistochemical and ultrastructural characteristics of the lesions combined with their functional neuroanatomic distribution indicate that shivers may be characterized by end-terminal neuroaxonal degeneration in the deep cerebellar nuclei, which results in context-specific pelvic limb hypermetria and myoclonia.
Further investigations using surface electromyography to characterize decomposition of multijoint movement in shivers horses and genome sequencing will be required to define the pathophysiology and aetiology of this highly unusual movement disorder.


The Synthetic Colloid Debate – Use them or Lose them?
Feary, D.J.

The use of synthetic colloids, specifically the hydroxyethylstarches (HES), has been a hot topic in human critical care medicine in recent years. The theoretical understanding of transcapillary fluid dynamics (the Starling equation) combined with positive results of large scale clinical trials in human medicine has resulted in widespread use of synthetic colloids in critically ill equine patients and during general anaesthesia. Two recent developments have challenged previous recommendations for colloid use in human medicine so that the balance of evidence is now against the use of synthetic colloids in most clinical situations.

Firstly, newer findings and modifications to the classic understanding of the Starling equation consider the effect of the endothelial glycocalyx layer and the no-absorption rule on transcapillary fluid movement, implying that improving plasma oncotic pressure is unlikely to reduce or prevent interstitial fluid accumulation or have a clinical effect on oedema formation. The rationale for the use of intravenous colloids has been questioned.

Secondly, a large body of literature supporting the use of HES in critically ill human patients published by one author have been retracted. The remaining studies, and results of recent meta-analysis, have failed to demonstrate a significant positive outcome effect with the use of synthetic colloids for fluid resuscitation and highlighted the increased incidence of adverse effects. The black box warning from the EMA that followed warned physicians not to use HES solutions in critically ill adult patients, and as a result many human hospitals throughout the world no longer use HES at all.

The applicability of these findings to veterinary patient populations is currently unknown. There is minimal safety and efficacy data pertaining to the use of synthetic colloids in veterinary clinical patients. From an equine veterinary point of view, evidence for clinically significant adverse effects of the available synthetic colloid solutions in horses is lacking. The clinical implications of the current colloid controversy are discussed in this session, but until appropriately sized clinical trials in horses are conducted, equine clinicians should carefully consider the choice of fluid in clinical situations where a colloid solution may be indicated.

Relevant literature

Radiological and ultrasonographic findings in horses with carpal sheath effusion—results from 122 cases

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Introduction

The flexor structures of the equine forelimb experience marked tensile load at high speeds. Most injuries occur at the mid-cannon area, ‘bowed’ tendons and suspensory desmitis; however, injuries of the superficial digital flexor muscle (SDF), deep digital flexor (DDF), their respective musculotendinous junctions (MTJ) and accessory ligaments (AL) at the level of the distal antebrachium, carpus and proximal metacarpus also occur, albeit, less frequently. These injuries can cause carpal sheath effusion (CSE), palmar to the carpus. This retrospective study was conducted to identify osseous and soft tissue injuries associated with CSE in horses.

Materials and Methods

Medical records from 121 horses with an ultrasonographic diagnosis of CSE were evaluated to document the incidence of osseous and soft tissue lesions and lameness at presentation. Ultrasonographically recognized lesions were localized to the distal antebrachium, the proximal metacarpus or both levels. Affected horses were categorized as young (<9 years of age), middle-aged (9-10 years) or older (> 18 years). Associations between case details and specific injuries or presenting signs were assessed using Chi square tests. Odds ratios were calculated to assess risk for lameness and specific injuries.

Results

CSE was diagnosed in a wide range of ages, breeds, and uses. Only 16 of the 121 CSE cases were racehorses, in stark contrast to the incidence of flexor tendon mid-cannon strain injuries. Most horses (89/121) were lame at presentation. Lameness was more prevalent in middle-aged and older horses than in young horses. Only eight cases were idiopathic, in that CSE was present in the absence of any other detectable lesion. Of the remaining 113 cases, osseous abnormalities were diagnosed in only 10 cases (seven distal radial osteochondromas, two distal radial exostoses, and one accessory carpal bone fracture), while soft tissue lesions were identified in 111 limbs (eight cases had both osseous and soft tissue lesions).

The majority of soft tissue injuries in these cases (93/111) were complex; involving two or more structures and extending from the caudo-distal antebrachium into the palmar metacarpus. The SDF/SDFT (98 cases) was the most commonly injured structure. In 20 of these cases, the lesion was restricted to the musculotendinous junction (MTJ) of the SDF. The accessory ligament of the superficial
digital flexor tendon (ALSDFT) was also frequently injured (64 cases); however, in all but seven of these cases, ALSDFT lesions were present in association with injuries to other structures.

Deep digital flexor (DDF) and DDFT lesions were less common, and were identified in 21 cases. In four of these cases, the DDF lesion was restricted to the MTJ. Lesions in the ALDDFT were recognized in 25 horses, but these injuries were associated with lesions in other structures in all but one case. Forty-two horses had ultrasonographic evidence of carpal sheath and/or retinacular damage. All these cases also had SDF/T and/or ALSDFT injuries. Forty horses had hyperechoic ultrasonographic patterns in the medial SDF MTJ consistent with fibrotic substitution. Injuries to the DDF and ALSDFT were also common in these cases, suggesting chronic/recurrent strain injuries to the flexor apparatus in these cases.

Older horses were significantly more prone to SDF (middle-age 2.88 times, older 5.05 times) and SDFT injuries (middle-age 6.96 times, older 8.99 times) than the ‘young horse’ group. Age was also significantly linked to SDF medial fibrosis. Middle-aged and Older horses were 10.67 and 15.0 times, respectively, more at risk for this condition than young horses.

**Discussion/Conclusions**

The results of this study highlight the fact that the large majority of CSE cases are not associated with osseous injuries. Skeletal lesions accounted for less than 10% of the cases. Further, although lesions in the SDF/T were identified in almost 90% of the cases, most horses had complex injuries, with ultrasonographic lesions in other structures, particularly the ALSDFT. This spectrum of pathology suggests that hyperextension strain/sprain injury of the proximal flexor structures affects all load-bearing structures to some extent. The significant association with age strongly suggests that cumulative strain injury at and around the flexor MTJs occurs in horses, somewhat analogous to Achilles tendinopathy in people. Medial fibrosis of the SDF MTJ in 40% of these cases was a new putative ultrasonographic diagnosis that was primarily identified in older horses. The relationship between this finding and pathology in other flexor structures will require further investigation. A comprehensive assessment of all soft tissue structures in the distal antebrachium, carpal canal and proximal metacarpus is indicated in all horses presenting with CSE.

**Acknowledgements**

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Stapled side-to-side gastrojejunostomy as a treatment for gastric outflow obstruction in two adult horses.

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Introduction

Primary gastric disorders are relatively uncommon in adult horses, however they include gastroduodenal ulceration, anterior enteritis, adhesions and stricture formation, fibrotic masses within the pylorus and gastric impaction. Surgical access to the equine stomach in the adult horse is somewhat limited, however, reported surgical procedures include, gastroduodenostomy and gastrojejunostomy. This article describes the diagnosis, surgical treatment and outcomes in two adult horses with gastric outflow disorders secondary to mural thickening and fibrosis of the pylorus and or proximal duodenum.

Materials and Methods

A retrospective analysis of clinical records of adult horses undergoing exploratory laparotomy and gastrojejunostomy for gastric outflow disorders were reviewed and cases that survived for 6 months or more were included. Two cases met the inclusion criteria during a 3 year period.

Results

Two adult horses with histories of moderate abdominal pain and anorexia were diagnosed with delayed gastric emptying at gastroscopy, despite being fasted ≥ 48 hours. Exploratory laparotomy was required to confirm the diagnosis of gastric outflow obstruction secondary to pyloro-duodenal masses. A stapled side-to-side gastrojejunostomy was performed in both horses. One horse was euthanized 6 months post-surgery due to recurrent abdominal pain and the other horse survived ≥ 3 years.

Relevance to equine clinical practice

Gastric outflow disorders are relatively uncommon in adult horses. Clinical signs of anorexia, mild to moderate persistent abdominal pain and delayed gastric emptying at gastroscopy should alert equine clinicians to a possible gastric outflow obstruction. Exploratory laparotomy is required to rule out other causes of abdominal pain and to evaluate for pyloro-duodenal obstruction. Surgical access to the equine stomach in the adult horse is limited, however stapled side-to-side gastrojejunostomy is achievable and is a viable surgical option for the treatment of gastric outflow disorders in adult horses. While it would seem the prognosis for medium term survival of adult horses with confirmed gastric outflow obstruction and subsequent side-to-side gastrojejunostomy is fair, larger case numbers are required to provide more meaningful data on long-term survivability.
Effect of thermal cautery of the soft palate on racing performance in horses with suspected and diagnosed palatal dysfunction

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Introduction

Palatal dysfunction (palatal instability, PI and dorsal displacement of the soft palate, DDSP) is commonly recognised as a cause of upper airway obstruction and poor performance in racing Thoroughbred (TB) and Standardbred (SB) horses. A number of treatments have been advocated, including thermal cautery which aims to increase tension in the soft palate. This study aims to investigate the effect of this procedure on racing performance.

Materials and methods

Clinical records were used to identify TB and SB racehorses that had undergone thermal cautery from 2009 – 2015. Method of diagnosis, surgical details and time from surgery to first start were recorded. Horses, which underwent concurrent laser ablation of the aryepiglottal folds were included. However, those that underwent other forms of upper airway surgery were excluded from the study.

Race records before and after surgery were analysed and race performance based on earnings and a performance index were calculated for 1-5 races pre- and post-surgery. Overall success rate was determined, with horses considered to be improved if their earnings and points per start had increased post-operatively.

Results

Twenty four horses (21 TB and 3 SB) met the inclusion criteria. Ten horses (2 SB and 8 TB) had a definitive diagnosis made during exercise endoscopy, whilst 14 received a presumptive diagnosis based on clinical history and resting endoscopy. All horses had raced before surgery. Twenty three (96%) horses returned to racing and had at least two post-operative starts. One horse underwent a second thermal cautery procedure. Standardbreds returned to racing significantly sooner than TB (mean = 47d vs 139d; \textit{p} = 0.015). No adverse effects were reported.

There was no significant difference in performance post-operatively between horses with a presumptive or definitive diagnosis or between horses of different age, sex and breed. There was a significant improvement in performance index for the first 3 starts (\textit{p} =0.02) and in both earnings (\textit{p}=0.02) and performance index (\textit{p}=0.03) for 5 starts post-surgery, compared with the last pre-operative start.
Overall, 60% of horses had improved performance for 3 starts, and 65% of horses had improved performance for 5 starts, post-thermal cautery.

**Relevance to clinical equine practice**

Thermal cautery has a similar success rate to other soft palate surgeries. The procedure is quick, cost effective, minimally invasive and allows a rapid return to racing.
**Haemothorax in two Thoroughbred racehorses.**

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**Introduction**
Haemothorax is a clinically important (although uncommon) condition in horses of all ages and has a wide range of aetiologies. The most common aetiologies are associated with trauma, neoplasia, coagulation disorders, vessel ruptures and iatrogenic causes (Groover and Wooldridge 2013). Haemothorax is diagnosed by detection of increased pleural fluid via ultrasound. Thoracocentesis confirms the hemorrhagic nature of the fluid. The two cases described illustrate acute haemothorax after strenuous exercise without any apparent trauma.

**Case studies**
Two Thoroughbred racehorses aged 4 and 2 years old, presented for investigation of intermittent pyrexia, inappetence, lethargy and declining packed cell volume, after trialling and racing. Antimicrobials were administered prior to referral.

Initial clinical examination was normal, other than dull mentation and elevated rectal temperature of 39.5 degrees. Case 2 presented with soft to absent lung sounds evident ventrally on both the left and right sides of the thorax. There was no evidence of rib fracture or trauma in either case.

Pleural fluid was detected in both horses via thoracic ultrasound. Thoracocentesis confirmed haemothorax. In both cases the described pleural fluid was a neutrophillic-macrophagic exudate, with an elevated white cell count and evidence of previous haemorrhage. No infectious agents or cytological evidence of neoplasia was seen on cytological examination.

**Clinical outcome**
In both cases, management consisted of indwelling thoracic drain placement and broad spectrum antimicrobials, Procaine penicillin (22mg/kg IM BID), Gentamicin (6.6mg/kg, IV SID), Metronidazole (15mg/kg PO TID) and flunixin meglumine (0.5mg/kg IV BID).

They were discharged from hospital 8 and 4 days after initial presentation, with antimicrobials to be continued for a further 4 weeks at home. Currently the horses are doing well with no further evidence of pulmonary pathology reported.

**Relevance to clinical equine practice**
The above cases demonstrate that haemothorax is a differential diagnosis for pyrexia and anaemia after strenuous exercise. The diagnosis is reliant upon ultrasonography and thoracocentesis. We propose that haemothorax may be an unusual manifestation of EIPH. Although thoracic drainage is not necessary
for resolution of haemothorax (Gillian, Dorothy, Ainsworth et al. 1999), drains were placed in both cases due to evidence for secondary bacterial infection of the pleural fluid. Horses affected by haemothorax may be successfully returned to racing, although clients should be prepared for a convalescence period of at least 3 months.

References


Dowling, B.A.\textsuperscript{a}, Williams, S.\textsuperscript{a}, Raffetto, J.\textsuperscript{b}, Wearn, J.\textsuperscript{b}, Ruppin, M.\textsuperscript{c}, Condon, F.\textsuperscript{c}, Spelta, C.W.\textsuperscript{a}, Stone, B.\textsuperscript{d} and Sandy, J.\textsuperscript{d}.

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Introduction

Primary disorders of the equine temporomandibular joint (TMJ) are relatively rare, but include subluxation, trauma, sepsis and osteoarthritis. The clinical signs attributed to TMJ disease in horses range from localized swelling, discharging sinus tracts, dysphagia, weight loss, masticatory muscle atrophy, head shaking and behavioural abnormalities. Due to the varied nature and insidious onset of clinical signs associated with TMJ disease, pathology is often advanced by the time a definitive diagnosis is confirmed. As such the response to conservative management is reported be variable. The surgical approach to mandibular condylectomy with or without meniscectomy has been described as a viable treatment option for horses with advanced TMJ osteoarthritis. This report describes the clinical findings, novel technique for unilateral mandibular condylectomy and outcome of an adult horse diagnosed with chronic osteoarthritis of the temporomandibular joint.

Case Report

A 5 year old Quarter Horse mare was presented for evaluation of localized firm swelling over the right TMJ, incisor malocclusion and behavioural problems when ridden with a bit. The horse had sustained trauma to the right TMJ region 15 months previously. Clinical examination revealed moderate right sided masseter and temporalis muscle atrophy, left sided deviation of the mandibular incisors, abnormal molar wear and reduced lateral and ventral mandibular range of motion. Radiographic examination of the right TMJ revealed periartricular osteophyte formation and subchondral lysis of the right mandibular condyle and temporal bone consistent with osteoarthritis of right TMJ. Under general anaesthesia, a modified technique was utilized to perform a unilateral mandibular condylectomy and meniscectomy using a gigli wire saw passed around the neck of the mandibular condyle with aid of a Deschamps needle. Recovery from anaesthesia was uneventful and the horse was discharged from hospital. Follow up at six months after surgery identified the horse to be in good body condition, with absent trismus, no pain evident on jaw distraction and markedly improved right sided masseter and temporalis muscle mass.
Discussion

The clinical signs of primary TMJ disease may be insidious in onset such that at the time of diagnosis the disease is often chronic with advanced pathology. It is generally thought that the pain associated with mastication and or ridden exercise accounts for the clinical signs observed in horses with TMJ disease. This novel surgical technique is believed to offer better surgical access to the mandibular condyle with less damage to surrounding tissue. Unilateral mandibular condylectomy and meniscectomy may offer a viable treatment option in selected cases with confirmed unilateral osteoarthritis of the TMJ. While it would seem that the medium term outcome for a horse following unilateral mandibular condylectomy and meniscectomy of the TMJ is good, larger case numbers are required to provide more meaningful data on long-term survivability outcome.
Psittacosis associated with exposure to equine foetal membranes
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Psittacosis, also known as ornithosis, is a systemic infectious disease caused by Chlamydia psittaci. It is characterised by fever, malaise, cough, myalgias and atypical pneumonia. Complications include myocarditis, endocarditis, hepatitis, disseminated intravascular coagulation reactive arthritis, and neurological abnormalities including cranial nerve palsy and meningitis. In November 2014, four cases of respiratory illness among staff and students at a veterinary school were reported to Health Protection NSW by the veterinary school through the Department of Primary Industries. The affected staff worked in the veterinary reproduction unit where the students were undertaking a veterinary reproduction rotation at the time. Active case finding, through notices at the university and interviewing of cases, identified a further case of atypical pneumonia at a local stud farm, where one of the students had worked. Through this outlier, a common exposure to the equine foetal membranes of a single mare, amongst all the cases, was established, generating the hypothesis that the membranes of this mare was the source of the outbreak. The foetal membranes subsequently tested RT-PCR positive for C. psittaci.

A team, coordinated by Health Protection NSW was established to conduct an investigation. A site visit to the stud farm and veterinary school was conducted and all persons exposed to the equine foetal membranes of the mare were interviewed using a standard questionnaire designed for the outbreak. A total of five cases of psittacosis were identified, with an attack rate of 56%. Two staff members were hospitalised. Direct contact with the foetal membranes and manipulation of the foetal membranes were associated with an increased risk of disease. There was no association between the hosing or scrubbing of floors and developing disease. Both cases that manipulated the membranes were subsequently hospitalised, indicating that a higher degree of exposure was associated with more severe disease.

This is the first study to demonstrate an association between exposure to abnormal equine foetal membranes and development of psittacosis.
Introduction & Abstract

Just describing all the manipulations that can be performed and observations that can be made when undertaking a neurologic examination doesn’t give the sense of efficiency and flow that is necessary to effectively perform neurologic examinations in practice. This presentation aims to share a format I use that gives a flow to the equine neurologic examination without detracting from the clinical dictum that “it is easy to look, but harder to see”: one has to know what one is looking for, to look for it, and to see it.

The primary aim of a neurologic examination is to confirm whether or not a neurologic abnormality exists. Because omission of parts is the most common mistake made during the neurologic examination, the order in which the examination is performed becomes important. I give here a precise practical format that is logical in sequence, easy to remember with practice, and emphasizes the need for an anatomic diagnosis - Where is the lesion? - to be made before an etiologic diagnosis - What is the cause of the condition? - is made. The rationale for the sequence of this examination is: firstly, it starts at the head and proceeds caudally to the tail; secondly, it is used for patients of all sizes and whether the patient is ambulatory or recumbent; thirdly, it considers the anatomic location of lesions as the examination proceeds. Even if parts of the examination must be omitted because of the nature of the patient, suspicion of fracture, or financial constraints, the sequence ought to be followed through mentally. Frequently, the presence of a neurologic lesion[s] cannot be deduced until the end of a thorough neurologic examination.

Table 1 gives an outline of the recommended format for neurologic examination of horses and an example of a recording form to record the results of the neurologic examination is given in Figure 1. Some comment as to differences important to recall when evaluating neonates also are indicated.

I encourage those readers who are not reasonably well practiced in performing neurologic examinations, and in recording the variety of responses obtained with direct observations, reflex testing and response induction in normal patients, to practice on a friendly, neighbourhood, mid-sized dog. The approach for such an examination will be used for young foals and miniature horses. Should the practice dog or a foal be small enough, the close aspects of the procedure used are readily performed by straddling above and behind the patient for restraint and comfort.

In actual fact what I do is to quickly examine the major regions [head, neck/FLs, trunk/HLs, tail/anus, gait & posture] deciding if, and in general terms where, a neurologic lesion(s) may be present. Then I go through a more thorough full examination emphasizing the suspect region(s), particularly remembering aspects that cannot be performed satisfactorily at that time, and those aspects that need to be re-evaluated later.
1. Head

For the routine equine examination, I evaluate what I can from a distance, preferably before the patient is disturbed, for the first observations of behaviour, mentation and head, neck, trunk and limbs. Head and neck deviations need to be assessed closely by straightening the neck along the midline to determine what asymmetry may be present. I allow the patient to smell my [often peppermint-tainted] hand for introduction and observe the face, particularly eyes and associated structures, for an expression response. Then I examine the parts of the head and neck for evaluation of cranial nerve function [Table 2].

At this stage I will make sure I have the patient’s attention by tapping lightly once or twice above the eye with my finger tips on a cupped hand to induce a combined visual and facial response of palpebral closure, proceeding to a menace response from nasal and temporal fields.

This is followed by observation of eye position and pupil size and symmetry using a bright pen torch from 40-50cm. Swinging the light quickly from the fundus of one eye to the other and pausing for about 3s at each pupillary aperture as the light source is brought closer in front of each eye, allows the immediate pupillary response to be observed, unencumbered by blinking. Any asymmetry or suspected deficit means that a dazzle response must be performed and the tests need to be reperformed in dim and in bright light, but not direct sunlight. With practice, the central fundus and optic disc can be directly inspected by looking directly along the shaft of light from a pen torch; otherwise an ophthalmoscope should be used. Evidence of optic atrophy, peripapillary retinal lesions, globe position and trembling and ataxic eye movements and nystagmus all can all be detected. Remember, the sun, and even bright daylight, is many, many times brighter than any penlight.

Eyeball position in the bony orbit, along with the size of the palpebral aperture and angle of the dorsal eyelashes then can be determined and both ventral movement and induced, normal, horizontal nystagmus of the globe then can be evaluated by first slowly raising the mandible to horizontal and then rotating the head to both sides through an arc of 60-90 degrees. Facial symmetry, reflexes, movement and especially muscle tone, all can then be observed as well as the bulk of the temporalis, masseter and pterygoid muscles being compared. During facial reflex testing with a blunt pair of needle holders sensation in the form of cerebral perception and resentment is evaluated from the nasal septum on each side. During this testing, any increased temperature and presence of sweat at the base of an ear will be evident. Nasal, oral, laryngeal, pharyngeal and hyoid region inspection and particularly palpation are performed and the thoracolaryngeal responses felt for.
2. Body

a. Neck & Thoracic Limbs

Attention is immediately moved to the neck where bone and muscle symmetry is assessed by close palpation and the local cervical and cervico-facial responses seen and felt. A solid blunt instrument such as 6-12 inch artery forceps or needle holders are best for performing this and other cutaneous testing although on occasion, with a very frightened patient, the tip of a rigid index finger may have to suffice as being more tolerated. A very firm vertical stimulus is required to be applied over sites at the level of and 10-15cm dorsal to the articular processes of cervical vertebrae.

b. Trunk & Pelvic Limbs

Testing can continue caudally to transfer to the thorax, testing the cutaneous trunci reflex over the mid region of the entire lateral thorax, again with forceful stimuli. There is a variable region over the point of the shoulder, about the C7-T3 dermatome region, where neither responses are obtained and the cutaneous trunci reflex usually fades in the caudal thoracic region. Flexion, extension and lateral bending of the thoracolumbosacral vertebral column then is conveniently tested with the instrument used to firmly stroke the lateral dorsum from the withers to the caudal gluteal region. Two-pincht technique is used to test dorsal dermatomes and limb autonomous zones for areas of hypalgesia if there is any evidence whatsoever of a reflex deficit or lower motor neuron spinal cord lesion. Regional loss of muscle bulk, bony asymmetry and areas of vascular engorgement or sweating also should invoke detailed scrutiny.

c. Rectum, Bladder, Anus, Tail

Coccygeal extension and flexion is evaluated during anal reflex and perineal sensory testing. A strong, blunt-probe stimulus applied to the anal ring results in its brisk contraction and flexion [clamping] of the tail, whereas a light stimulus results in an anal reflex and with a stroking, tickling stimulus results in tail extension.

3. Gait & Posture

The minimal components of evaluation of posture and gait consist of the following.

- Walking in straight line viewed from the side as well as from behind and in front.
- Pulling laterally on the tail with the patient standing still and while walking forward, assessing stride length and trajectory and placement of all four limbs.
- Trotting away from and back toward the examiner.
- Watching the patient walking in circles and turning tightly in circles in both directions.
- Taking the patient oneself and by walking backwards lead the patient in a serpentine path to observe limb placement and lead in a manner such that the direction of each forefoot in turn is required to change direction during its swing phases.
- Leading the patient to walk in circles and to turn tightly in circles in both directions, continuing these manoeuvres while pulling on the lead rope and the tail simultaneously assessing strength of resistance.
- Releasing the tail abruptly and stopping turning tightly to observe for adoption of and standing with abnormal limb positions. Manually placing the limbs in abnormal positions and placing them with the dorsum of the hoof resting on the ground have not proven consistently useful in detecting neurologic motor or proprioceptive abnormalities.
- Hopping the patient on at least the thoracic limbs.

Defining the presence and severity of gait and postural abnormalities [Table 3], along with an interpretation of evidence of upper and lower motor neuron abnormal reflexes and function [Table 4] can assist in determining the site(s) of lesions in the nervous system.

A patient that is or can be placed in recumbency can be tested for classical limb reflexes. In practical terms these simply consist of the flexor reflexes in thoracic and pelvic limbs and the extensor or patellar reflex in the pelvic limbs. A reflex is regarded as present or hyperactive in a limb if such a response is detected in the recumbent while the limb is uppermost OR is dependent. All other reflex testing really does not usefully contribute to a neuroanatomic diagnosis and results of such additional limb reflex testing should not be used to alter a neuroanatomic diagnosis achieved by interpretation of the remainder of the examination.

At the conclusion of the gait evaluation, any issue that is unclear can be returned to for further evaluation and confirmation, and further testing can be considered as appropriate. This will frequently include reassessing the menace and pupillary responses and nasal septal sensation, observing the patient for facial weakness and head deviation while it is resting quietly and undisturbed, blindfolding and, for the smaller patients, hemi-walking, hoping on pelvic limbs and wheel barrowing on thoracic limbs with head and neck held extended.

For documentation, further study and possible consultation purposes, taking a video of any possible neurologic signs displayed by a patient is worth considering. It must be recalled however that a badly produced video clip is likely to be worse than a verbal or written description. At best, video sequences of neurologic movement abnormalities, particularly involving the gait, are less precise and accurate than in real life.
Results of the neurologic examination should be documented and not left to memory [Figure 1].

After completion of the neurologic examination, the examiner may be able to decide if and where any possible lesion exists. There certainly are many syndromes described in various detail in which one suspects a neuromuscular lesion to account for the signs but none have been found to date [Table 5]. This may be because we haven’t looked in the right place for the morbid lesion, or there is a functional abnormality [e.g. channelopathy, transmitter defect] or a behavioural [hormonal] derangement.

Bibliography

### TABLE 1: Outline of recommended format for the neurologic examination.

<table>
<thead>
<tr>
<th>Region</th>
<th>Evaluation</th>
<th>Division</th>
<th>General Comment</th>
<th>Neonate</th>
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<tbody>
<tr>
<td>Head</td>
<td>Behaviour</td>
<td>Forebrain</td>
<td>History important</td>
<td>Adj. over 2-7d</td>
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<td>Seizures; esp mild and focal</td>
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<td>Mentation /</td>
<td>Thalamus</td>
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<td>Adj. - 24h</td>
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<td>Midbrain</td>
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<td>Head Posture &amp;</td>
<td>Physical causes</td>
<td>Head tilt verses head turn</td>
<td>Flexed head posture</td>
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<td>Movement</td>
<td>Forebrain – turn</td>
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<td>Jerky movements</td>
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<td>Vestibular – tilt</td>
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<td>Tremor – check eyeballs</td>
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<td>Cranial Nerves</td>
<td>CN II - XII</td>
<td>Evaluate regions of head</td>
<td>Menace deficit &lt;7d</td>
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<td>Brainstem</td>
<td>Subtle asymmetry in menace</td>
<td>Eye posture</td>
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<td>Cervical sympathetic supply</td>
<td>response &amp; nasal sensation</td>
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<td>Neck &amp; Forelimbs</td>
<td>C1-T2</td>
<td>Particularly asymmetry</td>
<td>All reflexes</td>
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<td>Flexor reflex only</td>
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<td></td>
<td>Trunk &amp; Hindlimbs</td>
<td>T1-S2</td>
<td>Particularly asymmetry</td>
<td>All reflexes</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L4 – femoral n.</td>
<td>Flexor &amp; patellar reflexes only</td>
<td>Hyperreflexia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L5 – cranial gluteal n.</td>
<td></td>
<td>Crossed extension</td>
</tr>
<tr>
<td></td>
<td>Rectum, Bladder,</td>
<td>S1-S5</td>
<td></td>
<td>Extensor thrust</td>
</tr>
<tr>
<td></td>
<td>Anus, Perineum</td>
<td></td>
<td>S1-2 is common fracture site</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tail</td>
<td>C01</td>
<td>Extension &amp; flexion</td>
<td></td>
</tr>
<tr>
<td>Gait &amp;</td>
<td>Orthopedic Problems!</td>
<td></td>
<td>Shoulder &amp; gluteal atrophy</td>
<td>SEPSIS!</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>common</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Possible analgesic trial</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Positional deficits</td>
<td>All CNS-PNS</td>
<td>Placing feet non-contributory</td>
<td>Prematurity</td>
</tr>
<tr>
<td></td>
<td>Extensor weakness</td>
<td>Brain stem, spinal cord, PNS</td>
<td>Especially LMN</td>
<td>Dominant extensor strength</td>
</tr>
<tr>
<td></td>
<td>Flexor weakness</td>
<td>Brain stem, spinal cord, PNS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ataxia</td>
<td></td>
<td>Spinal ataxia</td>
<td>Irregular position &amp; placement</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cerebellar ataxia</td>
<td>Hypermetria characteristic; F &gt; H</td>
<td>Normal to degree</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vestibular gait</td>
<td>Crouched posture</td>
<td>Wide based to degree</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Deliberate [predictable] stepping</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wide based, staggering gait</td>
<td></td>
</tr>
</tbody>
</table>

**ORTHOPEDIC PROBLEMS!**

- Shoulder & gluteal atrophy
- Common
- Possible analgesic trial

**SEPSIS!**

- Placing feet non-contributory
- Prematurity
- All reflexes
- Hyperreflexia
- Crossed extension
- Extensor thrust

- Brain stem, spinal cord, PNS
- Especially LMN
- Dominant extensor strength
FIGURE 1: NEUROLOGICAL EVALUATION

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Owner’s Name</th>
<th>Tel. No.</th>
<th>Animal’s Name</th>
<th>Species</th>
<th>Ref. Vet.</th>
<th>VTH Clinician</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

HISTORY:

PHYSICAL EXAMINATION:

NEUROLOGICAL EXAMINATION

HEAD

<table>
<thead>
<tr>
<th>Behaviour, seizures</th>
<th>Head Posture [tilt, turn]</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Head, Neck &amp; Trunk Movement &amp; Coordination</td>
</tr>
</tbody>
</table>

Mental Status

NEUROLOGICAL EXAMINATION

Cranial Nerves

<table>
<thead>
<tr>
<th>Eyes</th>
<th>LEFT</th>
<th>RIGHT</th>
<th>Vestibular/Ear</th>
<th>LEFT</th>
<th>RIGHT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Eye drop, normal/abnormal</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nystagmus; normal vestibular</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Abnormal</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Blindfold</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tongue</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strabismus; III, IV, VI, VIII</td>
<td>Mass &amp; use; XII, cerebrum</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Face

<table>
<thead>
<tr>
<th>Sensation; Vs, cerebrum</th>
<th>Voice; IX, X</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Muscle mass/jaw tone; Vm</th>
<th>Swallow; IX, X, cerebrum</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Ear, eye, nose, lip reflex; V-VII</th>
<th>Endoscopy</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Expression; VII, cerebrum</th>
<th>T-L reflex</th>
</tr>
</thead>
</table>

Sweating; Symp.

GAIT & POSTURE

<table>
<thead>
<tr>
<th>Paresis</th>
<th>Thoracic limb &amp; Neck</th>
<th>Pelvic Limb &amp; Trunk</th>
<th>Thoracic limb &amp; Neck</th>
<th>Pelvic Limb &amp; Trunk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ataxia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypometria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypermetria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posture</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postural Responses [hop, hemi, knuckle]</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lameness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

LOCALISING SIGNS [reflexes, sensation etc]

<table>
<thead>
<tr>
<th>Thoracic limb / Neck</th>
<th>Pelvic Limb / Trunk</th>
<th>Tail, Anus, Bladder &amp; Rectum</th>
</tr>
</thead>
</table>

ASSESSMENT

Site of lesion[s]

<table>
<thead>
<tr>
<th>General [circle]: cerebral</th>
<th>brainstem</th>
<th>peripheral CNs</th>
<th>cerebellum</th>
</tr>
</thead>
<tbody>
<tr>
<td>spinal cord</td>
<td>peripheral nerve</td>
<td>muscle</td>
<td>skeleton</td>
</tr>
</tbody>
</table>

Specific:

Cause of lesion[s]

PLAN

Diagnostic

Therapeutic

Prognostic

KEY: 0 = absent; 1+ = hyporeflexic; 2+ = normal; 3+ = hyporeflexic; 4+ = hyporeflexic & clonus
### TABLE 2: Assessment of cranial nerve function

<table>
<thead>
<tr>
<th>CRANIAL NERVE</th>
<th>MAJOR FUNCTION</th>
<th>REFLEX/RESPONSE/ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Olfactory</td>
<td>Sense of smell</td>
<td>Challenge</td>
</tr>
<tr>
<td>II Optic</td>
<td>Sensory for vision and light</td>
<td>Menace response  Pupillary light reflex  Swinging light test</td>
</tr>
<tr>
<td>III Oculomotor</td>
<td>Pupillary constriction  Extra-ocular muscles (medial)</td>
<td>Pupillary light reflex  Medial movement of globe</td>
</tr>
<tr>
<td>IV Trochlear</td>
<td>Extraocular muscle (dorsal oblique)</td>
<td>Ventrolateral rotation of globe</td>
</tr>
<tr>
<td>V Trigeminal</td>
<td>Sensory</td>
<td>Ear, eyelid and lip (facial) reflexes  Pain perception: head, nasal septum</td>
</tr>
<tr>
<td></td>
<td>Motor</td>
<td>Chewing, jaw tone, muscle mass (temporalis, masseter, pterygoid)</td>
</tr>
<tr>
<td>VI Abducens</td>
<td>Extra-ocular muscle (retractor oculi)  Extra-ocular muscle (lateral rectus)</td>
<td>Eyeball retraction (corneal reflex)  Lateral movement of globe</td>
</tr>
<tr>
<td>VII Facial</td>
<td>Motor to muscles of facial expression</td>
<td>Ear, eyelid and lip (facial) tone, reflexes, and movement  Facial symmetry</td>
</tr>
<tr>
<td>VIII Vestibular</td>
<td>Afferent branch of vestibular system</td>
<td>Head posture  Induced eyeball movement  Normal vestibular nystagmus  Normal gait  Blindfold test</td>
</tr>
<tr>
<td>Cochlear</td>
<td>Hearing</td>
<td>Response to noise</td>
</tr>
<tr>
<td>IX Glossopharyngeal</td>
<td>Sensory / motor to pharynx</td>
<td>Swallowing (observation and palpation)  Gag reflex (nasal tube)</td>
</tr>
<tr>
<td>X Vagus</td>
<td>Sensory / motor to pharynx and larynx</td>
<td>Endoscopy</td>
</tr>
<tr>
<td>XI Accessory</td>
<td></td>
<td></td>
</tr>
<tr>
<td>XII Hypoglossal</td>
<td>Motor to tongue</td>
<td>Tongue size and symmetry</td>
</tr>
</tbody>
</table>
TABLE 3: Gait and postural abnormalities present with neurologic lesion at different locations.

<table>
<thead>
<tr>
<th>LESION LOCATION</th>
<th>POSTURAL DEFICITS</th>
<th>PARESIS</th>
<th>ATAXIA</th>
<th>HYPOMETRIA</th>
<th>HYPERMETRIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrum</td>
<td>+++</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Brain Stem</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Vestibular</td>
<td>+++</td>
<td>O</td>
<td>++</td>
<td>++</td>
<td>O</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>++</td>
<td>O</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Spinal Cord / UMN</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Peripheral Nerve / LMN</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>(++)*</td>
<td>(++)*</td>
</tr>
<tr>
<td>Musculo-Skeletal</td>
<td>+</td>
<td>++</td>
<td>O</td>
<td>+</td>
<td>O</td>
</tr>
</tbody>
</table>

O = not usually expected
+ = mild if present
++ = usually present
+++ = quite characteristically present
* = usually only with selection sensory fibre involvement
TABLE 4: The common clinical features of Upper and Lower Motor Neuron Defects

<table>
<thead>
<tr>
<th>FUNCTION</th>
<th>DEFECT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>UPPER MOTOR NEURON</td>
</tr>
<tr>
<td>Paralysis [flaccidity]</td>
<td>Normo- to hypertonic</td>
</tr>
<tr>
<td>Muscle atrophy</td>
<td>None or disuse*</td>
</tr>
<tr>
<td>Muscle fasciculations</td>
<td>NOT present</td>
</tr>
<tr>
<td>Reflexes</td>
<td>Normo- to hyperactive</td>
</tr>
<tr>
<td>Resistance to tail-pull</td>
<td>At rest</td>
</tr>
<tr>
<td>While walking</td>
<td>Weak</td>
</tr>
</tbody>
</table>

* becomes evident quickly in proximal muscles with lameness and disuse

TABLE 5: Syndromes in which neurologic lesions may be suspected but usually not proven.

- Prominent toe dragging
- Intermittent & unusual lameness
- Shivering
- Stringhalt-like movements
- Hyperreflexic movements
- Prominent sinking with dorsal lumbar pressure
- Throwing to the ground when saddle applied
- Rearing violently when first ridden
- Extreme difficulty in rising
- Localized hypertonia & tremor
Differential diagnosis of ataxia and weakness; and distinguishing neurologic syndromes from lameness

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Introduction & Abstract

In a 2008 AAEP study, experienced equine practitioners agreed on the existence of lameness in the forelimbs and hind limbs by only 25% and 15% above chance, respectively! The 95% confidence interval for a single AAEP lameness score [grades 1 to 5] was +/- 1.5 grades! Thus if a horse showed a mild lameness of 1 grade, experienced practitioners would grade the severity as anything from 0 to 2.5 out of 5! If we then add in degrees of neurologic gait abnormalities it is most likely that there will be even greater variability in grading the abnormalities. Indeed this is where some of the newer techniques of neuromuscular assessment such as magnetic motor evoked potentials [mMEP] (Nollet et al, 2008) and quantitative electromyography [Q-EMG] (Wijnberg et al, 2004) and more complex gait analyses (Emil Olsen, PhD, University of Copenhagen, pers. comm. 2013) may improve our clinical acumen. Notwithstanding, distinguishing orthopaedic from neurologic gait abnormalities is thwart with errors of interpretation. This paper cannot do justice to describing orthopaedic gait abnormalities (Ross, Dyson, 2011) but attempts to more clearly define the characteristics of neurological gait abnormalities that helps to distinguish them from gait changes resulting from orthopaedic disorders; of course both can occur together. The presence of cranial nerve abnormalities and changes in sensorium herald brainstem disease that may also have all forms of ataxia and demonstrate weakness, this distinguishing them from spinal cord and peripheral spinal nerve diseases. To follow on

Gait and Postural Abnormalities

Musculoskeletal and painful disorders causing gait and postural disorders are far more commonly encountered in horses than those involving just the nervous system [Figure 1]. Thus, two observations to be made when evaluating gait and posture are firstly, which limbs are abnormal, and secondly, is there evidence of lameness suggesting a musculoskeletal or painful cause for any gait abnormality?

The essential components of neurologic gait and postural abnormalities are paresis and ataxia and considerable effort needs to be made to define the presence, characteristics and severity of these findings when evaluating patients suspected of suffering neurologic disorders. In my experience, the most frequent mistake made in observing for, describing and teaching about characteristics of gait and postural abnormalities are failure to describe accurately what is seen before interpreting what any abnormalities might represent. Thus, we do not see ataxia, weakness, lameness and pain, but we do see irregular and unpredictable foot placement, toe dragging, head nodding and abnormal postures that can be interpreted as such.

Paresis or weakness can be defined as poor ability to initiate a gait, to maintain a posture, to support weight of the body or its parts, and to resist gravity. Often it can be determined that such weakness is predominantly involving extensor or flexor muscle groups, or both. As a generality, extensor, anti-gravity weakness is most indicative of lower motor neuron paresis. This is often seen as a bouncing
gait with short stride length, trembling, buckling and bunny-hopping, and a lowered neck carriage. This results in an apparent weight bearing lameness due to the exaggerated head nodding, particularly when it is asymmetric. For clarification, bunny-hopping is a non-specific gait characteristic, more often seen with musculoskeletal disorders, but when representing paresis reflects the added weight support offered when two limbs are used together.

In distinction, upper motor neuron paresis often is seen as a delay in initiating movement, therefore usually involving flexor muscles. Thus, a slow onset of the protraction or swing phase and a swinging, longer stride with decreased joint flexion [degrees of hypometria], are characteristics of upper motor neuron paresis. On this basis, with a prominent C6-T2 lesion involving grey and white matter in a smaller patient, one can see a very characteristic gait whereby there is a short-stride, bouncing gait in the thoracic limbs and a slower, long-stride with toe dragging in the pelvic limbs. This can be called a two-engine gait, with lower motor neuron, extensor paresis in thoracic limbs and upper motor neuron, flexor paresis in pelvic limbs.

In addition for observing for signs of abnormal gait and posture indicative of weakness, three most useful postural reaction tests for determining the presence of weakness in the limbs of a horse suffering from spinal cord disease are the tail pull, the tail and halter pull and thoracic limb hopping. Pulling the tail while the patient remains standing initiates an extensor, quadriceps contraction, mimicking performance of a patellar reflex. This reflex is poor when there is a lower motor neuron lesion at the level of L3-5 and therefore the patient will demonstrate weakness in resisting a tail pull while standing still as well as voluntary weakness while moving. In contrast, a wobbler horse with an upper motor neuron cervical lesion will have good resting muscle tone and be difficult to pull to the side in a singular movement while standing still, but is easily pulled to the side while walking. The first example demonstrates depressed extensor reflexes in the pelvic limbs while the wobbler demonstrates intact or even hyperactive extensor reflexes in the pelvic limbs in the face of voluntary, upper motor neuron weakness. The tail pull test is very useful in detecting extensor weakness in the pelvic limbs but often it is performed far too vigorously; it is not a contest between examiner and patient! It is best to apply constant lateral tension to the tail and determine what voluntary pull the patient exerts against that tension while it is weight bearing on the nearest limb.

The tail and halter pull test is performed by pulling on a lead rope and on the tail while circling the horse around the examiner and is testing a postural reaction that also evaluates voluntary strength. In addition it can exaggerate a patient's tendency to pivot on a hindlimb, thus demonstrating either flexor weakness or hypometria, and to manoeuvre limbs in an ataxic fashion. Again, ease in pulling the patient to the side during circling occurs because of weakness resulting from descending upper motor pathway involvement or a lesion that involves ventral horn gray matter level with the limb, or the peripheral nerves or muscles constituting a lower motor neuron lesion. With the latter, extensor weakness is often profound and it is easy to pull such a patient to the side while it is standing still and while circling. In
contrast, a weak animal with a lesion of the upper motor pathways usually can reflexly fix the limb in extension when pulled to one side by the halter and tail, whereas while circling, the patient does not have the voluntary motor effort necessary to resist the pull.

Hopping a patient laterally on one thoracic limb while the pelvic limbs are free to move may reveal that a horse is weak by a tendency for it to tremble on a thoracic limb when the opposite thoracic limb is held up to initiate the hopping test. Such a patient will also have difficulty in hopping to the side, and may stumble, when pushed away with the examiner's shoulder.

Flexor paresis often is evident when an animal drags its limbs, has worn hooves, and has a low arc and long swing phase to the stride. When an animal bears weight on a limb demonstrating extensor weakness, the limb often trembles and the animal may even collapse on that limb because of lack of support. While circling, walking on a slope, and walking with the head elevated, an animal frequently will stumble on a limb having extensor weakness and knuckle over at the fetlock.

With severe weakness in all four limbs, but no ataxia and hypometria, neuromuscular disease must be considered. However, tetraparetic horses will tend to tremble while standing still for a while and may seem agitated in movement while trying to lay down. They do not tend to drag all feet while walking as the extensor weakness is usually more prominent than the flexor weakness. Profound weakness in only one limb is suggestive of a peripheral nerve or muscle lesion in that limb. Weakness occurs with descending, upper motor neuron pathway lesions in the brainstem and spinal cord, and is present in the limb[s] on the same side and caudal to the lesion. A patient with peracute peripheral vestibular syndrome may appear weak in the limbs on the same side as the lesion because of the decreased extensor tone and tendency to fall in that direction, and the increased extensor tone in the contralateral limbs.

Ataxia is a term that, by its Greek derivative, means a lack of order or an inconsistency, and in this context is a proprioceptive dysfunction causing abnormal rate, range and force of movement and placement of the limbs and other body parts, including head, neck, trunk and even at times, the eyes. What the examiner must see to interpret as ataxia is irregular and mostly unpredictable movement and placement of the limbs, head, neck, or trunk. To accomplish this, the patient is observed while standing still, walking, trotting, turning tightly and backing up, and while the patient moves in a serpentine path with the head held elevated and while moving on a slope. The best way of accomplishing the latter manoeuvres is to walk backwards in a zigzag manner while holding the lead rope high to extend the patient’s head and neck. The aim is to alter the intended direction of the patient’s limbs while they are in protraction by turning the lead abruptly such that there must be a change in direction of each foot to be placed in the site the examiner intends for it to be placed. Some horses will not obligingly turn in tight or even large circles for examination. With practice, circling can be accompanied best by walking the horse forwards then start to turn in one direction slowly making the turn slightly tighter as the examiner moves from in front of the horse to level with the shoulder to level with the middle of the trunk, while coaxing the horse by flicking the rump with the free end of the lead rope. This way the
patient turns around the examiner, not the examiner around the horse. Essentially, these manoeuvres comprise the postural reaction tests for large animals. Thus, input to the upper motor centers is altered through changes in many modalities, including the visual horizon, vestibular stimulation and neck and limb proprioception that are synthesized into refined motor system signals. Subtle neurologic abnormalities, which may be compensated for under conditions of normal gait, are exaggerated during these manoeuvres. It is important for the examiner to observe the patient performing these manoeuvres from a distance and also to make the patient perform them oneself. Ataxic movements can be seen as irregular and mostly unpredictable foot flight and placement. To detect subtle asymmetry in limb protraction and the length of stride it can be useful to walk parallel to, or behind the animal, matching step for stride. An ataxic gait sometimes is most pronounced when an animal is moving freely in a paddock especially when attempting to stop abruptly from a trot or canter when the limbs may be wildly adducted or abducted.

Three descriptors are often used to identify the inconsistent movements that comprise ataxia. **Hypermétrie** is used to describe a lack of direction and increased range of movement, and is seen as an overreaching of the limbs with excessive joint flexion. Hypermétrie without paresis is characteristic of spinocerebellar and cerebellar disease. **Hypométrie** is seen as stiff or spastic movement of the limbs with little flexion of the joints, particularly the carpal and tarsal joints. This generally is indicative of increased extensor tone, and of a lesion affecting the descending motor, or ascending spinocerebellar pathways to that limb. A hypometric gait, particularly in the thoracic limbs, is seen best when the animal is backed up or when it is maneuverer on a slope with the head held elevated. The thoracic limbs may move almost without flexing and resemble a marching tin soldier. The short-stride, staggering gait seen with vestibular disease may be considered hypométrie. Also, movement of a limb with prominent flexor weakness can result in poor joint flexion and dragging of toes as with hypométrie but the movement and placement of the limb is relatively repetitive and predictable. Finally, **dysmétrie** is a term that incorporates both hypermétrie and hypométrie. Animals with severe cerebellar lesions may have a high stepping ataxic gait, but have limited movement of the distal limb joints, especially in thoracic limbs. This is best termed dysmetrie. In all these various situations we do need to take other abnormalities into consideration in defining the presence and characteristics of ataxia.

Ataxic movements are thus seen as a swaying from side to side of the pelvis, trunk, neck and sometimes the whole body. It may also appear as a weaving of the affected limb during the swing phase. Such abnormalities can be seen whilst an assistant manoeuvres the patient but also as one walks the horse with the head elevated and while pulling on the tail. The aim of the latter two manoeuvres is to change the direction of limb flight during mid-stride to promote errors due to proprioceptive abnormalities. This often results in abnormal foot placement in abducted or adducted positions, crossing of the limbs, or stepping on the opposite foot especially while the animal is circling or turning tightly. Any animal that is substantially ataxic for any reason tends to pace when walking with both feet on the same side.
off the ground at the same time. Circumduction of the outside limbs when turning and circling is also considered a proprioceptive abnormality. Walking an animal on a slope with the head elevated often exaggerates ataxia, particularly in the pelvic limbs. This manoeuvre also frequently allows expression of a hypermetric or hypometric component of ataxia in the thoracic limbs. When a weak and ataxic animal is turned sharply in circles, it leaves the affected limb in one place while pivoting around it. This may also occur when backing up.

Ataxia can also be classified into three syndromes by the quality of the signs seen and the functional system or pathway involved in the nervous system. These are general proprioceptive ataxia, cerebellar ataxia and vestibular ataxia, and after observing characteristics of a gait abnormality in a patient it is reasonable to attempt to define which of one or more of these syndromes are present.

**General proprioceptive ataxia** results from involvement of afferent proprioceptive pathways in sensory nerves and more commonly in spinal cord and brainstem tracts. Proprioceptive deficits are caused by lesions affecting the general proprioception sensory pathways, which relay information on limb and body position to the cerebellum for subconscious proprioception, and to the thalamus and cerebral cortex for conscious proprioception. The gait is irregularly irregular and most particularly is unpredictable. There is a delay in onset and a swaying or floating swing phase and subsequent variable foot placement exaggerated by manoeuvring the patient. This movement and placement may include adduction and abduction, and hyperflexion in hind limbs and hypoflexion or hypometria in forelimbs is common. General proprioceptive deficits likely contribute to scuffing toes and stumbling, especially on thoracic limbs. Obviously some of these signs are also associated with upper motor weakness, but because general proprioception and upper motor neuron tracts are adjacent in most parts of the central and peripheral nervous system, and involved in disease processes together, it is not necessary to distinguish which gait characteristics is due to dysfunction of one or the other.

**Cerebellar ataxia** can have characteristics of general proprioceptive ataxia but changes in limb placement and movement tend to be more abrupt in onset and excessive. The best definition of cerebellar ataxia being alterations in the rate, range and force of movement. Thus jerky onsets of movement and hypermetria are often seen, becoming more pronounced with more complex manoeuvres such as hurriedly regaining an upright posture from recumbency or abruptly turning to flee from being frightened. There is no upper [or lower] motor neuron paresis accompanying cerebellar disease but other signs of cerebellar involvement including head tremor and defective menace responses often are present. Signs of vestibular involvement also can be present with pan-cerebellar disease.

Concerning **vestibular ataxia**, although the limb movement and foot placement accompanying mild to moderate vestibular disease are irregular, and therefore can be called ataxic, they are somewhat less unpredictable. For example, if thoracic limb movement is forced to change in direction while the patient is lead with its head raised, the resulting correction will be predictably abducted. Also on turning a patient with mild vestibular disease, the wide movement and placement of an outside hind limb will not
usually be accompanied by hypermetria and any hurried movements to maintain a balanced posture will be strong and multiple, thus again somewhat predictable.

Normal horses react in different ways to blindfolding from extremes of excitement or distress to acting very calm and subsequent movements they make while blindfolded then often depend on this behavioural response. Vestibular ataxia and loss of balance often will be markedly exacerbated when a blindfold is applied to a horse suffering from vestibular or occasionally diffuse spinocerebellar and cranial cervical spinal cord disease. On the other hand, observing the posture and gait in response to blindfolding a horse suspected of suffering from typical mid to caudal cervical spinal cord compression usually does not add anything substantial to the neurologic evaluation. Damage to the sensory, C1-3 dorsal nerve roots can produce vestibular ataxia and this may be expected to exacerbate with blindfolding the horse.

Regarding assessment of posture and postural abnormalities, flexing the foot to attempt to make the animal stand on the dorsum of the pastern and determine how long the animal leaves the foot in this state before returning it to a normal position, is said by some to be a test for conscious proprioception in dogs and cats. Almost certainly this involves somatic afferent pathways as well and a very weak patient may not be able to move the foot from many abnormal positions. This test can be attempted in horses, but in my hands has not been helpful at all. Inactive and sleepy patients often allow the foot to rest on the dorsum for prolonged periods. Horses need to have almost total paralysis of the limb, or a nociceptive sensory deficit in the limb before they allow such postural anomalies to be accomplished. Other tests, such as manually crossing the limbs or placing one limb on a sack and slowly sliding the sack to the side, have been tried to test conscious proprioception but again in my hand have proven to by non-contributory to the examination process. Rather than manually placing limbs in abnormal positions, it appears more reliable to maneuver the horse rapidly, say in a circle, and stop the maneuver abruptly [Figure 1]. This often results in an initial awkward placement of the limbs and then the examiner can determine how long the horse leaves the limbs in such an abnormal posture to determine the presence or not of conscious proprioceptive deficits. This procedure probably does test for deficiencies in conscious proprioception. Examination of horses walking across kerbs has not proven to be a useful test of proprioceptive dysfunction. Normal horses, particularly if distracted, often will stumble and those that are moving cautiously, even if quite weak and ataxic, often can maneuver such obstacles.

Gait alterations can occur in all four limbs with lesions affecting the white matter in the caudal brainstem when head signs such as cranial nerve deficits are used to define the site of the lesion. Subacute to chronic lesions affecting the forebrain cause no substantial change in gait. However, postural reactions, such as hopping, are abnormal and sometimes the gait is slowly initiated on the thoracic limb contralateral to the side of a forebrain lesion.
In smaller patients, other postural reactions can be performed. These primarily help detect signs of subtle proprioceptive and motor pathway lesions when the gait is normal. Wheelbarrowing the patient to make it walk on just the thoracic limbs, hopping it laterally on each individual thoracic and pelvic limb and hemistanding and hemiwalking the animal by making it stand and then walk sideways on both left, then both right limbs, are three useful postural reactions to perform. Even in large, adult animals, particularly horses, it is possible to perform a modified hopping response test with the thoracic limbs. This is done by lifting each thoracic limb in turn while using the shoulder to make the horse hop laterally on the other thoracic limb. This test can help the clinician decide if there are subtle neurologic abnormalities in the horse’s thoracic limb control. Brainstem and spinal cord lesions appear to result in postural reaction deficits on the same side as the lesion, whereas cerebral lesions produce contralateral abnormalities.

At the conclusion of the examination, a most likely site of any acute nervous system lesion frequently can be defined accurately by determining the precise characteristics and severity of any gait and posture abnormalities present and the degree of weakness, ataxia, hypometria, hypermetria and conscious postural deficits should be graded for each limb [Table 1].

### Table 1: Prominent gait and postural abnormalities present with neurologic lesion at different locations.

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Gait and Postural Abnormalities</th>
<th>Postural Deficits</th>
<th>Paresis</th>
<th>Ataxia</th>
<th>Hypometria</th>
<th>Hypermetria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrum</td>
<td></td>
<td>+++</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Brain Stem</td>
<td></td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Vestibular</td>
<td></td>
<td>+++</td>
<td>O</td>
<td>++</td>
<td>++</td>
<td>O</td>
</tr>
<tr>
<td>Cerebellum</td>
<td></td>
<td>++</td>
<td>O</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Spinal Cord / UMN</td>
<td></td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Peripheral Nerve / LMN</td>
<td></td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>(++)*</td>
<td>(++)*</td>
</tr>
<tr>
<td>Musculo-Skeletal</td>
<td></td>
<td>+</td>
<td>++</td>
<td>O</td>
<td>+</td>
<td>O</td>
</tr>
</tbody>
</table>

O = not usually expected  
+ = mild if present  
++ = usually present  
+++ = quite characteristically present  
* = usually only with selective sensory fibre involvement
With peracute lesions, particularly those of an inflammatory nature and those with soft tissue compression of the spinal cord such as with caudal cervical arthritis and synovial cyst formation, resulting signs can wax and wane quite dramatically over periods of hours to days. Such signs usually stabilize with subacute to chronic lesions. In contrast, a horse suffering from chronic spinal cord disease may show quite different neurologic signs. For example, a horse that has suffered a single insult of cervical spinal cord compression a year prior to examination may have an unusual, perhaps hypermetric, mild ataxia in the pelvic limbs with no evidence of pelvic limb weakness and no signs in the thoracic limbs other than a questionably poor response to hopping. The anatomic diagnosis in such cases may be a thoracolumbar, cervical, or diffuse spinal cord lesion. A moderate or severe abnormality in the pelvic limbs, and none in the thoracic limbs, is consistent with a thoracolumbar spinal cord lesion. With a mild, and a severe change in the thoracic and the pelvic limb gaits respectively, one must consider a severe thoracolumbar lesion plus a mild cervical lesion, or a diffuse spinal cord disease. Lesions involving the brachial intumescence at C₆-T₂, with involvement of the grey matter supplying the thoracic limbs, and diffuse spinal cord lesions may both result in a severe gait abnormality in the thoracic limbs and the pelvic limbs. A severely abnormal gait in the thoracic limbs, with normal pelvic limbs, indicates lower motor neuron involvement of the thoracic limbs; a lesion is most likely present in the ventral grey columns at C₆-T₂, or thoracic limb peripheral nerves or muscle.

**Interpretation of Signs in Spinal Cord Disease**

**Neck and Forelimbs**

If a gait alteration was detected in the thoracic limbs and there were no signs of brain involvement, then this part of the examination can confirm involvement of the C₁-T₂ cervicothoracic spinal cord or peripheral nerves or thoracic limb muscles; it should also help localize the lesion within these regions.

Results of the thoracolaryngeal adductor response or slap test can be a useful part of the complete neurologic evaluation of horses suspected to be suffering from lesions of the vagal or recurrent laryngeal nerves, caudal medulla oblongata or cervical and cranial thoracic spinal cord. As most emphasis is placed on its utility in diagnosing cervical spinal cord disease in wobbler horses, some aspects of testing will be reiterated here. The test can be performed in co-operative horses by palpating the dorsal and lateral laryngeal musculature while simultaneously slapping the contralateral dorsolateral thoracic region from the cranial withers to near the last rib during expiration. If there is difficulty in interpretation of this test, observing the larynx via an endoscope while performing the test may be necessary. It should be emphasized that the thoracolaryngeal response is not consistently absent in horses with cervical spinal cord disease or caudal brain stem disease and can be absent in horses with no evidence of CNS disease. However, a reduced or absent slap reflex on the left side of the larynx must be taken as strong evidence for the presence of idiopathic recurrent laryngeal neuropathy or prior laryngeal surgery.
although treadmill exercise and endoscopic examination of the horse will be necessary to confirm any clinical problem of reduced laryngeal function and roaring. Bilateral absence of the response without other signs of severe laryngeal or cervicomedullary disease must be interpreted cautiously, particularly in an excitable horse. A normal response on the left side of the larynx and absent response on the right side most often indicates a neurologic disease other than classical idiopathic recurrent laryngeal neuropathy. The commonest cause of acute, acquired, severe bilateral laryngeal paralysis is hepatoencephalopathy.

Observation and palpation of the neck and forelimbs will detect gross skeletal defects, asymmetry in the neck and muscle atrophy. These signs may be associated with neurologic disease and thus be localizing findings. The neck should be manipulated to assess normal range of movement. Interpretation of what appears to be reluctance to move the neck passively or actively in any direction as indicating neck pain is fraught with difficulties. On the other hand, if a horse will not lower or bend its head to eat, drink or reach for a treat this usually indicates a mechanical or painful disruption to movement of the cervical vertebrae, particularly in the caudal neck. Cervical vertebral arthrosis, involvement of cervical nerve roots, and marked cervical spinal cord disease can cause scoliosis and even torticollis. Importantly, as musculoskeletal diseases are far more common than neurologic disease and as disuse atrophy can occur within at least weeks of onset of lameness, evidence of muscle atrophy, especially common over the scapula, should be taken as evidence of an underlying lameness until there is additional evidence that it is neurologic in origin.

Clearly delineated regions of cervical and thoracic sweating can be useful indicators of localized spinal cord or peripheral nerve disease in that they can represent sympathetic denervation or decentralization of the vasculature in the skin, resulting in increased circulating adrenalin stimulating sweat glands to secrete. Care must be taken in interpreting patchy sweating that is not well delineated. Very asymmetric patchy sweating can occur in horses that are excited or distressed, particularly when in a draughty box, without a specific sympathetic lesion being present. Involvement of the peripheral pre- and postganglionic sympathetic neurons in the horse result in localized sweating; this can be an extremely helpful localizing sign. Horner’s syndrome will result if the cervical sympathetic trunk is damaged. In the horse, dermatomal patterns of sweating on the neck and cranial shoulder occur with involvement of the C3-C8 branches of the sympathetic fibres. These arise segmentally from the vertebral nerve that follows the vertebral artery up the neck after the vertebral nerve has left the stellate ganglion near the thoracic inlet.

When the skin of the lateral neck of a horse just above the jugular groove from the level of the atlas to the shoulder is prodded firmly with a blunt probe, the cutaneous colli muscle contracts, which causes skin flicking. The sternocephalicus and brachiocephalicus muscles often contract also, causing the shoulder to be pulled cranially and even the head to be flexed laterally. This response tends to be less obvious in other species. In horses, there also is flicking of the ear rostrally, blinking of the eyelids, and
contracture of the labial muscles inducing a smile when the test is performed. Originally introduced by Rooney, these are termed the local cervical and cervicofacial responses, respectively. The precise anatomic pathways are not known, although they must involve several cervical segments and the facial nucleus in the medulla. Cervical lesions that involve grey and white matter can cause depressed or absent cervical responses however interpretation of abnormal responses may need to be expressed as imprecisely as for example "consistent with a caudal cervical lesion" or "consistent with a cranial cervical lesion". In contrast, the cutaneous trunci reflex can be very useful in delineating the precise cranial extent of a thoracic spinal cord lesion particularly when such a lesion is asymmetrical.

Sensory perception from the neck and forelimbs must be assessed. This can be difficult to evaluate accurately in stoic animals. Perception of a noxious stimulus is noted by observing the animal’s behavioural response while observing the local cervical responses and continuing the skin prodding over the shoulders and down the limbs to include testing the autonomous zones for the thoracic limbs [Figure 2]. As with any cutaneous sensory test, a two-step technique is recommended 25. This is accomplished by initially tenting and grasping a fold of skin between the jaws of heavy duty haemostats or needle holders. After pausing to allow the patient to settle, a second, sharp skin pinch is applied to determine superficial sensation. There may be reflex withdrawal of the part with or without a cerebral response, such as vocalization or moving the whole body away from the stimulus; the latter taken as representing conscious perception of the noxious stimulus.

If an adult horse has significant gait abnormality and it is feasible to cast it, then this should be done to assess the spinal reflexes. If the animal is ambulating well, it may be assumed that the spinal reflexes are intact. These reflexes can be studied in all smaller patients.

When evaluating wobblers with evidence of a neurologic abnormality in both thoracic and pelvic limbs and no evidence of brain disease, one should allow for a lesion to be present anywhere from C1 through T2. Conversely, when there is evidence of a mild neurologic gait abnormality in the pelvic limbs but not the thoracic limbs then the possibility of a lesion anywhere from C1 through S2 must be considered. If the signs of ataxia and/or paraparesis are moderate or even marked then a lesion can be considered anywhere in these segments especially C6 through S2. The reason to include lesions sites at C6-T2 is because such lesions, when intramedulary, can be very selective and spare tracts involving the thoracic limbs resulting in no definitive thoracic limb signs. Such has been the case in adult horses suffering from S. neurona myelitis, fibrocartilage thromboembolism, granulomatous meningoencephalomyelitis and migrating helminth parasites affecting C6-T2 spinal segments.
Trunk and Hindlimbs

If the examination of the head, gait and posture and neck and thoracic limbs reveals evidence of a lesion, then an attempt should be made to explain any further signs found during examination of the trunk and hindlimbs to have been caused by that lesion [Figure 2]. If there are only signs in the trunk and hindlimbs, then the lesion(s) must either be between T2 and S2, or in the trunk and pelvic limb nerves or muscles. This part of the examination helps localize such lesions more precisely. However, the examiner must remember that with a subtle, grade 1+ neurologic gait abnormality in the pelvic limbs, the lesion may be anywhere between the midsacral spinal cord and the rostral brainstem.

The trunk and hindlimbs must be observed and palpated for malformation and asymmetry. Lesions affecting thoracolumbar grey matter cause muscle atrophy, which is a helpful localizing finding. With asymmetric myelopathies scoliosis of the thoracolumbar vertebral column often occurs, initially with the concave side opposite the lesion. Once again, evidence of muscle atrophy, especially common over the gluteal region [Figure 3], should be taken as evidence of an underlying lameness until there is additional evidence that it is neurologic in origin.

Sweating in the horse over the trunk and hindlimbs, excluding the neck and face, can be a helpful localizing sign. Ipsilateral sweating caudal to the lesion signals involvement of the descending sympathetic tracts in the spinal cord caudal to T3. Lesions involving specific pre- or postganglionic peripheral sympathetic fibers that are second and third order neurons cause patches of sweating at the level of the lesion.

Figure 2: Autonomous zones for areas of desensitivity expected with individual peripheral spinal nerves to the limbs are not functioning [after Blythe, 1998].
Firm prodding of the skin over the trunk, particularly the lateral aspects of the thoracic wall, causes a contraction of the cutaneous trunci muscle, which is seen as a flicking of the skin over the trunk. The sensory stimulus travels to the spinal cord in thoracolumbar spinal nerves at the level of the site of stimulation. Transmission is then cranial in the spinal cord to C₈-T₁, where the lower motor neuron cell bodies of the lateral thoracic nerve are stimulated resulting in contraction of the cutaneous trunci muscle. Lesions anywhere along this pathway may cause suppression of the response, which is easiest to detect with an asymmetric lesion. In addition to this, an assessment of sensory perception from the trunk and hindlimbs must be made. This appears as a cerebral, behavioural response to a two-pinching stimulus described above, that includes assessment of the autonomous zones for the pelvic limbs [Figure 2]. Degrees of hypalgesia and analgesia have been detected caudal to the sites of thoracolumbar spinal cord lesions, but only when they are severe.

Stroking firmly with a blunt probe or pinching and pressing down firmly with the fingers over the thoracolumbar paravertebral muscles causes a normal animal to extend into a slightly lordotic stance and fix the thoracolumbar vertebral column. It also resists the ventral motion and usually does not flex the thoracic or pelvic limbs. Continuing this stimulus to the dorsal sacral region results in a degree of flexion and a kyphotic stance. A weak animal usually is not able to resist the pressure by fixing the vertebral column and thus it over-extends or over-flexes the back and begins to buckle in the pelvic limbs. Prominent back pain can result in poor responses and evidence of pain perception by, say, a grunt from the patient.

**Recumbent Patient.**

Every effort should be made to help a recumbent patient stand and walk, unless there is suspicion of bone fracture. By so doing, one can learn as much or more about voluntary effort and lesion localization than one can from reflex testing. Heavy animals in particular should be moved early in the course of recumbency to avoid secondary problems like decubital sores, decreased blood supply to limbs and dehydration, which make evaluation difficult.
A patient that has recently become recumbent, but uses the thoracic limbs well in an attempt to get up, most likely has a lesion caudal to T2 – but there are exceptions [Figure 4]! If such an animal cannot attain a dog-sitting posture, the lesion is likely to be in the cervical spinal cord. If only the head, but not the neck, can be raised off the ground, there probably is a severe cranial cervical lesion. With a severe caudal cervical lesion, the head and neck usually can be raised off the ground, although thoracic limb effort decreases and the animal usually is unable to maintain sternal recumbency. Assessments of limb function must not be done while a heavy animal is lying on the limb being tested. Muscular tone can be determined by manipulating each limb. A flaccid limb, with no motor activity, is typical of a lower motor neuron lesion to that limb, but in heavy recumbent animals there can be poor tone and little observable voluntary effort in a limb that has been lain upon. A severe upper motor neuron lesion to the thoracic limbs at C1-C6 causes poor or absent voluntary effort, but there will be normal or sometimes increased muscle tone in the limbs. This is because there is a release of the lower motor neuron that is reflexly maintaining normal muscle tone from the calming influences of the descending upper motor neuron pathways. Interestingly, such a hypertonic paralysis in the pelvic limbs also can be seen with lesions between C6 and T2 if little or no grey matter is affected. A Schiff-Sherrington phenomenon of short duration, with excessive extensor tone in the thoracic limbs in the presence of good voluntary activity and normal reflexes, has been seen rarely in horses, and usually follows a cranial thoracic vertebral fracture.

![Figure 4: Heavy patients with various neuromusculoskeletal disorders can have difficulty rising. Such patients, especially ruminants and pigs that adopt a dog-sitting posture for several seconds to minutes while getting up, usually have lesions caudal to the thoracic limbs and T3. However adult ruminants are seen to rest in the field in such a posture without having an overt neuromusculoskeletal explanation. Also, occasionally patients such as this horse suffering from mild caudal cervical spinal cord compression caused by CVM may also adapt and maintain such postures. This particular horse also was a tongue sucker.](image)

Spinal reflexes are tested in the thoracic limbs. The flexor reflex in the thoracic limb involves stimulation of the skin of the distal limb with needle holders and observing for flexion of the fetlock, knee, elbow, and shoulder [Figure 5]. This reflex arc involves sensory fibres in the median and ulnar nerves, spinal cord segments C6 to T2, and motor fibres in the axillary, musculocutaneous, median and ulnar nerves. Lesions cranial to C6 may release this reflex from the calming effect of the upper motor neuron pathways and cause an exaggerated reflex with rapid flexion of the limb. The limb may remain flexed for some time and even show repetitive movements or clonus. Such lesions also may result in a crossed extensor reflex, with synchronous extension of the untested limb. This usually occurs only with severe and chronic upper motor neuron lesions. Thus, an animal affected by such a lesion may
demonstrate considerable reflex movement following stimuli, but usually will have little voluntary motor activity in the limbs being tested. A spinal reflex can be intact without the animal perceiving the stimulus and the latter must be observed for independent to the local reflex movement. Cerebral responses associated with perception include changes in facial expression, head movement and phonation. Conscious perception of the stimulus will be intact only as long as the afferent fibers in the median and ulnar nerves, the dorsal grey columns at C6-T2, and the ascending sensory pathways in the cervical spinal cord and brainstem are intact.

Figure 5: The three important spinal limb reflexes to perform on any patient that can be placed in lateral recumbency are the flexor reflexes in the thoracic [A] and pelvic limbs, and the extensor or patellar ligament reflex in the pelvic limb [B]. All other reflex testing can be problematic in interpretation and results do not change an anatomic diagnosis. Normal hyperactive reflexes and crossed extensor reflexes were present as expected in this normal neonatal calf. Reflexes should be tested in both pairs of limbs while uppermost and while dependant, the most prominent result being taken as real. Occasionally a particular reflex cannot be elicited in a normal patient, usually bilaterally.

Interpreting results of testing the tendon reflexes in the thoracic limbs is problematic and does not usually assist in defining the site of neurologic lesion, perhaps with the exception of neonatal animals. Also, patients with profound diffuse neuromuscular paresis can have reflexes that are at least present. However a general description of two of these reflexes follows, testing the remainder being superfluous. To perform the triceps reflex the relaxed limb is held slightly flexed and the distal portion of the long head of the triceps and its tendon of insertion is balloted with a rubber neurology hammer in smaller patients or a heavy metal plexor in larger patients while observing and palpating for contraction of the triceps muscle, which causes extension of the elbow. The triceps reflex involves the radial nerve for its afferent and efferent pathways and spinal cord segments C7 to T1. The triceps reflexes, although present, can be extremely difficult to demonstrate in heavy, adult, recumbent patients. The musculotendinous portion of the extensor carpi radialis muscle can be balloted to produce extension of the knee when the relaxed limb is held in a partially flexed position. This extensor carpi radialis reflex involves afferent and efferent fibres also in the radial nerve but the reflex may not always be present in normal adult animals.

All these reflexes usually are active in normal neonates and there is a prominent crossed extensor reflex present, and these slowly subside through the first weeks of life.

The pelvic limb spinal reflexes may also be evaluated in all animals that can be restrained in lateral recumbency and in all recumbent patients. In addition, the amount of voluntary effort and muscle tone present in the pelvic limbs is assessed in recumbent patients. As described for the thoracic limbs, this can be done while watching the animal attempt to get up, or by observing its struggle in response to
stimuli while lying in lateral recumbency. Consideration must be given to possibly exacerbating a fracture.

The patellar reflex and the flexor reflex are the two most clinically important spinal cord reflexes involving the pelvic limbs. The patellar reflex is performed by supporting the limb in a partly flexed position, tapping the intermediate patellar ligament with a neurologic hammer or a heavy metal plexor, and observing for a reflex contraction of the quadriceps muscle resulting in extension of the stifle [Figure 5]. The sensory and motor fibers for this reflex are in the femoral nerve and the spinal cord segments involved are primarily L4 and L5. The flexor reflex is performed by pinching the skin of the distal limb with needle holders and observing for flexion of the limb. The afferent and efferent pathways for this reflex are in the sciatic nerve and involve spinal cord segments L5 to S3.

Although two other reflexes can be elicited in most neonatal animals, they frequently are not clearly reproducible in adult patients and thus results of testing them do not usually contribute to defining the site of neurologic lesions. The gastrocnemius reflex is performed by balloting the gastrocnemius tendon and observing and palpating for contraction of the gastrocnemius muscle, which is accompanied by extension of the hock. This reflex involves the tibial branch of the sciatic nerve and spinal cord segments L5 to S3. Secondly, the cranial tibial reflex causes contraction of the cranial tibial muscle with hock flexion occurring when the muscle is balloted and the relaxed limb is held partially extended. Variable limb movement in response to mechanical stimulation may be interpreted falsely as a positive reflex muscle contraction when both these reflexes are tested.

Distinguishing characteristics of lower motor neuron paresis with poor to absent reflexes and paralysis from upper motor neuron paresis with good reflexes can be straightforward and assist in anatomically localizing the site and extent of spinal cord and peripheral nerve lesions in many patients. However, in recumbent heavy patients and those with chronic disease and disuse these classic characteristics can merge such that this distinction can be problematic.

Skin sensation of the pelvic limbs should be assessed independently from reflex activity using the two-pincht technique. The femoral nerve is sensory to the skin of the medial thigh region, the peroneal nerve to the dorsal tarsus and metatarsus, and the tibial nerve to the plantar surface of the metatarsus. As for the thoracic limbs, lesions of the peripheral nerves to the pelvic limbs, such as the femoral and peroneal nerves, result in specific motor deficits; however, the precise sensory deficits can be difficult to define.

The patellar reflex is hyperactive in newborn foals. Also, the cranial tibial and gastrocnemius tendon reflexes are easily performed in healthy, cooperative newborn patients. As with the forelimbs, these patients have normal, strong, crossed extensor reflexes. In addition, an extensor thrust reflex is obtained, in very young foals at least, by rapidly overextending the toe while the limb is already partially extended. This results in forceful extension of the limb, and possibly represents a Golgi tendon organ reflex.
Interpretation of Signs in Peripheral Nerve Disease

For accurate interpretation of signs of peripheral nerve disease some consideration must be given to the neuropathological classification of damage to peripheral nerves that can result in degrees of loss of function:

- **Neurapraxis** is temporary loss of function with no morphological changes.
- **Axonotmesis** is damage to axons with preservation of myelin sheaths resulting in prolonged loss of function until axonal regrowth re-establishes innervation of muscle.
- **Neurotmesis** is severance of axons and their myelin sheaths with prolonged to permanent loss of function, sometimes with partial re-innervation depending on both the distance between the proximal and distal nerve segments and between the lesion and the muscle.

With loss of somatic efferent innervation due to axonal or whole nerve fibre damage there is muscle atrophy, which occurs relatively rapidly although in horses it may take one to three weeks to become clinically prominent. Electromyographic changes indicating denervation of muscle may take even longer, and be 3 to 6 weeks to become prominent in the horse. Surprisingly, disuse atrophy appears to occur quite rapidly in the horse and therefore distinguishing neurogenic atrophy from disuse atrophy clinically can be fraught with problems. A good example of the significance of this would be an unusual asymmetric hindlimb gait abnormality in a horse with accompanying gluteal muscle asymmetry. Unless profound, such asymmetric muscle atrophy should be taken as evidence for disuse due to lameness until proven otherwise.

From a practical point of view peripheral nerves are very difficult to injure directly or to stretch unless they are fixed *in situ*, they overlie a bony structure such as the case for portions of the facial and suprascapular nerves or there is a penetrating injury.

Presumed peripheral nerve irritation and vascular compromise can result in unusual syndromes in horses. Perhaps the best example of these is the abrupt onset of distress involving one limb when the horse will kick out and repeatedly stomp the foot on the ground that can be referred to as a form of claudication. This occasionally is seen following an intramuscular injection, presumed to be adjacent to a peripheral sensory or mixed nerve. The other example would be the similar syndrome that can appear upon recovery from general anaesthesia wherein there is no evidence of a myopathy or motor neuropathy, the most likely explanation being the onset of paraesthesia or as it is referred to in humans, pins and needles. Such unusual syndromes can occur spontaneously in horses sometimes with no associated or predisposing incident, sometimes associated with exertion. Most often these signs dissipate rapidly, with occasional notable exceptions.

Compared to small animals, the specific areas of desensitivity relating to each major spinal nerve, referred to as autonomous zones, are quite variable from horse to horse [Figure 3]. The variable
analgesic zones found following tibial and peroneal, and medial and ulnar nerve blocks undertaken during orthopaedic evaluations attest to this. Determining such precise areas of analgesia can be extremely useful, albeit often frustrating, in helping to localize a peripheral neuropathy although their absence should not exclude such syndromes. On the trunk and proximal limbs the two pinch technique outlined above is preferable for sensory testing.

With the exception of those affecting the cauda equina, peripheral nerve lesions usually result in a gait abnormality involving only one limb. Classically the further the lesion is away from the central nervous system the more selective are any motor and sensory deficits. This is less true in the horse for several reasons particularly because of peripheral nerve anastomoses and secondly because incomplete peripheral neuropathies frequently occur.

The gait abnormalities present after several days following onset of selective median or ulnar neuropathies are minimal. The same can be said of tibial and peroneal nerve lesions although sometimes there will be a change in stride with occasional stumbling.

The radial nerve is probably rarely damaged alone. However, the commonly recognized signs of typical proximal radial nerve paralysis, including lack of carpal and fetlock extension and an inability to bear weight on the limb with a dropped elbow, usually results from partial brachial plexus involvement. Theoretically this syndrome should be distinguishable from myopathy involving the triceps or the extensor carpi radialis muscles, elbow joint lameness, humeral fracture and bicipital bursitis. However in practice such distinction can be difficult to achieve without a thorough evaluation usually requiring ancillary aids including ultrasonography, radiography, synovial fluid analysis and electromyography. Because partial brachial plexus lesions are quite common following thoracic limb injury this problem should be considered foremost when an inability to bear weight on an otherwise pain-free thoracic limb is being evaluated.

Musculocutaneous nerve problems are rarely, if ever, encountered alone and after some initial stumbling any permanent gait abnormality may be difficult to detect.

Immediately after an episode of shoulder injury, signs of damage to the purely motor suprascapular nerve often include a degree of lameness, presumably associated with adjacent soft tissue and periosteal damage. Suprascapular muscle atrophy will ensue in a week or two but the shoulder abduction that occurs on weight bearing, or so-called shoulder slip, that is seen with thoracic limb trauma and is presumed to be lateral laxity to the shoulder joint likely results from loss of lateral support of the shoulder due to suprascapular paralysis alone. Other signs, such as sensory deficits over the caudal neck and shoulder and ensuing muscle atrophy elsewhere in the limb, must make the clinician suspicious of more than suprascapular nerve involvement such as additional damage to the brachial plexus.

The femoral nerve is incredibly well protected from external injury although damage to it will ultimately result in quadriceps atrophy. Even with moderate muscle atrophy and posturing with the pelvis flexed
and back arched, horses with partial unilateral femoral nerve lesions can have a remarkably normal gait at the gallop but athletic performance probably is curtailed. Femoral nerve lesions must be quite proximal in the limb before medial thigh hypalgesia resulting from saphenous nerve involvement can be detected.

Cauda equina involvement most frequently results from a fractured sacrum or from polyneuritis equi. Such signs may begin acutely or may be delayed following onset of the disease. A slightly abnormal gait may be detected in the pelvic limbs but the cause may not be identified until the perineal region is evaluated closely when other signs of cauda equina involvement became apparent.

Other characteristic gait abnormalities do strongly suggest peripheral nerve disease. Stringhalt is one example where there is exaggerated flexion of the limb during protraction with excessive hock flexion and digital extension resulting from excessive contraction of the digital extensor muscles or lack of opposition from digital flexor muscles. This syndrome can occur with spinal cord disease as well as peripheral nerve disease and probably lesions at other sites. A thorough musculoskeletal examination including radiographic and ultrasound evaluation of the affected limb may reveal abnormalities detected within the lateral digital extensor muscle, tendon or sheath, or in the hock. Any abnormalities detected often are assumed to initiate the abnormal neural reflexes thus increasing tone in the digital extensor muscles during protraction. Another interesting gait that results from mechanical interference to contraction of caudal thigh muscles, or perhaps sometimes because of reflex hypertonia involving these muscles, is referred to as fibrotic myopathy. In this syndrome the gait classically results in excessive slapping of the foot to the ground at the end of protraction, thus shortening stride length. Mild to moderate fibrotic myopathy usually does not appear to interfere with high speed performance, however, dressage horses, show horses and trotters and pacers do show an abnormal gait during performance.

Variations in these characteristic gait abnormalities occur. These include repetitive or intermittent mild abduction of the hindlimb during protraction and caudal jerking of the distal hindlimb after the initiation of protraction. It is possible to explain these and other movement disorders by initiation of abnormal muscle spindle activity, as in Stringhalt, with a result that certain muscles or groups of muscles contract too early or too late, or excessively or poorly at a particular phase of the stride. Thus, intermittent abduction and caudal jerking in the hindlimb may result from hypertonia and hyperreflexia involving the biceps femoris muscle during the swing phase of stride.

Cantering with synchronous movement of the hindlimbs is referred to as bunny-hopping and is seen with numerous musculoskeletal problems. It is rarely the result of primary neurologic disease but can occur with certain congenital or acquired spinal cord malformations. Overt evidence of peripheral nerve or spinal cord disease, or identification of bilateral and synchronous hindlimb reflexes determined during recumbency need to be present before a neurologic cause for bunny hopping can be confirmed.

Finally, horses diagnosed as shiverers demonstrate a wide variety of signs including slightly excessive flexion of the hindlimbs along with thigh muscle and tail trembling at the onset of backing, reluctance
to have the hindlimbs picked up with degrees of thigh muscle trembling, inability to back-up and spontaneous and induced episodes of muscle trembling with hindlimb and forelimb and neck extension that may wax and wane. Interestingly, an acquired lameness can abruptly exacerbate the syndrome. A few horses demonstrating shivering suffer from mild spinal cord disease, others from marked lumbar arthropathy, others from destructive lesions of the lumbosacral vertebrae and others from painful conditions involving the distal hindlimbs. More often than not, no site or cause of the lesion is determined in such shiverers.
Bibliography

Cervical imaging in the horse

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Introduction

Imaging of the cervical region is frequently undertaken as part of the diagnostic assessment of horses with neurological disease (e.g. gait deficits) and neuro-anatomical localisation to the cervical spinal cord. In addition, cervical imaging may be undertaken in horses with other presenting complaints, including forelimb lameness, neck stiffness, trauma and head shaking. Comprehensive imaging of the cervical vertebral column and accompanying soft tissues (including the spinal cord) is problematic in adult horses, given animal size and limitations in access to the tissues with several modalities. However, imaging of the cervical region may yield important diagnostic information, both in terms of diagnostic inclusion and exclusion, complementing other aspects of the neurological examination and ancillary testing.

Radiography

Radiographic assessment of the cervical vertebral column from the occipital-axial joint to C7-T1 is commonly undertaken for detection of vertebral malformation or damage to vertebrae from trauma. On occasion, radiographic examination may reveal changes associated with other conditions resulting in extra-dural spinal cord compression, including congenital malformation, osteomyelitis, discospondylitis, neoplasia and disc prolapse. Even in large horses, latero-lateral radiographs to the C7-T1 level are achievable with suitable radiographic equipment. Conversely, dorsoventral (DV)/ventrodorsal (VD) radiographs are more problematic, both in terms of positioning and image quality, given the amount of soft tissue from the mid cervical region caudally. As such, DV/VD radiographs are often restricted to foals and the C1 to C3/4 region in adult horses. Acquisition of radiographs is usually facilitated by sedation of the horses to reduce reaction to positioning of the anode and plate: however, heavy sedation (especially with α2-adrenergic agonists) should be avoided due to risks of exacerbating ataxia and truncal sway and development of movement artefact.

Radiographs present a two dimensional image of the complex anatomy of the cervical vertebrae, which can influence the sensitivity and specificity of the diagnostic procedure. Of particular importance is the radiographic appearance of the dorsal articular process joints (APJ) on latero-lateral radiographic projections and the ability to detect osteoarthropathy and/or fractures involving these regions. In light of the complex anatomy and age-related enlargement of the APJ that may not be associated with
Further consideration during the interpretation of caudal cervical radiographs is a large number of anatomical variations in both the spinous and transverse processes of vertebrae can be detected: some with breed or sex predisposition (Santinelli et al., 2014). Unilateral or bilateral transposition of the ventral process of the C6 onto C7 and variation in the size and shape of the spinous process of C7 and T1 may be appreciated and it is important that these normal variants are not interpreted as new bone formation (Santinelli et al., 2014).

Plain latero-lateral radiographs of the cervical vertebrae provide valuable information for the diagnosis of cervical vertebral stenotic myelopathy (CVSM) in horses. Subjective changes of vertebral change may be present in horses with CVSM (caudal extension of the dorsal laminae, evidence of vertebral instability, caudal epiphyseal flaring, abnormal ossification and degenerative joint disease of the dorsal articulations) (Hudson and Mayhew, 2005; Mayhew et al., 1993); however, these cannot be used as sole diagnostic criteria, due to limitations in sensitivity and specificity (Hudson and Mayhew, 2005). Sagittal ratio values of the vertebral canal are frequently used for the detection of canal stenosis and diagnostic cut-off values for both intravertebral (Moore et al., 1994) and intervertebral SRVs (Hahn et al., 2008) calculations are published. These calculations require high quality latero-lateral radiographs without obliquity. While SRVs below the diagnostic cut-off values are highly predictive of spinal cord compression, false positives and false negatives can occur (Hahn et al., 2008; Levine et al., 2007), possibility due to biological influences, variation in soft tissues and measurement variability (Hughes et al., 2014; Scrivani et al., 2011).

Myelography (positive contrast radiography) is useful for the detection of compressive lesions of the cervical spinal cord and space-occupying lesions of the vertebral canal. While assessment of plain radiographic images, including SRVs, may provide supportive evidence of spinal cord compression in horses with CVSM, the location of compression cannot be accurately determined. Myelography may allow identification of sites of compression to direct decompressive/stabilising surgical intervention and/or confirmation of the diagnosis of CVSM for insurance requirements. While a valuable procedure, myelography is not with risk or limitations. General anaesthesia is required and adverse reactions may occur associated with general anaesthesia, myelographic technique (including contrast material and neck manipulation) and recovery from anaesthesia. In a recent large retrospective study, 34% of horses undergoing myelography had an adverse reaction either in the intra- or post-myelographic period (Mullen et al., 2015). Reactions included seizures, increased neurological gait grade, peripheral neuropathy, altered mentation, hypotension and musculoskeletal injury (Mullen et al., 2015). In an effort to limit adverse reactions associated with myelography, variations in technique have been described. Standing myelography via lumbosacral puncture has been reported, however adequate flow
to the cervical region was variable and inadequate in 3 of 8 horses, limiting the usefulness of this procedure for detection of cervical spinal cord disease (Rose et al., 2007). Ultrasound-guided puncture into the subarachnoid space of the anaesthetised horse to prevent inadvertent spinal cord injury and ensure correct needle placement for delivery of contrast medium has been described (Audigie et al., 2004) and may be useful in reduce potential complications associated with myelography, especially in horses with pre-existing vertebral disease (e.g. atlanto-occipital fusion) that prevents conventional needle placement (Aleman et al., 2014).

Interpretation of myelographic images for spinal cord compression is not without limitations in accuracy. The sensitivity, specificity and predictive values for diagnosis of CVSM will depend on the diagnostic criterion used to define compression and neck position (neutral, flexed or extended) (Hudson and Mayhew, 2005; van Biervliet et al., 2004). In general, as with most diagnostic testing, criteria with greater sensitivity (i.e. ability to detect compression) are offset by lower specificity (increases in false positive results). As there is no ‘gold standard’ myelographic interpretative method, criterion selection and cut-off decision value will depend on the vertebral site/position and implications for disease detection (Hudson and Mayhew, 2005; van Biervliet et al., 2004). Further consideration for myelographic image acquisition and interpretation is the potential advantage of oblique views for the diagnosis of spinal cord compression from pathology of the APJ (Claridge et al., 2010).

Ultrasound

Sonographic examination of the APJ may complement radiographic assessment of APJ disease through imaging of peri-articular soft tissues, vertebrae and margins of the APJ (Berg et al., 2003). Further, the sonographic appearance of the APJ presents a readily identifiable step-like echogenic surface, and this characteristic landmark is the target for ultrasound-guided needle placement for intra-articular injection of corticosteroids in horses with osteoarthropathies of the APJ (Mattoon et al., 2004; Nielsen et al., 2003). Techniques for ultrasound-guided thecal puncture in horses have been described. In anaesthetised horses, sagittal or parasagittal ultrasound-guided atlanto-occipital puncture can be used for collection of CSF and myelography and has been proposed these methods may reduce the rate of complications of myelography (Audigie et al., 2004). More recently, methods for ultrasound-guided thecal puncture at the atlanto-occipital site (Depecker et al., 2014) and the C1-C2 site (Pease et al., 2012) in standing horses have been described. These procedures use lateral or parasagittal approaches, cause minimal/no reaction in adequately sedated horses and CSF can be obtained rapidly, usually without blood contamination (Depecker et al., 2014; Pease et al., 2012). Through these standing techniques, the risks of injury during recovery from general anaesthesia in neurologically-compromised horses, the technical challenges of lumbosacral puncture and the occasional violent reaction of horses during lumbosacral puncture are avoided. Ultrasound-guided lumbosacral puncture has also been
described and may decrease the risk of repeated trauma during the procedure and blood contamination in both standing and recumbent horses (Aleman et al., 2007).

**Advanced imaging**

Computed tomography (CT) and magnetic resonance imaging (MRI) are used infrequently for the assessment of horses with cervical spinal disease, due to limited availability and logistical considerations, including inability to image the caudal neck. While promising results for accurate determination of vertebral canal stenosis and diagnosis of CVSM have been obtained in *post-mortem* MRI studies (Janes et al., 2014; Mitchell et al., 2012), translation to a clinical application currently is unfeasible due to an inability for imaging of the entire vertebral column on horses with existing MRI (or CT) equipment.

**Other imaging modalities**

Cervical vertebral canal endoscopic techniques have been described for examination of the epidural space ('epiduroscopy') or subarachnoid space ('myeloscopy') in horses (Prange et al., 2011a; Prange et al., 2011b). These techniques involve passage of a thin flexible videoendoscope (4.9mm external diameter) via the atlanto-occipital space into the epidural or subarachnoid space in anaesthetised horses and may allow accurate identification of the site of compression in horses with CVSM. While these techniques are predominantly research tools, myeloscopy was successful for identification of the site of spinal cord compression in a horse with CVSM in which myelography results were misleading (Prange et al., 2012).

**References**


Review of the Management of Traumatic Brain Injury in Horses
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Blunt trauma is the most common cause of traumatic brain injury (TBI) in horses and is often a result of a kick from another horse, falling over, colliding with a stationary object, or poll impact from falling over backwards. The management of brain injury in horses begins with recognising the presence of signs that may indicate that a central neurologic injury has occurred. In human medicine the routine use of computed tomography (CT) and magnetic resonance imaging (MRI) is central to the evaluation and management of patients with acute head trauma. Although advanced neuroimaging is becoming increasingly available in equine referral practice, it is rarely indicated or practical during initial management of the acute case. Therefore equine practitioners must rely on thorough physical and neurologic assessment to recognise the presence and/or worsening of neurologic signs and to monitor and treat TBI appropriately.

Most of what we do in the management of brain injury in critically ill veterinary patients is based on an extensive body of research and recommendations available in human medicine. An understanding of the pathophysiology of TBI and the human medical research on this subject is fundamental to making evidence-based management decisions, as above all else, we strive to ‘do no harm’. Can we apply these human guidelines to our equine patients? In the absence of species specific randomised controlled clinical trials, we should be aiming to follow these general principles as much as practically possible. We recognise that in veterinary medicine we have very different practical goals for ‘outcome’ compared to human patients. An acceptable outcome in our equine patients is often more than long-term ‘survival’, rather we require an acceptable long-term quality of life and functionality, at the very least. However, hasty prognostic conclusions should not be made based on the initial appearance of an animal suffering from TBI. Humane euthanasia should be reserved for when the patient fails to respond to appropriate therapy and after a period of time, as recovery from neurologic injury is often possible given an adequate time period. Furthermore, veterinary patients seem to have a remarkable ability to compensate for loss of cerebral tissue. This review will focus on those current human general practice guidelines that can be applied to equine brain injured patients.

The pathophysiology of TBI can be conceptually understood as the result of 2 phases: primary and secondary brain injury, although in reality these processes are a continuum of anatomical and cellular events. Primary injury is the immediate, direct, mechanical injury to brain tissue that occurs at the time of impact (e.g. concussion, laceration). This type of injury is complete, non-reversible and not amenable to specific treatment. In severe cases this phase of injury is deadly. Secondary brain injury refers to the cascade of cellular events that occurs in the hours to days following the initial insult that eventually
leads to neuronal cell damage and death. The delayed nature of secondary injury allows for medical and sometimes surgical intervention and is the major focus of TBI treatment. These secondary mechanisms include hypoxia, ischaemia, extracellular accumulation of excitatory amino acids and calcium, ATP depletion, oxidative damage, inflammation, cerebral oedema, haemorrhage, and increased intracranial pressure (ICP).

The key concepts in the management of TBI are to promptly recognise and specifically treat the factors that significantly contribute to secondary brain injury and poor neurologic outcome. Human TBI treatment guidelines centre on the maintenance of adequate cerebral perfusion pressure (CPP). The most significant factors to recognise and treat early include increased ICP, hypotension, and hypoxaemia. Other systemic factors that also contribute to neurologic injury include hypo and hyperglycaemia, hypo or hypercapnia, acidaemia, hyperthermia, and systemic inflammation.

**Patient Assessment**

A logical and prioritised approach to assessment of the horse with head trauma and potential TBI may involve;

A. First address the ABC’s (airway, breathing, cardiovascular status), as with any other critically ill patient. Hypotension and hypoxaemia must be recognised and treated as a priority, as they are independently and strongly correlated with a poor outcome in human TBI. The combination of physical examination and analysis of arterial or venous blood gas and lactate, indirect blood pressure and pulse oximetry will provide a rapid and acceptable assessment of cardiopulmonary status, if available.

B. Once fluid therapy and oxygenation have been initiated the patient is carefully examined for other traumatic injuries, skull fractures, wounds, etc. Nasal passages and ears should be examined for the presence of blood that may suggest skull fractures, and also for evidence of cerebrospinal fluid (CSF). The presence of CSF in nasal or aural discharges is suggestive of basilar skull base fractures.

C. Initial neurologic assessment should focus on the patient’s level of consciousness, motor activity (including posture), breathing pattern, pupil size and responsiveness, vision, ocular position and movements. If not recumbent, the presence of a head tilt, circling, ataxia, weakness should be assessed. Once the likely site and extent of injury has been determined, a specific treatment plan commences. Care should be taken to interpret the initial neurologic examination in light of the cardiovascular status, as level of consciousness will be depressed in hypovolemic and acidaemic patients. A more detailed neurologic examination and re-evaluation can be performed following systemic stabilization.
D. Further diagnostic evaluation following cardiopulmonary stabilisation is determined by the neurologic status of the patient and should carefully consider the risk vs value of the test. Diagnostic evaluation of horses presenting for acute head trauma and possible TBI should include indicators of systemic perfusion and oxygenation at a minimum, followed by survey head radiographs (including dorsoventral view) to detect the presence of skull fractures or pneumocephalus, endoscopy of the upper respiratory tract including guttural pouches (if considered minimal risk for rapid head movement or falling over). CSF sample collection and analysis is only indicated if there is suspicion of a septic process secondary to fracture.

Neuroimaging using CT or MRI in horses with TBI is limited by the risks of recovering a brain injured horse from general anaesthesia, cost, and availability, and is not required in the majority of patients with minor head injury. If available, neuroimaging may be indicated in the presence of major head injury, need for prognostic information, or possible surgical intervention. CT is the preferred imaging modality during the first 3-24 hours after injury and MRI is recommended for subacute (48-72hrs)and chronic TBI, although either examination can be performed at anytime.

Medical Management of TBI

**Fluid Therapy** - In contrast to previous beliefs that aggressive fluid therapy may contribute to brain oedema, fluid therapy should never be restricted in TBI. Rapid correction of hypovolaemia and hypoperfusion must be a priority in management of horses with TBI. Controversy does exist regarding the optimal fluid choice for resuscitation in human TBI. However, isotonic crystalloid solutions are likely to be the most appropriate resuscitation fluid for most brain injured patients. If hypovolaemic shock and/or an abnormal level of consciousness is present then use of hypertonic saline bolus at a dose of 4ml/kg (7.5% NaCl) or 5.3 ml/kg (3% NaCl) is indicated for rapid cardiovascular volume expansion and reduction of ICP, in combination with isotonic crystalloid fluid therapy to avoid tissue dehydration.

The co-administration of a colloid solution is useful for rapid and sustained blood volume expansion using a smaller volume of fluid. However, much controversy exists in human medicine regarding the use of the synthetic colloids in critically ill patients, with current recommendations against their use in TBI.

**Hyperglycaemia** - Hyperglycemia is believed to worsen neuronal injury and is associated with increased mortality and neurologic outcomes after TBI in humans and laboratory animals. The presence of hyperglycaemia has not been associated with a worse outcome in dogs and cats with TBI. However,
iatrogenic hyperglycaemia should be prevented by avoiding glucose supplementation during large volume fluid replacement unless the patient is hypoglycaemic, and avoiding corticosteroid use.

**Oxygen therapy** – Oxygen supplementation in the form of nasal insufflations is recommended in the initial management of horses with TBI, if it is tolerated. Struggling, head tossing, anxiety and snorting may increase ICP. Oxygenation status may be estimated using non-invasive techniques such as pulse oximetry; an $\text{SpO}_2$ value below 95% warrants oxygen therapy. If arterial blood gas sample collection and analysis is possible, the $\text{PaO}_2$ should be maintained at or above 80-90 mmHg.

**Hyperosmolar therapy** - If clinical evaluation of the equine patient with TBI suggests the patient has, or is at risk of developing increased ICP, such as obtunded mentation, unresponsive pupils, progressive mydriasis, or any deterioration of neurologic status with treatment, then hyperosmolar therapy is indicated to reduce ICP.

Invasive ICP monitoring is standard in human patients with severe brain injury, with the goal of maintaining ICP less than 20 mmHg and CPP between 50 mmHg and 70 mmHg. Intracranial pressure monitoring in anaesthetised and conscious horses has been successfully and repeatedly performed in a University research setting, but is not currently practical for clinical case management. In the absence of direct indicators of intracranial hypertension, the equine clinician must rely on serial neurologic evaluation to decide if hyperosmolar therapy is indicated.

Both mannitol and hypertonic saline are effective hyperosmolar solutions for reducing ICP after TBI. Mannitol is the traditional ICP lowering therapy in human medicine, given at a dose rate of 0.5 to 1.5 g/kg as a slow IV bolus over 15-20 mins, but only after adequate fluid resuscitation. The bolus may be repeated at 2-4 hourly intervals as indicated. The main disadvantage of mannitol use is that its diuretic effect prevents it from being used in a hypovolaemic patient and requires ongoing volume support. There is growing literature to support the use of hypertonic saline in favour of mannitol for the treatment of increased ICP. Hypertonic saline is especially valuable in the cardiovascularly compromised TBI patient and has other potentially beneficial effects of reducing brain excitotoxicity and modulating the inflammatory response. The presence of chronic hyponatraemia must be attempted to be excluded before treatment with hypertonic saline.

The historical use of furosemide concurrently with mannitol to decrease CSF production is not supported by evidence, and may contribute to hypovolaemia, thus its use in TBI management is no longer recommended.

**Analgesic and anti-inflammatory therapy** – Opioids are the most commonly used agents for pain management in critically ill patients due to their reduced negative cardiovascular effects. Butorphanol,
morphine, and fentanyl are appropriate analgesic agents for horses with TBI. Non-steroidal anti-inflammatory agents, either flunixin or phenylbutazone are also indicated in horses with TBI.

**Corticosteroids** – Glucocorticoids deserve a particular mention because their use is currently contraindicated in acute TBI. Large-scale studies in human clinical trials have shown high-dose corticosteroids to have no effect on decreasing ICP or improving outcome ICP, and have actually been shown to cause negative effects that may worsen outcome. In light of this evidence, corticosteroid use in equine TBI should be avoided until randomised clinical studies in veterinary clinical patients prove or disprove a clinical benefit of their use.

**Seizure management** - Prompt and effective anticonvulsant therapy with a benzodiazepine (diazepam or midazolam), or barbiturate (phenobarbital) is critical because seizures are associated with hyperthermia, hypoxaemia and cerebral oedema and may exacerbate increased ICP. Drugs that increase cerebral metabolic rate, such as ketamine, should be avoided. Horses with acute head injury may be recumbent, frantic, and thrashing due to shock and incoordination rather than seizure activity, and can be difficult to distinguish from the seizuring horse. Sedation with xylazine or low-doses of detomidine can be used judiciously to facilitate examination and management, and minimize hypotensive effects. Care should be taken not to allow the horses head to drop lower than shoulder level to avoid increases in intracranial pressure.

**Adjuntive therapies** -

**Thiamine** (Vitamin B1) plays a very important role in glucose metabolism and energy production, where it functions as a required co-factor for certain enzymes involved in glycolysis, the citric acid cycle, and the pentose phosphate pathway. Thiamine is also important in nerve and muscle function, where it plays a role in neurotransmission and excitation. Given the increased susceptibility of damaged neuronal tissue to inadequate energy production and supply, the practice of thiamine supplementation at a dose of 1-5mg/kg IV in neurologic injury in horses appears justified.

**Hypothermia** to a moderate reduction in body temperature (32°C-33°C) reduces cerebral metabolic rate, suppresses inflammation and free-radical production, reduces ICP via vasoconstriction, and decreases glutamate release. Although it is impractical to induce this degree of hypothermia in the horse for the required period of time (48 hours), and the side effects are likely to be limiting, it may be reasonable to avoid active warming in hypothermic patients, and to aggressively cool hyperthermic patients by way of alcohol baths and fans placed in the stall.

The effect of **head position** on ICP has some implications in equine patients. For the standing horse, the head should not be allowed to drop below the level of the shoulder, which should be taken into consideration when the horse is sedated. For the recumbent horse, slight head elevation of 10-30° will
assist in lowering ICP\textsuperscript{15}. It is also important to avoid obstruction to jugular venous outflow, such as tight catheter wraps, thrombophlebitis, or a sharp head-neck angle with head elevation.

**Summary**

Predicting the outcome for an individual case of TBI is challenging. Horses with traumatic head and brain injury typically present with some degree of hypovolemia that may worsen the initial clinical and neurologic assessment. The most important goals of initial treatment are to provide fluid therapy and oxygen, followed by a hyperosmolar agent if there is evidence of increased ICP, and to avoid corticosteroid administration. The equine clinician is encouraged to re-evaluate physical and neurologic status following reasonable efforts at aggressive restoration of intravascular volume and oxygen deficits. Some patients with acute head trauma will have complete resolution of clinical signs over a period of days to weeks, and others with more severe injury may have residual neurologic deficits that may take months to resolve, or never fully resolve.

There are still many questions to answer regarding optimal management of TBI in horses. Equine clinicians should aim to follow the available evidence-based recommendations in human medicine within reasonable and practical limitations. Diligent monitoring and assessment of cardiopulmonary parameters and neurologic signs in response to therapy is practical and guides ongoing management and prognosis in horses with TBI.

**Relevant literature**

Equine echocardiography – How to and what’s new

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Introduction

Echocardiographic examination is an invaluable component of the cardiovascular examination of horses, particularly those with one or more of the following: murmur, dysrhythmia, unexplained tachycardia, pyrexia of unknown origin or poor performance. As a non-invasive methodology, echocardiographic examination facilities assessment of the structural and functional integrity of the heart and allows accurate determination of the location, cause and haemodynamic importance of cardiac murmurs. With the increasing availability and capacity of ultrasound machines in equine practice, a sound echocardiographic examination is achievable and should be considered in horses with suspected or confirmed cardiac disease. The purpose of this presentation is to describe the approach to the echocardiographic examination of the horse and outline recent advances in the acquisition and interpretation of sonographic information.

Notes on the general approach to echocardiography

The echocardiographic examination is performed at both left and right parasternal cardiac windows, from which multiple imaging planes are used to obtain sonographic information. These imaging planes have been described (Long et al., 1992) and allow standardisation of image acquisition for comparison to reference ranges and longitudinal monitoring of individual patients. If available, a concurrent electrocardiogram is obtained during echocardiography for accurate timing of cardiac events and standardised image acquisition. Both long and short axis views are required to permit a thorough integration of the cardiac structure and function and a structured approach to the examination should be followed. In most circumstances, the three principal modalities of echocardiography should be incorporated into the examination: two dimensional or brightness mode (2D), motion (M) mode and Doppler imaging.

Two dimensional imaging allows the determination of the structural characteristics of the heart, including chamber and great vessel dimensions, and overall valve and myocardial systolic function can be appreciated. It is important to note, however, that more specific assessment of cardiac function during
systole and diastole is better achieved using other echocardiographic modalities. M-mode imaging allows accurate measurement of anatomical structures and chamber/vessel dimensions, valve movement, determination of various systolic time intervals and calculation of several indices of global ventricular myocardial function. While these M-mode indices of myocardial function (most notably left ventricular fractional shortening percentage) are simple and readily acquired, only a global assessment of myocardial function is obtained and many are influenced by left ventricular preload and afterload, in addition to myocardial contractility. Conventional Doppler imaging is used to assess blood flow direction and velocity between cardiac chambers, ventricular outflow tracts and across abnormal communications between cardiac chambers and great vessels. Using 2D images to guide Doppler placement, colour flow Doppler and spectral Doppler (pulsed wave and continuous wave) imaging can be performed (Reef, 2004). In general, 2D and M-mode imaging have good repeatability and low intra-observer variability (Buhl et al., 2004; Kriz and Rose, 2002; Young and Scott, 1998): as such, small changes in these variables within an individual horse can represent altered cardiac and haemodynamic function. Greater intra-animal variability of Doppler measurements has been reported (Buhl et al., 2004; Kriz and Rose, 2002; Young and Scott, 1998) and warrants consideration when serial Doppler measurements are made for monitoring within an individual horse.

Normal ranges for 2D, M-mode and spectral Doppler measurements and derived indices are published and/or derived from repeatability studies (Kriz and Rose, 2002; Long et al., 1992; Patteson et al., 1995; Slater and Herrtage, 1995; Zucca et al., 2008) and serve as valuable resources for the assessment of individual horses. In addition to measurement repeatability, important considerations during the interpretation of echocardiographic data include the effects of sedation on myocardial contractility (Buhl et al., 2007), influence of animal body weight (Al-Haidar et al., 2013; Rovira and Munoz, 2009; Rovira et al., 2009; Slater and Herrtage, 1995), breed differences (Al-Haidar et al., 2013; Slater and Herrtage, 1995) and animal age (Al-Haidar et al., 2013; Collins et al., 2010; Rovira and Munoz, 2009).

**Update on advances in echocardiography**

The field of equine echocardiography continues to evolve, influenced by advances in echocardiography in human medicine and technology advances including high-frame rate imaging, digital cine memory capacity and expanding computer processing algorithms/available software. Through the expansion of sonographic technologies, the principal advance is greater accuracy in assessment of cardiac function. Recent advances have also been made through the application of conventional and new methodologies in innovative ways: such an example is the development of pharmacological stress testing protocols with potential for detecting exercise-induced myocardial dysfunction without the need for treadmill exercise (Sanderson et al., 2006). Anatomical M-mode is a modified M-mode that allows free placement of the cursor on the 2D image independent of the sector apex and can be used to replace conventional
M-mode measurement of LV dimensions in horses, including as a post-processing technique from stored 2D cine loops (Grenacher and Schwarzwald, 2010).

The greatest advance in equine echocardiography over the last few years has been the investigation of methods that may allow scrutiny of myocardial function beyond global systolic function. Tissue Doppler imaging (TDI) and 2D speckle tracking (2DST) represent methods that may provide information of regional myocardial function during systole and diastole, with the potential for more accurate assessment in comparison to global indications obtained from 2D and M-mode imaging, through the determination of longitudinal, radial and circumferential changes in myocardial segments during systole and diastole (Decloedt et al., 2011, 2012; Schwarzwald et al., 2009a; Schwarzwald et al., 2009b). While promising modalities in equine echocardiography, further investigation is required to better understand the usefulness of TDI and 2DST for assessment of global and regional myocardial function and to determine robust reference ranges. However, recent evidence supports the applicability of these imaging techniques: both TDI and 2DST were found to be useful for the detection and quantification of LV dysfunction in horses post exposure to lasalocid (Decloedt et al., 2012).

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Athletic Heart Syndrome

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Cardiac enlargement and remodelling occurs with exercise training in horses (Young 1999, Buhl et al 2005). A similar syndrome ‘Athletic heart syndrome’ is observed in human athletes, particularly those participating in strenuous aerobic events (Maron and Pellicia 2006). An increase in cardiac chamber size and valvular regurgitation is a physiological adaption to exercise (Young 1999, Maron and Pellicia 2006). Increased ventricular chamber size allows for greater stroke volume, cardiac output and enhanced oxygen delivery. There is a positive association between heart size and performance in Thoroughbreds and Arabian horses (Young, Rogers and Wood 2005, Sleeper et al 2014). Emerging evidence in human athletes suggests that remodelling is not entirely benign and could parallel pathological heart disease, increasing the risk for arrhythmia and sudden death (Maron and Pellicia 2006). Cardiac enlargement and valvular regurgitation in horses is associated with increasing age, race distance and race type (Buhl et al 2005, Young, Rogers and Wood 2008). In human athletes, a period of deconditioning (rest) leads to a reduction in heart size (Marron and Pellicia 2006). Rest and re-evaluation may present an alternative to retirement for horses with moderate-severe regurgitation and cardiac enlargement.

Cardiac murmurs are common in racehorses (Kriz et al 2000) and echocardiography is necessary to determine the aetiology of the murmur. Mitral regurgitation is seen in 10-20% and tricuspid regurgitation in 20-40% of thoroughbreds, depending on race type (Young and Wood 2000, Young, Rogers and Wood 2005). The prevalence of regurgitation increases with fitness and does not affect performance Young and Wood 2000, Young, Rogers and Wood 2008). Mitral regurgitation leads to left atrial dilation and increases risk of atrial fibrillation De Clercq et al 2014. Mitral regurgitation is associated with ventricular arrhythmia in humans (Maron and Pellucia 2006). It is not clear whether valvular regurgitation increases the risk for pathological arrhythmia other than atrial fibrillation in horses. No association was found between heart size, valvular regurgitation and arrhythmia in a study of 26 standardbreds (Buhl et al 2013), but was limited by small sample size. Current guidelines recommend exercising ECG in horses with moderate to severe mitral regurgitation (Reef et al 2014).

References


Interpretation of arrhythmias does not need to be intimidating. Obtaining a good quality ECG trace is the first step in making a correct diagnosis. There are many different types of ECG monitoring devices. These include traditional paper trace, digital wired devices, digital wireless or telemetric devices, and smart phone apps. Each of these devices have their advantages. Selection of the correct device depends on cost and intended use. It is important that the device is able to make a recording for adding to the patient file and for later review. Some devices can be used remotely. This offers convenience for long term recording (ie over 24 hours) or during exercise.

The quality of the ECG obtained depends largely on the contact between the electrode and the horses skin. Application of adhesive electrodes reduces artefacts. Some electrode dots are better than others. KRUUSE electrodes are particularly good. Alcohol or gel can also be applied to enhance the contact and improve the trace quality. Reinforcement may be needed to prevent electrodes from falling off during an exercising ECG.

For a resting ECG a base apex configuration is used. Standard limb leads may also be used in horses but are limited in usefulness because they do not give information about heart size. The major objective when performing an ECG in a horse is to record timing of cardiac electrical events and evaluate any change in morphology of the complexes.

The P wave reflects atrial depolarisation. Slight variation in the p wave morphology or a biphasic p wave can be a normal finding in horses. The QRS complex reflects ventricular depolarisation and is usually of uniform morphology. Changes in QRS morphology represent ectopic foci. T wave morphology and orientation can vary markedly between horses and is generally not helpful in the diagnosis of cardiac disease.

Event timing is a very important part of ECG interpretation. Dysrhythmias may involve blocks and second degree AV block is a normal, common finding in horses. Atrial fibrillation is maintained with re-entry of the waveform in the atria and is the most common performance limiting arrhythmia in horses. Supra-ventricular and ventricular premature contractions are pathological if observed frequently. Occasional supraventricular and ventricular premature contractions are a normal finding in exercising Thoroughbred and Standardbred horses and are associated with vagal tone (Ryan et al 2005, Physick-Sheard 2010). They can be difficult to distinguish in an exercising ECG. Ventricular premature contractions are associated with a compensatory pause.
References


Further Reading


Cardiology panel/case discussion

Kristopher Hughes and Laura Nath

A collection of cases will be presented by the members of the imaging panel to generate case discussion conference attendees and panel members.
IMAGING OF THE HEAD

How to take better dental X-rays of the horse

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Introduction

This article aims to assist any veterinary practitioner to feel comfortable taking dental radiographs in the horse. The techniques described are relatively easy to perform in the field without specialised equipment and when used correctly can provide good quality images for diagnostic workups or case referral. With dental disease being one of the most common conditions affecting the horse, radiographs are of increasing importance in accurately determining the cause of disease and the likely outcomes for the patient.

Equipment

Any X-ray machine capable of 80kv is suitable for dental x-rays in the horse. Portable machines allow easy rotation and positioning and the ability to take images in the field. Good quality images are possible with film, computed radiography and digital radiography, however when using film it is vital that the film used is matched to the screen in the X-ray cassette. With CR and DR careful selection of software and time spent with the supplier will yield better results as there is wide variation in the appropriateness of standard algorithms when they are used to take dental X-rays. It is really important to spend some time developing a factor chart for the specific equipment used in the practice and to label all images correctly. As a practitioner receiving images from other veterinarians seeking assistance with interpretation, the three most common errors with the images I receive are unlabelled or incompletely labelled images, poor positioning and incorrect exposure. Therefore dramatic improvements in the diagnostic quality of dental images (particularly for referral) are often possible by simply addressing these 3 issues.

Technique

Proper restraint, sedation, patient support and positioning will provide both ambulatory and hospital based practitioners with good quality dental radiographs. Patients can be radiographed both in and out of a crush however bars and gates on the crush may sometimes interfere with positioning.
Patients need to be adequately sedated and adequate sedation is heavy sedation eg Detomidine HCL 0.01mg/kg and Butorphanol tartrate 0.01mg/kg. Several landmarks should be used by practitioners to assist with positioning these include the facial crest and the intersection of the mandibular and maxillary cheek teeth.

Some form of head/chin support is required to prevent horses from moving their heads up and down, and a table, feed bin or “Wheelie bin” are good options. If using a crush with an extendable head support a dental halter can also be used, however some of these halters contain metal bars so care must be taken with positioning to avoid artefacts.

There are a number of methods for holding and positioning X-ray cassettes, commercially available cassette holders provide an external framework and a long extension arm. These offer the benefit of decreasing radiation exposure to assistants, however the weight of the cassette on the end of the long extension or handle can make them tiring or cumbersome, they also usually only allow the use of a single size of plate. Some practitioners advocate the use of elastic straps or bungee cords to attach the plate directly to the patient, this has the advantage of eliminating the need for an assistant however it raises issues associated with the use of elastic straps and metal hooks under tension. The author prefers to use broad lipped “vise grips” with or without an extension handle. This allows more control over the orientation of the plate while not inducing the same fatigue as the commercially available holders; it does however require the use of an assistant.

Due to the levels of sedation often required to obtain good quality images, it may also be necessary for the practitioner and or assistant to support or position the patients head to avoid axial rotation.

Placement of a wooden block between the upper and lower incisors allows the practitioner to take open mouth oblique views and may also assist with allowing the horses muzzle to rest comfortably on the head support.

**Standard views**

**Incisors**

Dorso ventral 20deg rostral (Maxillary incisors and canines) **intraoral**

Ventro dorsal 20deg rostral (Mandibular incisors and canines) **intraoral**

**Cheek teeth**

Lateral
Latero 30 deg dorsal latero ventral oblique

Latero 35- 45 deg ventral latero dorsal oblique

Open mouth lateral oblique

Dorso ventral offset mandible

Dorso ventral 20deg rostral (Maxillary incisors and canines) intraoral

This view involves placing the cassette between the horses incisors (the patient needs adequate restraint and sedation to avoid damage to the cassette) The X-ray machine is then positioned dorsally and rostrally to the horse’s muzzle. The position relative to the midline will vary depending on which tooth is of primary interest, for central incisors the machine should be in line with the midline, for middle and lateral incisors the machine will need to be positioned on a more oblique angle with the rear of the machine positioned more laterally. Correct positioning will allow good visualisation of structures such as pulp cavities and periodontal space.

Ventro dorsal 20deg rostral (Mandibular incisors and canines) intraoral

This is view is essentially similar to the one above except that it is shot from a ventral position. This can be awkward as it involves placing the machine under the horse and care needs to be taken with safety and with positioning.

**Lateral view**

This view is of limited value in examining dental structures however it is useful for examination of the sinuses and to look for the presence of fluid. To assist with this it is best if the horse is positioned with the lips resting on some form of support and the bridge of the nose as near to vertical as possible. The Xray plate should be positioned so it is centered over the rostral point of the facial crest with the base of the plate parallel with the ground. This allows easy determination of the location of fluid lines in the sinuses by allowing the practioner to know the location of the horizontal plane relative to the horses head when viewing images.

**Latero 30 deg dorsal latero ventral oblique**

Oblique views like this one provide the most convenient method of producing separate views of the right and left cheek teeth arcades. The plate is placed against the affected side usually centred over the facial crest and the beam is then directed from the opposite side of the horse at about 30 degrees to the
horizontal plane. The dorsal long edge of the plate should be roughly parallel with the bridge of the horse’s nose. The exact angle required will vary from patient to patient especially with regards to age. As a general rule a steeper angle will be required in younger horses due to the long reserve crown of the teeth where as a shallower angle maybe necessary in older horses.

The use of a bisecting angles technique will minimise elongation or shortening of images. The technique is simple but often complex to describe. Essentially place the plate parallel with the horses face, then move the plate out to where it is parallel with the end of the X-ray machine, now move it back to where it is halfway between these two positions.

**Latero 35- 45 deg ventral latero dorsal oblique**

This view is used to visualise the mandibular arcades, again a bisecting angle technique is used.

**Open mouth lateral oblique**

A wooden block, piece of PVC pipe or a suitable speculum is used to keep the patients mouth open. The open mouth oblique views are used to examine the crowns of cheek teeth; these views are particularly useful for assessing the impact of periodontal disease.

**Dorso ventral offset mandible**

This view requires placing two loops of rope around the horse’s premaxilla and mandible and applying pressure to each to offset the jaw alternatively commercial devices are available to offset the mandible. A cassette is then positioned ventrally along the mandible. This view reduces superimposition of the maxillary cheek teeth and allows for assessment of structures such as the nasal septum.

**Problem solving**

As previously mentioned, time taken to properly label images, care with correct positioning and developing an exposure chart will eliminate many of the common problems. Another important point is to remove head halters or collars from the field of interest (esp. those with metal buckles). The use of a large plate and coning the primary beam to include the entire field of interest should also help in many instances.
Dental anatomy

The radiographic appearance of horses teeth varies significantly with age, due mainly to the hypsodont nature of their teeth. Care needs to be taken when interpreting dental radiographs and knowledge of age appropriate issues is important. For example the apical region of cheek teeth in a young horse (age 3-5) will vary markedly between individual teeth and due to the development of eruption bumps (or cysts) normal anatomical changes such as widening of the periodontal space at this age may mimic those seen with advanced dental pathology in another age group of horses.

Practitioners should always radiograph the contralateral arcade and utilise it for comparison, the pulp horns of teeth become more distinct as the horse ages and there begins to be more apical development. The periodontal ligament is visible as a thin black line between the tooth and the alveolus, and disruption or widening of this structure is often associated with dental disease

Additional radiographic techniques

The use of metallic markers such as paper clips, surgical staples and shoeing nails to identify areas of facial swelling, can be useful when evaluating the relationship between such swellings and involvement of dental structures. In cases where a sinus tract is present malleable metallic probes can be useful tools for examining the depth and location of such tracts, as well as for confirming the location/involvement of affected teeth. Contrast material (such as Iohexol) can also be injected into these to provide further diagnostic information.

Conclusion

High quality dental radiographs can be taken under field conditions when using portable equipment, time taken to ensure correct positioning combined with the use of a factor chart and knowledge of the relevant anatomy will aid in this outcome. Horses need adequate sedation and head support and clinicians will benefit from understanding the use of a bisecting angles technique.
References

2. Barakzai S. How to obtain quality radiographs of the head and teeth 2010 Vol 49 BEVA proceedings
3. Puchalski S. Skull and dental imaging 2011 Vol 33 Bain Fallon proceedings
Intra-oral radiography for equine dentistry

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DVM PhD

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Intraoral radiography has been introduce to equine veterinary for many years but was limited to modified wet or CR film. The advantage of intraoral over extraoral technique are the elimination of superimposition of the opposite tooth arcade and the reduction of distortion of the tooth shape by applying the bisecting angle technique to the projection. There are two positional and angle techniques involve in intraoral radiography, firstly a basic parallel technique which will apply for coronal region of mandibular cheek teeth and occlusal view. Secondly, a bisectional angle technique for maxilla cheek teeth, incisor and canine.

Equine intraoral radiography tools

Until recently, the commercial available of intraoral compute radiography was introduce for equine veterinary. This IO-CR tools come with three difference sizes and cassette holder, light protective sleeve and handle (Figure 1).

![Intraoral compute radiography tools](image.png)

**Figure 1** Intraoral compute radiography tools.
Bisectional angle technique

This technique is applied in the area where parallel technique is impossible due to poor access, this technique will provide true dimension of the interested tooth. This technique is based on the principle of aiming the x-ray beam at right angles to an imaginary line which bisects the angle formed by the longitudinal axis of the tooth and the plane of the receptor.

![Diagram of the bisecting angle technique](image)

**Figure 2**

IO-CR positioning and beam direction for maxilla cheek teeth
The intraoral cassette with IO-CR plate was placed parallel to the occlusal surface of the maxillary cheek teeth and approximately parallel to the hard palate. In order to avoid superimposition of adjacent teeth, the x-ray beam was aimed parallel to the interproximal angle of the tooth of interest and the adjacent tooth.

**IO-CR positioning and beam direction for coronal region of mandibular cheek teeth**

The intraoral plate was placed parallel to the long axis of the cheek teeth arcade. The x-ray beam was aimed at the tooth of interest and directed perpendicular to the tooth. The x-ray beam was also parallel to the interproximal angle of the tooth of interest and adjacent tooth in order to avoid superimposition.
Imaging of the Head - Fundamentals

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The equine head is an anatomically complex structure and presents numerous challenges with respect to imaging. This presentation will focus on the use of radiology for the equine head, particularly in relation to dental and sinus disease.

*Indications*

The two most common indications for radiography of the equine skull are nasal discharge and a clinical suspicion that dental disease has extended beyond the oral cavity. Facial swelling or deformity, draining tracts, pain when eating are all signs that may indicate more severe dental disease.

*Radiography*

Selection of technical factors for skull radiography will depend on individual equipment (generator and detector), however typical exposure factors required are 80 - 100 kVp and 1.0 - 2.0 mAs. Large detectors allow capture an entire row of cheek teeth in one exposure; many digital detectors will require two projections to evaluate each dental arcade. Small detectors may be used for intra-oral radiography.

Common radiographic views for evaluation of dental structures and sinuses:

<table>
<thead>
<tr>
<th>Projection</th>
<th>Tips</th>
<th>Anatomy highlighted</th>
</tr>
</thead>
<tbody>
<tr>
<td>standing lateral</td>
<td>Head at a natural standing angle. Centre at rostral end of facial crest.</td>
<td>sinuses and bones of the skull, gross dental abnormalities.</td>
</tr>
<tr>
<td>lateral - 30° - dorsal oblique</td>
<td>Extend head so it is close to horizontal. Side of interest against the detector, beam from contralateral side. Avoid rostro-caudal angulation. Centre about 3cm above facial crest. Marker at the top, and same as side close to cassette.</td>
<td>maxillary cheek teeth apices and sinuses</td>
</tr>
<tr>
<td>Projection</td>
<td>Tips</td>
<td>Anatomy highlighted</td>
</tr>
<tr>
<td>------------------------------------</td>
<td>----------------------------------------------------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>lateral - 35-45° - ventral oblique</td>
<td>Extend head so it is close to horizontal, use a head rest. Side of interest against the detector, beam from contralateral side. Avoid rostro-caudal angulation. Centre at level of 08/09 teeth. Marker at the bottom, and same side as close to cassette.</td>
<td>mandibular cheek teeth apices and mandible</td>
</tr>
<tr>
<td>dorsoventral</td>
<td>Extend head so it is close to horizontal. Try to avoid left/right rotation of the head. Detector ventral to the head, parallel to the mandible.</td>
<td>nasal cavity, nasal septum, sinuses especially ventral conchal sinus, displaced teeth</td>
</tr>
<tr>
<td>open mouth oblique (10-15°, angled in the opposite direction to closed mouth views)</td>
<td>Place 5cm tall wooden ‘bite block’ or similar between incisors.</td>
<td>occlusal aspects of cheek teeth</td>
</tr>
</tbody>
</table>

The American Veterinary Dental College also advocate taking intra-oral views particularly of the incisors and canines. To take adequate quality intra-oral views, an understanding of the bisecting angle technique is required.

Tips:
• Observe appropriate radiation safety protocols - cassette holders, lead gloves, lead aprons and thyroid collars, and wear radiation dosimeters.
• Adequate standing sedation without significant ataxia is required.
• For intra-oral radiography, tongue movement may be reduced by using butorphanol or diazepam in the sedation.
• Use a rope halter or no halter
• A head rest will help minimise movement.
• Use a large detector and collimate to include entire cheek teeth dental arcade.
• Bungee cords can be used to secure the cassette on the head, avoiding hand-holding of detectors, reducing radiation exposure, and reducing motion artefact from use of cassette holders or hand-holding detectors.
• Take both left and right oblique views for comparison.
• Place small radiopaque marker on soft tissue swellings.
• Use blunt radiopaque probes in draining sinuses.
• Take additional views centred on teeth of interest.
• Use relatively higher exposure factors for cheek teeth, lower exposure factors for the sinuses.
Conventions for displaying images

Standard convention for displaying skull radiographs - allows rapid review and interpretation by allowing comparison with our mental ‘percept’.

- Lateral views are displayed with nose to the viewer’s left, dorsal to the top.
- DV views are displayed with the right side on the viewer’s left.

‘Labial mounting’ convention for displaying dental radiographs - as if the viewer is looking into the patient’s mouth.

- Right cheek teeth: horse’s nose on viewer’s right, maxillary teeth ‘crown down’.
- Left cheek teeth: horse’s nose on viewer’s left, maxillary teeth ‘crown down’.
- Dorsoventral views: horse’s nose down, right side on viewer’s left.
- Straight lateral:
  - left side closest to cassette - nose to the viewer’s left (BUT owing to magnification, apices of right cheek teeth are more visible)
  - right side closest to cassette - nose to viewer’s right (BUT owing to magnification, apices of left cheek teeth are more visible)

Principles for interpretation:

A knowledge of normal anatomy and the expected change in dentition with age is essential. Skull models and normal reference images are helpful resources. Use a systematic approach to interpretation, first dividing the skull into different anatomic regions, and considering various pathologic changes that occur in that region. For subtle lesions, comparison to the normal contralateral side is helpful. If there are suspicious teeth but not convincing findings, repeat radiographs in one to two months, or consider head CT.

The triadan system for naming teeth is recommended; this system is based on three digits, the first digit representing the quadrant (1 = right maxillary, 2 = left maxillary, 3 = left mandibular, 4 = right mandibular), the next two digits representing the tooth, commencing with the central incisor as 01 and increasing caudally.

Carefully examine all bone contours and surfaces for bone lysis and periosteal reaction. Evaluate all sinuses for increased opacity, and use the straight lateral view to detect sinus fluid lines. Check both
crows and apices of all teeth, including evaluation of the occlusal surface for hooks, diastema, and evidence of uneven wear.

Radiographic signs of periapical infection include periapical sclerosis of the surrounding alveolar bone, periapical lysis forming a ‘halo’ around tooth roots, and clubbing of tooth roots. Destruction of the lamina dura may be present, but this sign alone is associated with false positive diagnosis. Maxillary teeth 08s to 11s are contained within the maxillary sinuses, and horses with periapical infection of these teeth commonly have secondary sinusitis. Periapical infection of mandibular cheek teeth may be associated with overlying soft tissue swelling and draining tracts. Periodontal disease is usually best identified through oral examination rather than radiographs, however severe periodontal disease may result in radiographic changes to alveolar crestal bone and may extend to periapical infection.

Radiographic changes of sinusitis are non-specific thus determining the cause requires information from history, oral examination, endoscopy and sometimes surgery. CT and scintigraphy are also useful in investigating the cause, but have limited availability. Radiographic signs of sinus disorders include fluid lines, increased soft tissue opacity due to thickening of sinus mucosa and/or inspissated purulent material, deviation of the nasal septum. Chronicity may lead to mineralisation of inspissated pus which appears as heterogeneous or speckled mineral opacity within the sinuses. The DV view is required to identify involvement of the ventral conchal sinus. Careful evaluation of tooth apices is required to see if sinusitis may be due to dental disease, however note that in one study, 29% of chronic primary sinusitis cases had concurrent radiographic dental changes (Dixon 2012). Distinct rounded soft tissue opacities within the sinuses may indicate sinus cysts or ethmoidal haematomas. Sinus cysts may also distort the shape of overlying facial bones through pressure-induced bone remodeling.

References:


Advanced imaging of the head

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Although radiography is the most widely available imaging modality for evaluation of the equine head, superimposition of structures and complex projectional anatomy can make interpretation of radiographs difficult. Ultrasound provides limited evaluation of the head, being unable to penetrate through bone and air, but has uses in imaging the temporomandibular joint, larynx, eyes, and soft tissues of the neck. Scintigraphy is sensitive for many diseases, particularly dental disease, but has limited availability, low specificity and poor anatomic resolution. Cross-sectional imaging is often preferred. Computed tomography (CT) provides excellent information regarding dental structures, bones and sinuses. Magnetic resonance imaging (MRI) has some use particularly when imaging the brain, but the large amount of dense bone and air of the remainder of the skull means there are few freely available protons for image formation in other regions of the head. Although there are clear benefits to use of CT and MRI, the requirement for general anaesthesia, limited availability of equipment, and relatively high cost means that these studies are not as commonly performed. This presentation will focus on the use of scintigraphy and CT in equine dental disease, however examples of other uses of advanced imaging of the head will be presented.

Scintigraphy

Scintigraphy is best thought of as imaging of a physiologic process. Bone scintigraphy involves the injection of a gamma-ray emitting radiopharmaceutical which localises in regions of high bone turnover by binding to exposed bone matrix. The head is then imaged using a gamma camera, which shows both distribution and intensity of radiopharmaceutical uptake (RPU). Bone scintigraphy is highly sensitive for increased bone turnover, able to detect changes before they are visible with CT or radiography, but is not specific for disease processes and has poor anatomic resolution. For equine dental imaging, scintigraphy is useful in confirming or refuting suspected periapical infection of cheek teeth. It has advantages over CT because it can be performed in the standing, conscious horse, and is more widely available than standing CT. In complex cases with multiple CT findings the relevance of these findings may make CT interpretation difficult, and the addition of scintigraphy may help localise relevant lesions.
The technique involves intravenous injection of $^{99m}$Tc-MDP, and acquisition of right and left lateral, DV, VD, +/- lateral oblique images of the head 2 to 4 hours later. Images are acquired as dynamic studies to allow application of motion correction which improves spatial resolution.

Interpretation requires an understanding of the normal patterns of uptake at different ages, as teeth erupt and surrounding bone remodels. Comparison with the contralateral side is useful, as diseases usually result in unilateral increase in RPU. The reserve crowns appear as ‘cold spots’ of low RPU, with surrounding high RPU of alveolar and interdental bone. Ethmoturbinates, temporomandibular joints, and atlanto-occipital joints appear as region of focal high RPU. Periapical infection appears as focal and intense increased RPU centred over the apex of the affected tooth. When accompanied by sinusitis, the affected tooth is surrounded by diffuse moderate increased RPU. Increased RPU may persist for up to 24 months post-extraction, as the alveolar bone remodels. Periodontal disease can be difficult to diagnose with scintigraphy, with mild increased RPU that is often diffuse and bilateral.

**Computed Tomography**

Computed tomography images are formed by rotating a tightly collimated x-ray beam around the patient, and detecting x-ray absorbance along multiple projection rays. The CT computer calculates x-ray absorbance within individual small volume elements (voxels). This is represented in a greyscale image where the grey value of a pixel represents x-ray absorbance within a voxel. Voxels containing bone are represented by bright pixels, soft tissues by intermediate grey, and gas represented by dark pixels. X-ray absorbance is quantified using the Hounsfield Units (HU) scale; gas has absorbance of -1000 HU, soft tissues 10 to 60 HU, dental tissues range from -1000 to +2400 HU depending on the tissue, and bone ranges from 700 to 3000 HU. Images are acquired in transverse plane, but can be reformatted into any plane and optimised to display bone or soft tissue.

Most CT set-ups require general anaesthesia, and specialised tables able to accurately translate the weight of a horse through the CT gantry with mm/sec precision. Some institutions offer standing equine CT, where images of the head are acquired under sedation only. Unfortunately, standing CT requires a handler to be in the room during acquisition, placing them at much greater risk of radiation exposure.

Computed Tomography has improved sensitivity and specificity for detecting dental and sinus disease, achieved through cross-sectional images that eliminate superimposition, and improved contrast resolution compared to radiographs. Interpretation of head CT requires an excellent knowledge of the sinuses and the complex internal structure of cheek teeth. Enamel is the most dense layer, with both peripheral and infundibular enamel folds clearly seen as hyperattenuating layers. The dentin is less dense thus more hypoattenuating, while cementum fills the infundibulum and is intermediate in
attenuation. Infundibular cemental hypoplasia is present to some degree in most maxillary cheek teeth, and should be considered normal unless they communicate with the oral cavity, when they are termed infundibular carries. Young teeth have a large pulp chamber at the apex of the tooth, and indistinct root morphology. With age roots become more distinct and the volume of pulp chamber reduces due to deposition of secondary dentine.

Early CT findings with periapical infection include gas within the pulp horns, hyperattenuating ‘pulp stones’, increased volume and irregular margination of pulp horns and widening of the periodontal space. More severe, chronic changes are seen as lysis of alveolar bone, clubbing and fragmentation of tooth roots. With chronicity, findings include alveolar bone sclerosis and mineralisation around the tooth apex indicating cementum proliferation. Multiple findings may be seen incidentally in teeth, and changes to the pulp cavity and clubbing of tooth roots are likely the most reliable indicators of periapical infection. Clinically significant infundibular carries have the combination of infundibular gas extending to the occlusal surface, expansion of the infundibulum due to bacterial erosion and destruction of cementum, dentine and enamel. Deep carious lesions may lead to tooth fracture in a sagittal plane, and periapical infection. Features of sinusitis include mucosal thickening, increased fluid attenuation within the sinuses, thickening of maxillary and frontal bones, subcutaneous soft tissue swelling, and remodelling of nasal cochae. Chronic cases may have inspissated pus, which is mineral in attenuation. If an oro-sinus fistula is present, food may accumulate within the sinuses and appear as a heterogeneous mix of air and soft tissue attenuating material. Complications following tooth extraction can be readily identified on CT, and may include fistulae, dental remnants and alveolar sequestrae.

Comparing radiography, CT and scintigraphy, sensitivity and specificity for detecting dental disease of cheek teeth:

<table>
<thead>
<tr>
<th>Modality</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Film-screen radiography</td>
<td>50%</td>
<td>95%</td>
<td>Weller 2001</td>
</tr>
<tr>
<td>Computed radiography</td>
<td>76%</td>
<td>90%</td>
<td>Townsend 2011</td>
</tr>
<tr>
<td>Computed Tomography</td>
<td>100%</td>
<td>96.7%</td>
<td>Manso-Diaz 2015</td>
</tr>
<tr>
<td>Scintigraphy</td>
<td>96%</td>
<td>80-86%</td>
<td>Weller 2001, Barakzai 2004</td>
</tr>
</tbody>
</table>
References


Sustained low-grade exercise induces weight loss and improves metabolic parameters in overweight ponies.

De Laat, M.A., Hampson, B.A., Sillence, N. and Pollitt, C.C.
Science and Engineering Faculty, Queensland University of Technology, Brisbane QLD 4000, Australia.

Introduction
Equine Metabolic Syndrome (EMS) is characterised by obesity, insulin dysregulation and a predisposition to laminitis. Current treatment recommendations for EMS include weight loss and exercise, but supportive data for these recommendations are limited. This study aimed to determine whether sustained, unsupervised daily exercise would reduce body mass and improve insulin sensitivity in overweight ponies.

Materials and methods
Eight, overweight ponies were housed on dry-lots and fed lucerne hay (2% bodyweight) for three months from either a custom-made, dynamic feeder, that induced low-grade exercise, or a stationary feeder (ethics approval SVS/043/14/MORRIS). Using a cross-over design, ponies received both treatments separated by a six week equilibration period. Body morphometrics, fat mass (deuterium dilution) and insulin sensitivity (combined glucose-insulin tolerance test) were measured in all ponies before and after both treatments. GPS tracking and a rating system were used to assess individual feeder use.

Results
The ponies travelled further (p=0.01) when using the dynamic feeder (3439 ± 608 m/day), than the stationary feeder (936 ± 150 m/day). The dynamic feeder reduced (p<0.05) bodyweight (3.1 ± 1.7%), fat mass (4.95 ± 1.6%), body condition and cresty-neck scores, compared to stationary feeder use. Fasting glucose-to-insulin ratios improved (before: 22.4 ± 5.9, after: 31.8 ± 6.2), and AUC_{Insulin} decreased (before: 6792 ± 2556, after: 4279 ± 1948) following exercise in A-rated ponies (p<0.05) and feeder-use rating was correlated (r²=0.69, p=0.06) with AUC_{Insulin}.

Relevance to clinical equine practice
Sustained, low-grade exercise induced using a simple, unsupervised feeding system reduced bodyweight, fat mass and condition, while improving metabolic parameters in overweight ponies. Thus, exercise of low-grade intensity, such as walking 3-4km/day, could help to reduce the incidence of equine obesity, metabolic disease and laminitis.

Declaration of interest
None declared.
The efficacy of Ammo allwormer for the treatment of resistant ascarids in foals across three regions in NSW.


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Introduction

Parascaris spp. are the most pathogenic parasites of immature horses and can result in poor growth, ill-thrift, colic and death subsequent to intestinal impaction or perforation. Anthelminthic drugs remain the mainstay of control programmes for these parasites in foals; however, there is increasing evidence of anthelmintic resistance in Parascaris equorum (PE). Resistance of PE to macrocyclic lactones (MLs) is recognized in many countries and additional treatment options for these parasites are desirable to maintain the efficacy of control programmes. Synergistic efficacy of anthelmintic combinations against nematode parasites of other species has been reported and such strategies may be useful in the management of PE in horses. The purpose of this study was to determine the efficacy of an abamectin (ABA) and morantel (MOR) combination (AMMO Allwormer) for the treatment of ivermectin (IVM) and ABA resistant PE. Two separate trials were conducted in this study, the first in the Sydney and Hunter regions (T1) and a second in Southern NSW (T2).

Materials and methods

Faecal samples were collected from foals aged 3-12 months and naturally infected with ascarids from properties with potential ML-resistant ascarid populations (ethics approval number 13/090). Faecal egg counts (FEC) for PE were conducted using modified McMaster techniques and foals with FEC >50 (T1) or >100 (T2) epg were recruited. Foals were randomly allocated for treatment with AMMO or ABA in T1, and AMMO, ABA, IVM or nil control (CON) in T2. FECs were repeated at days 28 and 56 in T1 and day 14 in T2. The efficacy of each anthelmintic was assessed using the faecal egg count reduction test (FECRT). In T1, 44 foals were administered 48 treatments: AMMO (n = 25) and ABA (n = 23: 4 subsequently treated with AMMO at d56). In T2, 37 foals were administered 44 treatments: AMMO (n = 20: 1 was initially CON), ABA (n = 8: 2 were initially CON), IVM (n = 9: 1 was initially CON) and 7 CON.

Results

<table>
<thead>
<tr>
<th>Treatment group</th>
<th>Trial/day</th>
<th>Mean of individual FECR</th>
<th>FECR group means</th>
<th>Mean of arcin transformed FECR</th>
</tr>
</thead>
<tbody>
<tr>
<td>AMMO</td>
<td>T1 Day 28</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>T1 Day 56</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>T2 Day 14</td>
<td>99.97</td>
<td>99.89</td>
<td>99.78</td>
</tr>
<tr>
<td>ABA</td>
<td>T1 Day 28</td>
<td>-54.4</td>
<td>-15.49</td>
<td>66.96</td>
</tr>
<tr>
<td></td>
<td>T1 Day 56</td>
<td>82.5</td>
<td>53.7</td>
<td>96.9</td>
</tr>
<tr>
<td></td>
<td>T1 Day 14</td>
<td>-208.4</td>
<td>-116.28</td>
<td>-376.92</td>
</tr>
<tr>
<td>IVM</td>
<td>T1 Day 14</td>
<td>49.71</td>
<td>57.43</td>
<td>15.74</td>
</tr>
<tr>
<td>Control</td>
<td>T1 Day 14</td>
<td>-26.19</td>
<td>10.34</td>
<td></td>
</tr>
</tbody>
</table>
The results of this study were consistent with the presence of IVM and ABA resistant PE when a conservative FECRT cut-off value of 90% was applied. AMMO was shown to be effective against these ABA and IVM resistant strains of PE.

**Relevance to clinical equine practice**
Control of anthelmintic-resistant PE is of relevance for equine veterinarians and horse owners in Australia as infections with ML-resistant *Parascaris* spp. can result in foal morbidity and/or mortality without appropriate treatment. This study documents the efficacy of AMMO Allwormer for the treatment and control of ML-resistant PE in horses in Australia.

**Declaration of interest**
Secondary author employed by CEVA Animal Health Pty Ltd, Australia.
The effect of generalised seizures on outcome in foals with neonatal encephalopathy: 246 cases.
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Introduction
Neonatal encephalopathy (NE) is a common cause of neurological disease in foals in the first 3 days of life. It may be associated with hypoxic ischaemic insults, inflammatory mediators or a combination of the two and is often seen clinically with other signs of neonatal syndrome. Clinical signs of NE include altered mentation, changes in responsiveness, altered muscle tone, behavioural abnormalities, loss of thermoregulatory control, vestibular signs, and seizures. Seizures are the clinical manifestation of excessive neuronal activity in the brain and can vary from focal seizures with subtle signs such as facial twitching, to generalised seizures with tonic-clonic movements and loss of consciousness. The concept that neonatal seizures are associated with a poor outcome has recently been challenged by studies in humans and animal models. The aim of this study was to determine whether generalised seizures in foals hospitalised for treatment of NE had an effect on short-term outcome (hospital discharge).

Materials and methods
Records of Thoroughbred foals <3 days of age admitted to Scone Equine Hospital’s Intensive Care Unit (2010-2014) with a primary diagnosis of NE were reviewed and foals with generalised seizures while hospitalised were identified. Foals with prematurity (<320 days gestation) and those euthanased due to financial constraints were excluded. Data analysed using a commercially available statistics program (JMP11, Carey, NC) included patient details, clinical and laboratory parameters, treatment and outcome. Univariable analysis was completed using Wilcoxon/Kruskal-Wallis Tests or Fishers’ exact test as appropriate. Variables were included in the multivariable analysis if the association with seizing was p<0.2. A backwards stepwise logistic regression was used to determine the multivariable model. Variables remained in the final model if p<0.05.

Results
Of the 261 foals presented for NE, 246 met the inclusion criteria. Of these, 202 (82.1%) survived to discharge. Generalised seizures were recorded in 48 of 246 foals (19.5%), of which 23 of 48 (47.9%) were discharged. Foals with NE that had generalised seizures were more likely to require hospitalisation for >10 days (OR 8.0, 95% CI 1.7-43.6), to require cardiovascular support (OR 5.46, 95% CI 1.6-19.1) to have had a red bag delivery (OR 4.49, 95% CI 1.7-11.9) and were less likely to be standing at admission (OR 0.16, 95% CI 0.02-0.7) than foals with NE that did not have generalised seizures. When controlling for severity of disease, foals that suffered generalised seizures were no less likely to survive to discharge than those that did not seize (p=0.14, OR 2.78, 95% CI 0.7-10.7).

Relevance to clinical equine practice
Foals with NE treated in the ICU generally had a good short-term prognosis. Those with generalised seizures required more intensive care but there was no significant difference in their short-term outcome when compared with those that did not seize. Results of this study are consistent with recent reports in human infants that seizures are unrelated to patient outcome. When taken as part of a full clinical picture, this study provides further information to aid in giving a more accurate prognosis for foals with NE.

Declaration of interest
None declared.

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Introduction
To report the incidence of thrombophlebitis in horses in an Australian university teaching hospital, and to prospectively evaluate possible risk factors contributing to the development of thrombophlebitis in the equine patient population.

Materials and methods
Prospective, observational study of equine patients admitted to the University Veterinary Teaching Hospital Camden for a 12 month period from May 2008- May 2009 was performed. All equine patients requiring indwelling venous catheterisation for >24 hours were included. Data collection consisted of two phases. Phase one describes independent observation by the primary author of placement of catheter, and phase two includes independent daily observation criteria for each indwelling catheter by the primary author. The primary observer did not influence the decisions of catheter placement or management of indwelling catheters. All results of observations were analysed by GenStat using logistic regression.

Results
A total of 92 venous catheters were placed in 75 patients over the study period. Of the 92 catheters placed, 17 developed thrombophlebitis resulting in an incidence rate of 18% thrombophlebitis in indwelling venous catheters at the UVTHC during the study period. In the final multivariable logistic regression model, administration of sedations/opioids (OR = 10.6) and absence of hyperfibrinogenaemia (OR = 10.88) were statistically significant (p < 0.05). This meant that the administration of sedation/opioids to equine patients with indwelling venous catheter had a 10.6 times risk of development of thrombophlebitis while hyperfibrinogenaemia was 10.88 times protective towards development of thrombophlebitis. All other factors evaluated in the two phases were not statistically significant.

Relevance to clinical equine practice
In a population of equine patients presented to an equine referral hospital, variations in placement and management of indwelling venous catheters did not contribute to an increased risk of thrombophlebitis. This was likely due to appropriate choices of catheter types for the diseases and patients presented, and reflects appropriate monitoring and management of indwelling catheters for the patients admitted to the UVTHC.

Declaration of interest
None declared.
Pulmonary function testing, bronchoalveolar fluid cytology and mast cell tryptase in a group of Western Australian horses.
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Introduction
The recent literature suggests that mastocytic/eosinophilic inflammatory airway disease (IAD), as characterised by bronchoalveolar lavage fluid (BALF) cytology, occurs with a higher prevalence than previously reported. Data from our hospital confirms that BALF mast cell relative percentages > 2% are common, and often occur without clinical signs suggestive of airway hyperreactivity (AHR). Alterations in airway function as measured by pulmonary function testing (PFT) and/or the measurement of specific inflammatory cell mediators within the BALF may more accurately reflect AHR. This study examined the relationship between AHR, relative mast and eosinophil cell percentage, total mast and eosinophil cell concentration, and mast cell tryptase (MCT) concentration in BALF.

Materials and methods
Twenty four healthy adult sedentary horses were included in the study (ethics approval R2423/11). PFT with histamine bronchoprovocation was undertaken using a commercial flowmetric plethysmography system (Open Pleth™). BAL was performed as previously described in the literature in horses ≤ 16 hours after PFT. Total nucleated cells counts and relative cellular percentages of cells were determined using examination of 400 cells. Horses were categorised as having IAD if a relative neutropenia (>5%) and/or a relative eosinophilia (≥1%) and/or a relative mastocytosis (>2%) were present. MCT was measured from the BALF supernatant using a commercial ELISA (EIAab Inc). Statistical analysis was used to determine associations between total cell count and relative percentages of mast cells, eosinophils, PC35 (an objective measure of bronchoprovocation), and MCT concentration. Data were then categorised to investigate the level of association.

Results
AHR was demonstrated in 53% of horses. Of the horses with IAD (92%) as determined by BALF cytology, the majority had mixed inflammatory cell profile. Neither the relative cell percentages nor the total numbers of mast or eosinophil cells were significantly correlated with AHR, but MCT was significantly correlated with AHR (p=0.05). MCT concentration was not correlated with relative mast percentage or total mast cell count, but was positively correlated with relative eosinophil percentage and the total eosinophil cell concentration (p≤0.05). When data were categorised, MCT concentration was significantly greater in the mastocytosis group (>2%) (p≤0.05), but was not significantly different between the mast cell 2-5% group and the mast cell >5% group. Those horses that were categorised as having a combined mixed mast cell, eosinophilic, neutrophilic response had a significantly higher MCT than all other responses (p≤0.05).

Relevance to clinical equine practice
It was concluded in this population of horses that the relative mast or eosinophil percentage may not be indicative of AHR. It is plausible that cells, although present in higher numbers, could be quiescent in the respiratory tract. A mixed mast cell inflammatory response may indicate an active release of MCT and an associated increased AHR. Given the often vague clinical signs of equine IAD, care should be taken in using existing global cytological definitions of BALF cytology as the sole method of confirming the diagnosis of disease in Australia.

Declaration of interest
This study was funded by the Rural Industries Research and Development Corporation.
Oesophageal lumen pH in yearling horses and the effect of management and administration of omeprazole.

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Introduction
Arytenoid chondritis occurs in humans and other animal species, including horses during preparation for sale. The disease is often performance limiting in racehorses and is a source of wastage and economic loss to the Thoroughbred industry. The aetiopathogenesis of the condition is poorly understood. In humans, arytenoid chondritis might be associated with chemical trauma of the laryngeal mucosa from reflux of gastric contents; treatment involves increasing the gastric pH to >4 with proton pump inhibitors. The objectives of this study were to: 1) evaluate oesophageal lumen pH in yearling horses in a paddock environment and undergoing sale preparation; and 2) determine whether administration of oral omeprazole increases oesophageal pH.

Materials and methods
The study was conducted using a blinded, randomised, placebo-controlled crossover design (ethics approval number 13/092). A pH catheter with two electrodes (proximal and distal) 15 cm apart was inserted into the oesophagus of 6 yearling horses. Luminal pH was recorded for 24 hours over three management protocols. Protocol A consisted of pH readings taken whilst horses were grazing in a paddock. During Protocols B and C, readings were taken from horses housed in stables under ‘sale preparation’ conditions and administered omeprazole paste (4 mg/kg orally once daily, (Protocol B)) or placebo paste (Protocol C) before feeding. The horses were randomly allocated to treatment groups. Following the first sale preparation protocol, horses were returned to the paddock for a 13-day washout period prior to a second Protocol A and crossover stable protocol. Paired t-tests (significance set at P < 0.05) and univariable and multivariable regression were used to analyse the data.

Results
Oesophageal lumen pH ranged from pH 4.9 to 9.7 and varied frequently during measurements for all horses across all protocols. A significant difference in pH was found between the proximal and distal electrodes (P<0.01). No significant difference was found between each Protocol A period (mean pH 7.44 for proximal, 7.26 for distal electrodes). No significant difference in pH was found between Protocol A and Protocol C. Oesophageal pH for the second Protocol A was significantly higher than for Protocol B (P=0.014 proximal; P=0.011 distal). Regression analysis suggested that variation in pH was best explained by the effect of individual horses. Location, feeding time and sex explained a small amount of the variation in pH. Other potential predictor variables (treatment, activity, body weight, amount of concentrate, day number, am or pm, day or night) did not explain pH variation.

Relevance to clinical equine practice
This study demonstrates that oesophageal lumen pH varies in yearling horses, and indicates that gastro-oesophageal reflux might occur in yearlings. Although omeprazole is an effective treatment of arytenoid chondritis in humans with gastro-oesophageal reflux, the current study did not find that omeprazole increased oesophageal pH in horses as the mean pH remained >4 during all management protocols. Further research into the aetiology of arytenoid chondritis and sources of variation of oesophageal lumen pH in horses is required. Research is also required to determine the potential benefits of omeprazole for this condition in horses.

Declaration of interest
Supported by CEVA Animal Health Pty Ltd, Australia.
Equine herpesviruses in the transported horse.
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Introduction
Equine herpesvirus (EHV)-1 and EHV-4 are important equine alphaherpesviruses and can cause significant respiratory and systemic disease. More commonly detected, but less well understood are the equine gammaherpesviruses, EHV-2 and EHV-5. Long distance transportation of horses is associated with immunosuppression and may result in opportunistic respiratory disease associated primarily with bacterial proliferation and possibly viral reactivation. The objective of this study was to examine horses that had experienced long distance transportation in order to detect any evidence of herpesvirus reactivation or shedding and to identify any potential contribution of these viruses to transport related respiratory disease.

Materials and methods
Twelve horses were subjected to an 8-hour road-transport event (ethics approval number 14/037. Whole blood samples were taken prior to, immediately after and 2 weeks following transport. Serum was tested for EHV-1 and EHV-4 antibodies using a type-specific glycoprotein G ELISA. Nasal swabs collected prior to and 5 days post transport were screened for EHV-2 and EHV-5 using qPCR and EHV-1 and EHV-4 using conventional PCR. Subclinical respiratory disease was evaluated by respiratory tract endoscopy and tracheal wash cytology performed on samples collected immediately prior to and after transportation.

Results
Horses were grouped according to the amount of airway inflammation following the transport event. Six horses had persistent neutrophilic airway infiltrates 5 days following transportation (i.e. subclinical respiratory disease) while the remaining 6 horses all had normal tracheal wash cytology. All horses were EHV-1 seronegative, whilst all but one horse was seropositive for EHV-4. None of the horses sampled were PCR positive for either of the alphaherpesviruses. With respect to the gammaherpesviruses, 6 horses were PCR positive for either EHV-2 or EHV-5 prior to transportation. Four of these six horses developed cytological evidence of lower airway inflammation after transportation. Three horses were EHV-2 PCR positive only after transportation, which suggests that they reactivated a previously latent infection after the long distance transport. Two of these three horses developed subclinical disease. Of the remaining 3 horses that were negative for both gammaherpesviruses, only one had subclinical evidence of airway inflammation.

Relevance to clinical equine practice
The clinical significance of EHV-2 and EHV-5 remains in question, however, these results suggest that long distance transportation may reactivate latent gammaherpesvirus infections and that infection with these viruses may be a risk factor associated with lower airway inflammation. The negative effect of transportation on the immune system has been documented previously. While much of the previous work in this field has focused on the role of the alphaherpesviruses, these results suggest that more work should be done to investigate the significance of the gammaherpesviruses, especially as these viruses have genes that encode immunomodulatory molecules such as interleukin 10.

Declaration of interest
None declared.
Serological testing to manage a Strangles outbreak on a large stud farm.
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University Veterinary Teaching Hospital, 410 Werombi Road, Camden NSW 2570, Australia.

Introduction
Strangles is a highly host specific, contagious disease of horses caused by Streptococcus equi subspecies equi. It imposes a significant burden on the equine industry in Australia and worldwide. Effective control of large outbreaks relies on implementation of strict biosecurity protocols as well as identification of recently infected animals and asymptomatic carrier animals. This report describes the management of a strangles outbreak on a large Australian thoroughbred stud with no history of previous infection, including using the recently developed antigen A and antigen C indirect enzyme-linked immunosorbent assay (A&C iELISA).

Materials and methods
The affected farm had a horse population of 314. In total, 71 mares foaled between August and December. All horses were kept in large paddocks, except during periods of intensive management such as parturition, weaning, yearling preparation and veterinary treatment, when horses were stabled or kept in small yards. The outbreak lasted 12 months, with the first case in January. The index case was a mare that returned to the stud having resided for breeding on another farm. In the first three months of the outbreak, disease was limited to two paddocks of mares and foals. At the time of weaning (April and May) disease spread through six of the eight groups of weanlings and to further pregnant and dry mares as well as some other horses. Biosecurity management principles were implemented early in May. These included the risk classification of horse groups and paddocks into green, amber and red zones based on exposure and/or infection status. All management and husbandry procedures were planned around zoning and separate red/amber and green foaling and post foaling facilities were established. Individual clinical cases were managed and treated in accordance with accepted guidelines based on the stage of disease, with emphasis on reserving antibiotic administration in cases to when all evident abscesses were draining. Serological and microbiological protocols for clearance of yearlings for sale and to control disease on the stud commenced in August. A&C iELISA testing of paired serum was used to determine if groups of horses contained recently infected, or asymptomatic carriers.

Results
Clinical signs were exhibited by 16 of 136 mares, 2 of 23 other mature horses, 49 of 70 yearlings and one of 85 foals (post vaccination). Clinical signs were milder in mature horses. One yearling died of infection complications. Serological testing indicated that there was a recently infected or asymptomatic carrier case in each of three out of seven groups tested. Horses in these groups underwent upper airway endoscopy and guttural pouch lavage samples were collected for culture. No S. equi was detected in the lavage samples. Nonetheless, horses with evidence of purulent material in the guttural pouch had sodium benzylpenicillin instilled into each guttural pouch followed by five days of IM procaine penicillin. Yearlings were prepared for sale without further biosecurity restrictions and mares were re-categorized to green. No new cases of strangles were reported in mares during or after foaling and yearlings were successfully entered and sold at their appropriate yearling sales.

Relevance to clinical equine practice
This report describes a new approach to managing an outbreak of strangles in a large horse population using biosecurity protocols and a combination of serological and microbiological testing to contain disease while minimising disruption to stud operations.

Declaration of interest
None declared.
Ultrasonographic diagnosis of desmitis of the collateral ligaments of the distal interphalangeal joint.

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Introduction
Injury to the collateral ligaments (CL) of the distal interphalangeal joint (DIPJ) should be considered as a possible differential diagnosis of foot lameness in horses. Clinical signs are usually non-specific, with few horses presenting with localising signs of deformation or swelling. Although MRI is considered the best diagnostic modality available for assessing soft tissue injuries in the foot, ultrasonography can be useful if MRI is not available or cost prohibitive. The purpose of this case series was to document the signalment, clinical history, diagnostic findings, treatment and outcome in horses with an ultrasonographic diagnosis of CL desmitis of the DIPJ.

Materials and methods
Medical records of horses examined for lameness that underwent ultrasonography of the foot between September 2008 and April 2015 were reviewed. Horses with a diagnosis of DIPJ CL desmitis were included in the case series. Signalment, duration and grade of lameness, response to diagnostic analgesia, radiographic and ultrasonographic abnormalities were recorded. Follow up included repeated clinical examination and ultrasonographic monitoring. Outcome was assessed by questioning of owners, and performance records where available. Ultrasonographic abnormalities were described and classified as Grade 1 (mild lesions involving <1/3 CSA), Grade 2 (moderate, 1/3 – 2/3 CSA), Grade 3 (severe, >2/3 CSA) and grade 4 (complete rupture). Standing MRI was also performed on 3 horses.

Results
Twenty four horses met the inclusion criteria. Mean age was 13 years (range 4-22 years). The duration of lameness prior to diagnosis ranged from 4 days to 1 year. Forelimbs were more frequently affected (22/24) than hindlimbs (2/24). Medial CL injuries predominated (23/26 lesions). Grade of lameness was variable. Radiographs were available for 20 horses: 15 had no significant abnormalities of the DIPJ, 4 had DIPJ osteoarthritis and 1 had osteolysis at the proximal origin of the CL. Ultrasonographic abnormalities included enlargement, alteration in fibre pattern and hypoechogenicity which was either diffuse or visible as a core lesion. Other changes evident in some horses included intra- and/or periligamentous fibrosis, mineralisation, chronic synovitis, and enthesophytes. Of the 26 lesions identified, 6 were grade 1, 10 grade 2, 9 grade 3, and 1 grade 4. In the 3 horses which also had MRI performed, the extent of injury to the CL was underestimated, and in 2 of the 3, additional soft tissue abnormalities were identified. Treatment consisted of rest, corrective shoeing and controlled rehabilitation. Repeated ultrasonographic examination was used to monitor repair and modify the exercise program. Overall, 16/24 (67%) returned to their previous level of exercise or competition, with a further 2 horses currently undergoing rehabilitation without lameness at the time of publication.

Relevance to clinical equine practice
DIPJ CL desmitis is a potential cause of foot lameness in horses. Ultrasonography should be considered when there is local deformation, DIPJ effusion, poor response to intra-articular medication, and in particular in horses with lameness improved with palmar digital nerve analgesia without significant radiographic abnormalities. Ultrasonography allows visualisation of the origin and proximal third of the CL, however, may underestimate the severity of distal lesions which can be identified on MRI. Further, MRI is also useful to identify the presence of concurrent lesions which may negatively impact prognosis. Despite these limitations, ultrasonography of the CL provides valuable information for the clinician dealing with lameness isolated to the foot, and can also provide the basis for monitoring repair and resolution of lesions. The prognosis for return to athletic activity was better than previously reported.

Declaration of interest
None declared.

Russell, J. and Russell, T.

Victorian Equine Group.

Introduction
Distal limb lacerations in the horse are common. Extensor tendons are often lacerated due to their superficial location. Functional healing relies on the extensor tendon forming adhesions to the third metatarsus. For this reason, we have found, that small wounds with minimal tissue trauma may take a long time to form a strong bond between the tendon and surrounding tissue. This is a problem in foals and weanlings, where longer cast duration may cause catastrophic laxity in the flexor tendons and suspensory ligament. We hypothesized that by creating a larger wound with raw surfaces, we could decrease the time needed for cast-coaptation.

Materials and methods
Horses with complete laceration of LoDET and LaDET were included into the study prospectively and followed up at 6 months post surgery. The paratenon of the distal portion of the lacerated digital extensor tendon was removed and a strip of periosteum on the dorsal surface of the third tarsal bone was removed. The debrided tendon surface was then sutured to the periosteum and surrounding soft tissues. The limb was placed in a cast for a maximum of 4 weeks.

Results
10 horses met the inclusion criteria (3 foals, 3 yearlings, 4 adults) and were included into the study. Of the 10 horses that underwent this procedure 90% (9/10) returned to functional soundness.

Discussion
By stripping the paratenon and adjacent periosteum, wound strength is sufficient after 4 weeks to allow cast removal. This is an important consideration when treating extensor tendon lacerations in young horses.
Transurethral endoscopic-guided surgical repair of bladder rupture in a standing mare.

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Introduction
Bladder rupture and uroperitoneum can occur as a foaling complication. The clinical signs are non-specific and include dull mentation, loss of appetite, dehydration, mild colic, and progressive abdominal distension. While the diagnosis can be easily confirmed through a combination of diagnostic techniques (laboratory data, abdominal ultrasound, abdominocentesis, cystoscopy); effective treatment can be challenging. Conservative medical management has been described for small tears. However, larger tears require surgical treatment with several techniques described; including celiotomy, laparoscopy, and following bladder eversion *per vaginum* with blind closure of the bladder tear. This case report is the first description of a novel minimally invasive transurethral endoscopic-guided approach to successfully repair bladder tears in a standing mare.

Case history
A six year-old Thoroughbred mare presented to Scone Equine Hospital two days post foaling with a history of being obtunded with a poor appetite. Clinical findings included moderate dehydration, tachycardia and tachypnoea. Transabdominal ultrasonography identified a large volume of peritoneal fluid. Laboratory data identified azotaemia and electrolyte abnormalities including hyperkalaemia. Abdominocentesis yielded a peritoneal fluid sample with a creatinine concentration four times higher than the serum creatinine concentration. Cystoscopy confirmed the diagnosis of bladder rupture with three separate tears being evident.

Clinical outcome
The mare was medically stabilised prior to surgery with intravenous fluid administration, abdominal drain placement, urinary catheter placement, broad-spectrum antibiotics, and non-steroidal anti-inflammatories. Following sedation and epidural administration, two of the three bladder tears were sutured via a transurethral endoscopic-guided approach with the aid of an automated articulating endoscopic suturing device (SILSTM Stitch); the third bladder tear was considered too superficial to necessitate surgical repair. The operative time was short (50 minutes). The peritoneal drain was maintained for one day post surgery and the indwelling urinary catheter for three days post surgery. Post-operative abdominal ultrasonography confirmed resolution of excessive peritoneal fluid and laboratory data identified resolution of azotaemia and electrolyte abnormalities. The mare had no complications following surgery and was discharged six days later. Cystoscopy 60 days post surgery identified complete healing of the bladder tears.

Relevance to clinical equine practice
This is the first description of a transurethral endoscopic-guided approach for the surgical correction of bladder tears in a mare. There are several advantages of this new technique; including avoidance of general anaesthesia, minimal tissue trauma, avoidance of an abdominal wall incision, excellent visualisation of the bladder tears, avoidance of the risk of excessive traction on the bladder leading to extension of a bladder tear, and shortened operative time relative to other surgical techniques. Disadvantages of this technique include the requirement for specialist surgical equipment, the need for experience in laparoscopic procedures, and the possible limitation of the technique for use in mares due to the wider urethral size relative to male horses and foals.

Declaration of interest
None declared.

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Objective
To determine the outcome of horses with cutaneous neoplasms treated with intralesional cisplatin.

Materials and methods
Medical records of all horses admitted to the University Veterinary Centre from 2007 to 2013 for treatment of cutaneous tumours were reviewed. Horses were eligible for inclusion if intralesional cisplatin was included in the treatment of the neoplasm and if a minimum follow-up period of 2 years was available. Any post-operative follow-up appointments were assessed and the findings noted. The owners of the horses were then contacted via telephone and questioned using a standardised list of questions.

Results
A total of 33 tumors were treated in 31 horses, with tumor type confirmed histologically in 18/31 patients. Tumour types included sarcoid (20), squamous cell carcinoma (5), melanoma (4), soft tissue sarcoma (2), fibrosarcoma (1) and peripheral nerve sheath tumour (1). All animals underwent conventional debulking of the tumor prior to cisplatin treatment. Cisplatin containing biodegradable beads were used in 30/31 horses and injectable cisplatin in 2/31 cases (with one horse receiving both treatments). Two horses were lost to follow-up after the first procedure. Nine horses had repeat cisplatin bead implantation via standing surgery. Twenty-one horses (72%) developed side effects following cisplatin treatment, the majority of which were considered minor. Twenty of 29 (69%) animals for which long-term follow-up information was available were relapse free 2 years after treatment. Tumor reoccurrence was observed in nine (31%) cases. Cosmetic outcome as determined by the owner was deemed good in 25/29 (86%) of cases.

Relevance to clinical equine practice
Results suggest that intralesional cisplatin is effective for the treatment of cutaneous neoplasms in horses. Owners should be forewarned of the potential for minor complications following treatment.

Declaration of interest
None declared.
Changes in vascular fill following deep digital flexor tenotomy demonstrated by digital venograms: 5 cases.
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Introduction
Vascular filling defects noted on a venogram often precede the classic radiographic findings of an episode of laminitis. Digital venogram allows practitioners to identify horses with early vascular changes, isolate those areas of the foot experiencing vascular compromise and monitor the horse’s response to treatment. Deep digital flexor tenotomy (DDFT) is recommended to limit the progression of a severe laminitic episode. As a complement to the surgery a shoe is applied parallel to the solar surface of the distal phalanx, (de-rotational shoeing).

Materials and methods
This was a retrospective case series focusing on horses examined between 2013 and 2014. Horses showing signs of laminitis, clinically deteriorating despite conservative management and showing decreased vascular filling on the initial venogram were included in the study. Horses selected had DDFT performed and a follow up venogram within 10 days of the surgery.

Results
Five horses fit the inclusion criteria for the study. The cases selected included a 16 year old thoroughbred broodmare with complications from grain overload, an 8 year old arabian broodmare with surgical enterolith removal, a 10 year old thoroughbred broodmare with supporting limb laminitis, a 19 year old stock horse stallion with acute colitis and a 4 year old thoroughbred mare that developed laminitis secondary to severe limb cellulitis. All horses demonstrated severe vascular compromise as a result of laminitis in one or both feet in the preliminary venogram. Two horses had unilateral DDFT performed and the remaining 3 horses had bilateral DDFT. Venograms after surgery and derotational shoeing demonstrated an improvement in vascular fill compared to initial venograms in both cases of the unilateral DDFT and in two cases of bilateral DDFT. The third bilateral DDFT showed an improvement in one foot while the other showed deterioration of vascular fill pattern. Three of the horses survived to discharge while the remaining 2 horses were euthanised due to further clinical deterioration. Of the surviving horses, the arabian mare made a full recovery and produced a live foal, the other mare survived to foaling but was euthanised due to continual deterioration. The stock horse stallion returned to light breeding for a year but was subsequently euthanised due to chronic pain.

Relevance to clinical equine practice
The venogram is a diagnostic tool to monitor changes in vascular supply even in the absence of significant radiographic changes in the laminitic horse. It can be used not only as an indicator of vascular compromise, but also to monitor response to therapy. Although severe cases of laminitis are difficult to treat, the venogram can be useful in guiding current and future therapeutic regimes.

Declaration of interest
None declared.
Effect of emptying the vasculature before performing regional limb perfusion with amikacin in horses.
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Introduction
Over the past years intravenous regional limb perfusion (IV-RLP) has become a common method for administration of local antimicrobials in the treatment of orthopaedic infections of the equine distal limb. IV-RLP achieves high concentrations of antimicrobial in the region of infection, which is thought to improve the success of therapy when treating bacterial infections.
Emptying the vasculature with an Esmarch bandage before intravenous regional anesthesia is commonly performed in humans to prevent leakage of the solution under the tourniquet but there is no evidence for its efficacy in horses for antimicrobial intravenous regional limb perfusion (IV-RLP).

Materials and methods
Eight clinically healthy horses underwent two IV-RLP with amikacin in a randomized, cross over design. The first treatment was randomly assigned to either the left or right front limb and subsequent treatment applied to the contralateral limb. A median, ulnar and medial cutaneous antebrachial perineural block was performed before application of the tourniquet. Horses received an IV-RLP with amikacin with or without exsanguination before applying a pneumatic tourniquet at the level of the forearm. The exsanguination was performed by wrapping around the limb a wide elastic tourniquet (Esmarch) starting from the coronary band to the distal aspect of the radius. Two grams of amikacin sulfate were diluted with sterile saline solution to a final volume of 60 mL. Blood was collected from the jugular vein (before tourniquet removal) and synovial fluid from the radiocarpal and metacarpophalangeal joints (5 min after tourniquet removal and at 24h) for amikacin determination. The procedure was video recorded to assess horse movement.

Results
There was no difference in amikacin concentrations in the plasma or synovial fluid from the radiocarpal joint between groups. There was higher concentration of amikacin in the synovial fluid from the metacarpophalangeal joint immediately after tourniquet removal (35 min post injection) in the group with exsanguination of the limb prior to IV-RLP. Horse movement did not differ significantly between groups.

Relevance to clinical equine practice
Emptying the vasculature with an Esmarch bandage before an IV-RLP can increase amikacin concentrations in the metacarpophalangeal joint of horses. This technique may improve efficacy of the regional limb perfusion when treating septic injuries involving synovial structures in the distal portion of the limb. The technique can be easily performed in clinical practice on standing horses, and does not require specialized facilities or equipment.

Declaration of interest
None declared.
Epidemiology of Thoroughbred racehorses entering and leaving the Victorian racing industry.
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Introduction
The thoroughbred racing population is highly dynamic with little formal data collection about horses entering and leaving the industry. This has led to significant gaps in knowledge around two year old racing, the proportion of horses born that race and what happens to horses at the end of their careers. Presently, the general public is very concerned about the welfare of horses despite little knowledge of how long horses remain in the industry, how they are cared for, or where they go after racing, creating an opportunity for misinformation to become fact. This study aimed to determine the time spent in training and racing for each horse and describe the destination and use of horses as they left the Victorian racing industry.

Materials and methods
Records for all foals born in 2005 in Victoria were obtained from the Australian Stud Book and formed the basis of the study population. Of the 2005 Victorian foal crop, the training and racing records for all horses that officially entered training were obtained from Racing Information Services Australia (RISA). Horses were defined as officially having entered training if they had a recorded stable return, barrier certificate or official trial result. For horses that were exported, racing and training records were obtained from Aurion Pedigrees. All data were obtained in 2014 when these horses were aged 9. The data were analysed to determine the number of horses entering the racing industry and the time spent in training and racing stables. These records were used to determine age of start of racing career and career duration. A phone survey and other database searches were conducted to describe the destination and use of the 2800 horses in the 2005 foal crop that were unraced or remained in Victoria.

Results
Of the 4115 foals born in Victoria in 2005, 3036 (74%) entered training with 2676 (65%) starting in a race anywhere during their career. Only 13% of horses in the 2005 foal crop started their racing careers as a two year old, with the majority of horses not starting in a race until their 3rd year. The median time spent in racing was 3 years, with horses on average spending 4 years in training. Horses that raced as 2 year olds had more careers starts, and also had longer racing careers than horses starting for the first time in subsequent years. Interestingly, while the largest proportion of the 2005 Victorian foal crop were registered in training stables as 3 year olds (62%), the largest proportion of these foals raced as 4 year olds (49%). Of the 2216 Victorian horses in the exit survey where a result was known, 1558 had previously entered training, of which 86% were alive at the time of their exit from the racing industry. Of the surviving horses 7% were still racing, 44% had left the racing industry and were re-homed, 24% were being used for breeding and 11% were unknown.

Relevance to clinical equine practice
This survey refutes widely held industry dogma, showing the majority of the 2005 foal crop successfully entered training and started in at least one race. The introduction of two-year old horses to training and racing appeared to have a positive association with career length, confirming previous results in Australia and overseas. In the subset of the 2005 foal crop for which exit data were available, 68% were either re-homed or being used for TB breeding. Improved traceability with inclusion of microchip identification on equestrian databases and recent implementation of the retirement rule AR.64J (1) could assist to determine destinations for horses exiting the thoroughbred racing and breeding industries.

Declaration of interest
Primary author EVA Executive Committee member.