



## Lameness Diagnosis

Chaired by Ellen Singer

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08.30–08.55

### How to: Recognise a subtle lameness

Sue Dyson

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The definition of subtle lameness depends on the experience and skill of the examiner, the type of horse and its temperament and natural gaits, whether it is a forelimb or hindlimb lameness and whether it is unilateral or bilateral. The degree of lameness exhibited by a horse is also highly dependent on the circumstances in which it is observed. An exuberant dressage horse may show no detectable lameness in hand in straight lines; however, following mild sedation subtle lameness may become apparent. I grade lameness on a scale of 0–8 (0 = sound; 2 = mild, 4 = moderate; 6 = severe; 8 = nonweightbearing). Lameness is graded independently at the walk and the trot and under each circumstance in which the horse is examined: in hand, on the lunge on soft and firm surfaces, and ridden.

Questions that we need to ask are: 1. Is the lameness subtle because either there is low grade pain or the horse has a high pain threshold? 2. Is the lameness subtle because the horse's exuberance is masking a more obvious lameness? 3. Is the lameness subtle because the horse has a bilaterally symmetrical lameness? 4. Is the lameness subtle because it is only evident under specific circumstances? e.g. canter pirouette in one direction. 5. Is the lameness subtle because the horse is adapting its posture and gait to minimise pain? e.g. the horse is leaning out or not bending correctly to protect itself. 6. Is pain-related back stiffness preventing the horse from showing concurrent lameness? 7. Is a performance problem due to subtle lameness? For example, is a horse's uneven contact with the bit a reflection of lameness? 8. Is the lameness subtle because the horse is under the influence of analgesic drugs? 9. Is the lameness subtle because the horse has been rested?

As with any lameness examination the horse should be examined carefully at rest and in hand with the performance of routine flexion tests. When examining the horse moving it is useful to have a systematic way of assessment, first at the walk and then at the trot, with a check list, bearing in mind that not all horses have a straight limb flight. Do the left and right fetlocks have a similar degree of excursion? Is the limb flight straight? How does the horse land on each foot? Does the horse track up? Does the horse sound as though it is landing symmetrically? Is there a toe drag? Does the horse look comfortable turning? Is there abnormal movement of the head and neck? Is there symmetrical movement of the *tubera sacrale* and hindquarters? Does the horse move as athletically as you would expect given its breed, type and use?

I believe that it is better to examine horses moving on the lunge than led in a circle because on the lunge it is easier to identify how the horse is holding its body, which may be an

adaptation to pain. The balance of the horse should be compared on the left and right reins. Does the horse look to the outside of the circle? Does the inside hindlimb cross in under the body during protraction? Is there a hindlimb toe drag and if so is this symmetrical? Does the horse pull the handler more in one direction than the other? Does the stride length and frequency change when the horse is lunged on a firm surface compared with a soft surface?

Examination of the horse ridden is invaluable, bearing in mind that a skilled rider can sometimes make adjustments that can mask lameness. It may therefore be useful to see the horse ridden by more than one person, both to a contact and on a long rein. The horse may have to perform to its maximum level of performance for some lameness to become apparent. The rider should be asked to perform rising trot, and to change the diagonal on which they are sitting without changing direction, which may result in lameness accentuation. Useful exercises to highlight low grade lameness are 10 m diameter circles and figures of 8; upwards downwards transitions from trot to walk and walk to trot; leg yielding to the left and to the right. If the horse appears to be moving symmetrically ask yourself if the balance, stride length, impulsion and engagement are what you would expect for the type of horse. If they are not this may reflect lameness. I can often feel a lameness better by riding a horse than I can see from the ground, particularly with some subtle forelimb lamenesses. Working with a skilled rider with good feel can give invaluable information, especially if the veterinarian is not a rider.

It may be worth working the horse hard, then allowing it to stand for >1 h and then reassessing it. Lameness may then be more obvious. Ultimately local analgesic techniques have to be performed to determine the source of pain causing lameness. If I can detect lameness, the lameness is not too mild to investigate using local analgesia, assuming that the horse is cooperative. But all this needs time, experience and a good eye and if a veterinarian does not have these, then it is in the best interest of the client that the horse is referred to someone with the prerequisite skills. If the horse is not cooperative and local analgesia cannot be performed safely it is not appropriate to use sedation to aid restraint. It is better to consider so-called 'diagnostic medication'.

#### Further reading

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#### NOTES



# Hall 1A ■ Friday 9th September

08.55–09.20

## How to: Perform cervical joint injections

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### Indications

Injection of the cervical joints is typically indicated for the treatment of osteoarthritis, most commonly affecting the caudal articulations (C5/6 and C6/7). Treatment may be required for cases presenting with neck stiffness, forelimb lameness or neurological compromise (due to compression of the spinal nerves as they exit the vertebral column or the spinal cord itself) and, less commonly, as a result of investigations into poor performance. It is also possible to assess and aspirate/treat the more cranial articulations, particularly the atlanto-occipital joint. This abstract will concentrate on the injection of the caudal joints.

### Technique

The horse should be sedated reasonably heavily and made to stand square, preferably in stocks. It can be very useful to use a head rest, so that the head and neck remain in the same position throughout the procedure.

Typically, I would assess the joints before clipping the hair, using liberal application of surgical spirit. The C5/6, C6/7 and C7/T1 joints appear similar and are most easily assessed with a microconvex transducer, although it is possible to proceed with a high frequency linear probe. In most horses the musculature of the scapula/shoulder prevents imaging further back than C6/7 but in some patients the C7/T1 articulation can be imaged. This should be borne in mind - until recently I had assumed that the most caudal joint imaged was always C6/7 but this may not be the case. If in doubt, a marker placed on the skin during radiographic assessment can be useful. Palpate the transverse processes of the caudal cervical vertebrae and place the transducer above these, just in front of the shoulder musculature, oriented vertically to produce a transverse image of the joints. Once the joints have been identified, the hair can be clipped so that the site of injection and contact area for the transducer can be prepared. Once clipped and after a short scrub, I then place a small bleb of local anaesthetic at the site of needle placement. The site for insertion of the needle is above (dorsal) to the contact point for the transducer and is estimated by imagining the course of the needle into the joint. However, the main rule is to start high as the angle of the needle direction should be steep to facilitate entry into the joint space which is angled sharply from laterodorsal to medioventral. After placing the bleb, the site is prepared thoroughly. Usually, as it is most common to inject C5/6 and C6/7 on both sides of the neck, I prepare both joints on the left side first so that they can be scrubbed together and then injected in one sterile procedure.

As a rule, I inject 5 mg triamcinolone acetonide into each joint, drawn up into 1.5 ml sterile saline. A separate 18 gauge 3.5 inch spinal needle is used for each joint. The transducer is covered with a size 8 sterile glove - the glove is held open by the operator and a colleague fills a finger or 2 with normal ultrasound couplant

gel before dropping the transducer into the glove. The microconvex probe easily fits down one of the fingers of the glove. Sterile couplant gel is applied to the gloved end of the probe and the joint to be injected is visualised. As I am right-handed, I hold the probe in my left hand and place the needle with my right; for the left side I usually face towards the rear of the horse (and towards the front of the horse for the right-sided joints). The transducer is positioned so that the joint space is seen at the side of the image, allowing more space to visualise the needle approaching the joint. The needle is pushed through the skin bleb and advanced towards the joint. As mentioned previously, the angle of approach should be steep, mimicking the angle of the joint, to maximise the chance of successful entry. The most important thing to remember during any ultrasound-guided technique is to be GUIDED BY THE ULTRASOUND! Take time to ensure that the needle follows the path of the ultrasound beam - failures are often attributable to the path of the needle diverging from the ultrasound. In some cases, the needle advances easily through the joint capsule and a distinct change in resistance indicates successful centesis. In other cases, the needle encounters bone. If the ultrasound image indicates that the tip of the needle is close to the joint, it is usually possible to 'walk' the needle into the joint. Once into the joint space, avoid advancing the needle too far as it is, in theory, possible to enter the vertebral canal. Removal of the stilette occasionally prompts spontaneous flow of synovial fluid but, more commonly, aspiration with a syringe is necessary to confirm correct placement. Synovial fluid should be aspirated in all cases but, as with other joints, there are occasions when the needle is correctly placed but fluid cannot be obtained. I limit myself to 2 repeat placements of the needle (without coming back out through the skin - if this is necessary a new needle should be used) before, if I am confident about the position, I will inject without obtaining fluid. Of course, it gets easier with practice. Generally, I do not hold the transducer while the injection is performed, preferring to put the probe to one side and hold the hub of the needle with one hand and the syringe with the other during the injection. The procedure is repeated for the next joint on the same side, before moving equipment to the other side of the horse and starting again.

The horse is treated with a single dose of i.v. nonsteroidal anti-inflammatory (usually phenylbutazone or firocoxib). I usually advise 3 weeks of restricted turnout following treatment before any further exercise or physiotherapy. In many cases I suggest that the horse is fed from a height for this 3 week period - attaching hay nets to the fencing or similar. This is based on the observation that most affected horses display difficulty/discomfort when reaching to the floor and on several cases they displayed acute episodes of either severe pain or neurological signs after grazing. It seems logical therefore to limit this (admittedly very normal activity!) for a short time after treatment.

### NOTES



09.20–09.45

## How to: Untangle the multi-limb lameness

**Michael W. Ross**

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Single-limb lameness occurs commonly in forelimbs and hindlimbs and clinical recognition is generally straightforward. Analgesia is used to localise the source of pain causing lameness and a diagnosis is established. In horses with chronic, degenerative conditions low-grade mild lameness may go unrecognised for weeks to months. Horses are effective at compensating for mild, chronic low-grade pain and compensatory, coexistent lameness often develops in predictable patterns. Bilateral lameness is most common and, if symmetrical, can be a source of poor performance that goes unrecognised. Bilateral front foot pain is a common cause of poor performance that leads to compensatory hindlimb lameness problems or gait abnormalities. Veterinarians are summoned to evaluate the horse for suspected hindlimb lameness, riders often complain the horse 'feels off behind', only to find the horse to be lame up front as a result of chronic foot pain. Horses may trot apparently without lameness in a straight line but lameness is easily recognised while circling the horse, and is particularly prominent on a hard surface. Palmar digital analgesia in one limb produces obvious contralateral forelimb lameness. The horse's apparent hindlimb gait deficit resolves once pain is abolished in the forelimbs. Thus, forelimb lameness mimics hindlimb lameness. Severe bilateral forelimb lameness can cause substantial hindlimb gait deficits, some of which resemble neurological abnormalities. Rarely, miniature horses or ponies develop severe bilateral forelimb lameness as a result of scapulohumeral joint dysplasia and osteoarthritis (OA), causing unusual hindlimb gait deficits. Bilateral mild laminitis, palmar foot pain, bilateral metacarpophalangeal or carpal OA, and bilateral slab fractures of the third carpal bone can cause horses to exhibit unusual hindlimb gait deficits.

More on forelimb lameness mimicking hindlimb lameness: horses with prominent unilateral forelimb lameness will appear lame in the contralateral hindlimb. For example, a horse with a *grade 3* right forelimb (RF) lameness has a shortened cranial phase of the stride, head elevation (unloading) during protraction of the RF and a head nod down while loading the left forelimb (LF), a straightforward RF lameness. The horse must shorten the cranial phase of the stride in the contralateral hindlimb, the left hind (LH), a stride-to-stride gait compensation to maintain symmetry, and appears 'lame' in the LH (as judged by a shortened cranial phase of the stride). Apparent LH lameness resolves once the source of pain causing RF lameness is abolished using diagnostic analgesia. Importantly, a pelvic hike consistent with LH lameness is not seen. The horse has no need to unload (pelvic hike up) the LH and load (pelvic drop, settling) the right hind (RH), differentiating this situation from genuine LH lameness.

Ipsilateral coexistent lameness is much more common than is contralateral coexistent lameness. In horses that gallop, canter or pace coexistent ipsilateral forelimb and hindlimb lameness is the

norm. Coexistent RF/RH and LF/LH is much more common than contralateral coexistent lameness with one exception - trotters. Trotters most often have coexistent contralateral lameness, LF/RH and RF/LH. Load sharing between the diagonal pairs is a simple explanation. However, another explanation involves the issue of interference at high speeds. For instance, a trotter with LF lameness reduces the cranial phase of the stride in the LF. Prolongation of the caudal phase of the stride puts a trotter at risk of interference of the LF with the LH; a protective mechanism is to shorten the cranial phase in the LH, so the trotter looks like it is 'running behind', or on the left lead. Simulating left lead gallop chronically overloads the RH and eventually coexistent LF/RH lameness develops. If there is coexistent ipsilateral lameness (LF and LH) there will be a head and neck nod commensurate with LF lameness and a pelvic hike associated with LH lameness and the cranial phases of the stride in both limbs will be reduced. Diagnostic analgesia must begin in the hindlimb since hindlimb lameness can mimic ipsilateral forelimb lameness and cause a head and neck nod.

Hindlimb lameness can mimic forelimb lameness - horses with substantial (lameness *grade* >2.5–3) hindlimb lameness can look like they are lame in the ipsilateral forelimb but may only be lame behind (or be lame in both hindlimb and ipsilateral forelimb, see above). A horse with LH lameness will have a pelvic hike up during protraction of the LH and settling during weightbearing of the RH and will have a shortened cranial phase of the stride in the RF. However, while trotting the horse will appear to have a head and neck nod similar to LF lameness, since load is shifted forward (LH and RF move together) and the head and neck nod down when the RF is weightbearing. If only the forelimbs are watched the examiner will mistakenly diagnose LF lameness.

Racehorses with multi-limb lameness develop a typical short, choppy, uncomfortable gait and appear as Dr R. Pilsworth would describe, 'all jarred up'. These horses appear to be sore all over or 'foot sore'. Subchondral bone pain arising from mal-adaptive bone remodelling of the distal aspects of the third metacarpal/metatarsal bones is the most common cause of this multi-limb gait deficit. And, the hindlimb component has the most profound influence on the development of this abnormal gait. An examiner can usually pick out a 'lamest limb' and blocking begins in this limb. The lamest limb can sometimes be determined while watching the horse carefully during deceleration at a trot in hand. Typically, lateral plantar metatarsal analgesia in one limb results in pronounced contralateral hindlimb lameness, much more obvious than in the initially assessed lamest limb. Lateral plantar metatarsal analgesia in the second hindlimb results in marked improvement - the horse may then show forelimb lameness or revert to showing clinical signs in the original hindlimb.

### NOTES



09.45–10.10

## How to: Perform tarsal and metatarsal analgesia

**Andrew P. Bathe**

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Diagnostic local analgesia of the tarsus and proximal metatarsal region is a crucial part of evaluating hindlimb lameness and poor performance, particularly in sports horses. The region has a relatively complex anatomy and understanding the inter-relationship of the different blocks in this region is critical. Particularly when trying to identify pain in the origin of the hind suspensory ligaments, it is important to appreciate that there can be a false positive block from blocking the tarsometatarsal joint. Also just performing a subtarsal block can lead to desensitisation of the distal tarsus and the fetlock. Therefore to positively identify pain as being from the suspensory origin, I prefer to block the tarsometatarsal joint first and then to perform a low 6-point block. If one then obtains a positive sub-tarsal block, one can be fairly certain that pain is originating from the proximal plantar metatarsal region. Another common problem with interpretation is that there is frequently proximal migration of local anaesthetic from a low 6-point block that can desensitise the origin of the suspensory ligament.

A low 6-point block is preferable to a 4-point block, to ensure desensitisation of the dorsal aspect of the fetlock. The most elegant technique for desensitisation of the suspensory ligament is to block the deep branch of the lateral plantar nerve. This is a one-needle technique which is clearly preferable when blocking a hindlimb and is more specific than other techniques. This is most easily done with the leg held up in a flexed position and a 23 gauge 1 inch needle is inserted on the axial aspect of the lateral splint bone, 1 cm distal to the tarsometatarsal joint. The groove between MT4 and the SDFT can be palpated and the needle inserted aiming slightly axiad. Local anaesthetic (3–4 ml) is then injected starting at the full depth of the needle and withdrawing

to about 1 cm of depth. If the needle is correctly positioned then there is free injection. Local infiltration can also be performed and for this I would use a 1.5 inch needle inserted from the lateral side 3 cm distal to the tarsometatarsal joint. This can sometimes desensitise more of the proximal metatarsal bone than blocking the deep branch of the lateral plantar nerve but this is a relatively nonspecific block and there is a much greater risk of desensitising the tarsus. A high 4-point or 6-point block is rarely needed. Analgesia of the tibial and fibular nerves can be used to completely desensitise the hock, but this is a relatively inconsistent block and I use this very rarely. I commonly see cases of proximal suspensory desmitis that have been missed in horses which have failed to block out to tibial and fibular blocks but which do block out to a subtarsal block.

Intra-articular analgesia of the tarsometatarsal joints is very easily performed using a standard approach just proximal to the head of the fourth metatarsal bone. It is important to only inject 3–4 ml of local anaesthetic as a greater volume can leak out and desensitise a larger area, leading to lower specificity. The central distal joint can communicate with the tarsometatarsal joint in a small number of cases and in a greater number of cases there can be diffusion from the TMT to the CD joint. However this cannot be relied upon and clinically there are cases of CD joint disease which do not block out to the TMT joint. Thus to be thorough, the CD joint must be blocked individually. This is a more difficult injection which is normally performed on the medial aspect of the joint between the central, third and second tarsal bones. An alternative dorsomedial approach will also be demonstrated. The tarsocrural joint is also easily blocked from a dorsomedial approach and always communicates with the proximal intertarsal joint.

### NOTES



10.10–10.35

## Provocative tests for lameness evaluation

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Examination for lameness represents a large portion of work for the equine veterinary surgeon. Although a proportion of lameness examinations are straightforward (foot abscess, obvious fracture), a large proportion are essentially a problem solving exercise with the over-riding goal being to reduce a long list of differential diagnoses to one specific diagnosis. Even more challenging is the examination prior to purchase, in which the veterinary surgeon is expected to predict a horse's long-term orthopaedic health based on an examination at one point in time. In an effort to improve the ability to predict either the site of lameness or the likelihood of long-term soundness, provocative tests have become an accepted part of clinical routines for the examination of horses. However, the efficacy of provocative tests continues to be a source of debate within the profession. The debate centres primarily on the consistency of the test between operators and the ability for the test to actually predict a future site of lameness.

Within this session, the following questions will be addressed:

- When do we use provocative tests?
- When should we use provocative tests?
- How do these tests cause or exacerbate lameness?
- How useful are provocative tests?
- Should we continue to use these tests?

### What are provocative tests?

In the context of the lameness or soundness evaluation, provocative tests would include manipulations of the musculoskeletal system that provoke (stimulate) a gait deficit or that increase an already visible gait abnormality. In other words, the tests will increase lameness above the observed baseline (Ross 2011). Included in this category should be the standard flexion tests (proximal and distal limb), some extension tests (wedge tests of the digit), pressure tests and perhaps ridden examination in some situations. As the common perception of a provocative test is a 'flexion' test, this is the topic on which the session will focus.

The most common statement found within the current and previous literature relating to flexion tests can be summarised as follows: these tests are poorly standardised across the profession and the interpretation of their results is difficult (Keg *et al.* 1997a,b; Verschooten and Verbeeck 1997; Busschers and van Weeren 2001; Meijer *et al.* 2001; Kearney *et al.* 2010; Ross 2011). Review of the literature, discussion with colleagues and observation of various methods of performing flexion tests reveals a reasonable variation in technique. Does the angle of the cannon bone to the ground matter when performing a distal or proximal limb flexion test? Does it matter to the result if the examiner flexes the distal and the proximal limb separately or if they flex an entire limb simultaneously? Does the timing of the flexion tests within the examination matter (before or after lungeing)? Is there any evidence-based documentation for any of the opinions? Based on a recent review of the available literature, the answers remain unclear, although some insight into the test results has been provided.

### How do provocative tests work?

The mechanisms by which the provocative tests induce transient lameness are not specifically known. There is obviously the physical pressure applied to the joint to place it in a flexed

position. For the fetlock flexion test, flexion of the joint likely results in pressure on the palmar/plantar soft tissue structures (proximal sesamoid bones, DDFT, SDFT, palmar annular ligament) and tension across the dorsal margin of the joint. Theories that have been proposed for flexion causing pain include an increase in intra-articular pressure and potentially an increase in intra-osseous pressure (Keg *et al.* 1997a,b; Kearney *et al.* 2010). In reality, the fetlock joint is rarely placed under such pressure in flexion during normal locomotion, as maximum flexion of the joint occurs during the swing phase when the limb is not loaded (Keg *et al.* 1997b). More realistic might be a test of hyperextension of the fetlock joint, which would mimic the position of the limb during stance. Flexion of the carpus is likely to be reasonably specific to this region, as flexion of this area can be performed with little effect on the other forelimb joints. Flexion of the upper hindlimb will result in flexion of the tarsus, stifle and coxofemoral joints together, with passive flexion of the metacarpophalangeal joint being unavoidable.

A number of studies have been performed in an effort to determine the optimum pressure at which a flexion test should be performed and to determine which structures are truly affected by flexion of the distal limb. The pressure during distal limb flexion has been measured in these studies using a 'Flex-o-meter' that measures Newtons of pressure created while the test is being performed, allowing some standardisation between operators (Keg *et al.* 1997a,b; Verschooten and Verbeeck 1997; Busschers and van Weeren 2001). Not surprisingly, as the duration of the flexion test increased (30–300 s) and the pressure applied increased (100–187 N), the response to the test increased with either an increased degree of lameness (Keg *et al.* 1997a) and/or an increased number of horses showing a positive response (Verschooten and Verbeeck 1997).

A study of a group of 100 clinically sound individuals found that, following flexion of the distal limb with 150 N for 60 s, 50% of horses demonstrated a slight lameness, 35% a mild lameness and 15% a distinct lameness at the trot (Busschers and Verbeeck 2001). The incidence of a positive flexion test increased with older horses and in mares as compared to geldings. In addition, this study noted an increase in the number of positive flexion tests if the test was repeated at short intervals (30 min) with no difference in the result if the test was repeated after 48 h. Interestingly, and perhaps most importantly, if the test was repeated after 6 months in the same horses, there was a decrease in the response to flexion (Busschers and Verbeeck 2001).

The most recent studies to concentrate on flexion tests of the distal limb were designed to determine which specific anatomic regions of the distal limb were affected by these tests (Meijer *et al.* 2001; Kearney *et al.* 2010). The methodology was similar between the studies, with a force of 250 N applied for 60 s which resulted in noticeable lameness in all horses prior to the study intervention. The first study showed that only diagnostic anaesthesia of the metacarpophalangeal joint resulted in a significant reduction in the response to distal limb flexion, as compared to intra-articular anaesthesia of the distal or proximal interphalangeal joint or the navicular bursa (Meijer *et al.* 2001). A more recent study that investigated the effects of perineural anaesthesia, reached a similar conclusion, with the low 4-point regional block resulting in a significantly greater reduction in



response to flexion as compared to anaesthesia of the palmar digital nerves or the palmar nerves at the level of the abaxial aspect of the base of the proximal sesamoid bones (Kearney *et al.* 2010). Based on the 2 studies mentioned, the distal limb flexion test primarily affects the MCP joint.

## When do we use provocative tests? Why?

As mentioned above, the main use of provocative tests is either to help localise a source of lameness or to assist in the prediction of lameness in a currently sound individual. There is no evidence of the sensitivity or specificity of provocative tests, for either of the common usages of these tests. As such, the interpretation of provocative test results is often based on individual opinion, personal experience and anecdote. Therefore, the question of why we continue to use these tests remains. Is there a different approach we should be taking to help predict lameness or to assist in a more streamlined lameness evaluation? Are the commonly used tests the best that are currently available?

The use of flexion tests for lameness evaluation is less controversial than their use in the examination prior to purchase, in which a positive response to flexion is often interpreted as an indication of an increased chance of lameness in the future. One study aimed to address this question by following horses from prepurchase examination prospectively; however, the results are inconclusive due to study design (Ramey 1997). At present, a

large scale study to investigate the sensitivity or specificity of provocative tests for lameness evaluation or prior to purchase examination has not been undertaken; however, this would be a valuable endeavour and might provide evidence to answer some of the questions posed above.

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## NOTES



Friday 9th September ■ Hall 1A

## Lameness: Keeping the Competition Horse Sound

Chaired by Ellen Singer

*Sponsored by University of Liverpool*



11.05–11.30

### Training regimes to minimise injury and maximise recovery in the performance horse

**Rachel Murray**

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Training is required to prepare the body for athletic activity. Too little training (demand) is likely to underachieve athletic potential while overtraining can exceed the capacity for adaptation leading to overtraining injury and potentially irreversible damage. Training involves the translation of mechanical stimuli into altered composition/structure, leading to improved structural and material properties to withstand forces during peak loading. However, there is always a balance between optimal training for different structures within the body, balancing demands for cardiorespiratory fitness with loading of the musculoskeletal system. It is common for a performance horse to be specialised for an individual sport, so training is focused on preparing the horse for this particular type of competition. However, like any athlete, repetitive training can be associated with repetitive overload injuries so it is important to develop training regimes that maximise performance at specific competitions while reducing risk of injury during training.

Training regimes should take into account the type of sport that a horse is undertaking, and also the level at which it is training and competing, because the demands of different sports and different levels of a single sport are frequently different, as are risk of different types of injury.

A training programme should include general physiological training, skills training, and both muscle strength and endurance training. Training type needs to be balanced based on the requirements of the individual sport so that the horse is strong enough to perform the exercise required, fit enough to keep repeating it, and skilled enough to perform the exercise safely. Balancing these demands is important to avoid development of problems. For example, concentrating purely on skills training when the horse is not strong and fit enough to perform these skills can lead to resistance or altered gait, so risking injury or conflict behaviour, particularly if the movement keeps being repeated to 'improve' it. Concentrating only on strength training (e.g. for jumping), could allow the horse to jump high, but for only a few successful repetitions which is a problem if the horse needs to complete a long cross country course. Alternatively, having the cardiovascular fitness to complete the galloping part of a course may not be adequate to perform successfully or avoid injury if there is inadequate muscle strength and endurance to achieve the height of the fences.

In principle, strength training requires small numbers of repetitions with increased loading, rest to recover and repeat. This increased loading could be based on height of fence, increased height of ground poles, or height/power of steps (e.g. in piaffe or half pass). Time for recovery between training sessions is necessary, so strength training is most appropriate only 2–3 times per week. Muscle endurance training needs to be balanced with increasing strength and includes higher numbers of repetitions at

lower power. This can be done by repeating a specific movement (such as a jumping effort over a small fence, or raised poles) or by gradually increasing the length of time in a movement (e.g. passage). It is important to monitor the horse for muscle fatigue to avoid incoordination and potential injury.

Skills training must be balanced with strength and endurance training or the horse will be unable to perform the required skills. Skills training involves building neural pathways, proprioceptive conditioning and flexibility. Building neural pathways requires many repetitions but these do not need to be at peak loading, e.g. repeating shoulder-in in a small trot and not at maximum power until the horse is coordinated and strong enough to do so. Proprioceptive conditioning includes use of poles, turns, bending in variable directions, slopes/inclines and variable surfaces, so hacking and turnout are useful. Gradually increasing flexibility demands during training can include smaller diameter circles, bending both ways on circles, lateral work and collection/extension transitions.

General physiological/cardiorespiratory fitness training includes canter/gallop training with interval training, frequent transitions and hill work based on the requirements of the sport and balanced against the musculoskeletal demands and risks.

Core muscle stability and strength are essential requirements for the sport horse. Core muscle conditioning needs to be included in training programmes and is important during rehabilitation following injury. Dynamic mobilisation exercises from the ground are useful. Feeding from the floor can be beneficial. Use of ground and raised poles in walk and trot, and training aids that improve posture can be included as part of a core muscle training programme. Ridden work with the head low and including transitions, half halts, grid work and increasing collection can be useful for core muscle conditioning.

Each training session should include warm up, training period and cool down parts. Warm up increases body temperature allowing exercise to proceed more safely and efficiently. Warm up should not be confused with stretching, which should only be performed following warm up or training. Warm up can include 3 stages: passive warm up where the horse's body temperature is increased externally, using rugs or putting the horse into a warm environment; general warm up which involves nonspecific general movements to increase temperature (generally active walk and slow trot/canter); and specific warm up which involves practising the type of movements that the horse will be doing in the actual training session. Warm up should take into account temperature and humidity. Cool down following hard training is important to return body temperature to normal and for muscle, cardiovascular and respiratory recovery.

A rational training programme is based on specific long- and short-term goals. Horses should be monitored carefully for stress,



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fatigue and illness and the schedule adapted as necessary. A training schedule can be tailored to peak at single or multiple events. Coming into a strenuous competition there can be value to tapering the volume (decreasing the total amount) of training beforehand, although maintaining the intensity (the quality) of the training to avoid precompetition muscle glycogen depletion and fatigue but preparing proprioceptive, neural and muscular pathways.

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## NOTES



11.30–11.55

## Options for managing performance limiting back pain

**Marcus Swail**

*EquiVET Ireland, Feighcullen, Rathangan, Kildare, Ireland.*

Back pain in the high level equine athlete is relatively common and can often cause insidious or overt loss of performance. Back pain can be both occupational - simply a result of the high level of exercise the horse is being asked to do - or a result of

underlying pathology such as, for example, spinous process impingement. In either case there are a number of possible treatment options to address the back pain and allow a return to normal function and level of performance.

### NOTES

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11.55–12.20

### **Palmar metacarpal soft tissues - what changes are compatible with continued competition?**

**Staffan Lidbeck**

*Loberods Hastklinik AB, Loberod, SE-24033, Sweden.*

In this presentation the author will, from his experience, try to answer the question "which soft tissue changes of the palmar metacarpal area are compatible with continued competition?"

Firstly, the most common structures in the area affected with changes in athletic horses will be described.

To be able to give a good answer or at least a qualified guess the author will point at some important aspects.

It is crucial that the examiner has a good knowledge and understanding of the workload and stress the various disciplines put on the horse's tissues in training and competition.

A complete anamnesis is a necessity. How long has the problem been there? Did it show up acute or develop over time? What kind of work, and of what intensity, has the horse done prior to the problem? Has the horse shown any lameness? etc.

The importance of a thorough clinical examination of the

horse, prior to the diagnostic imaging, will be discussed. Some very important aspects are palpable findings of the structure in question and if lameness is present. Not to be forgotten is the possibility of other concurrent problems in the horse that can influence the treatment and prognosis.

The author mainly uses ultrasound as a diagnostic tool for soft tissue injuries. In rare cases MRI will be considered.

Useful aspects of how the ultrasound examination is performed such as preparation, comparison of actual findings to the opposite leg, tilting of the probe, etc. will be discussed.

Case studies will be used to visualise and discuss changes in the above described structures.

To achieve a good long-term result, the examiner's role of giving advice regarding training regimen and other management factors of importance for continued competition will be discussed.

#### **NOTES**

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12.20–12.45

## Quick fixes - what can be done in the 24 hours before competition?

**Marcus Swail**

*EquiVET Ireland, Feighcullen, Rathangan, Kildare, Ireland.*

Veterinarians with high level competition horses under their care are often faced with musculoskeletal problems just prior to a competition, or a particular phase of competition in the case of 3-day eventing. This often results in having to establish a diagnosis under stressful and difficult circumstances. A logical, reasoned

approach is critical to the process at this time. It is important to recognise and accept when a horse is unable to continue to compete or should not compete. Where the horse has an issue that is compatible with continued competition, treatment within the relevant rules is possible.

### NOTES

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## Lameness: Medicating/Treating the Competition Horse

Chaired by Leo Jeffcott

*Sponsored by Boehringer Ingelheim*



13.45–14.10

### Medicating/treating the competition horse: Historical perspective

**Leo Jeffcott**

*Faculty of Veterinary Science, University Veterinary Teaching Hospital, 410 Werombi Road, Camden, New South Wales 2570, Australia.*

I have been a listed Event Veterinarian for the Federation Equestre Internationale (FEI) for 34 years, and have seen some dramatic changes in attitudes to medication and its use during equestrian competition during that time! In the early years there was no proper system of medication control or forensic testing in the FEI at all.

#### Historical perspective

In the mid-70s the FEI rules published a list of 'Forbidden Substances', but there was no mention of corticosteroids or nonsteroidal anti-inflammatories (NSAIDs). It was not until 1981 that 'anti-inflammatory substances, other than phenylbutazone' were included. For this drug there was a maximum permitted level for all disciplines. This was originally  $<4 \mu\text{g/ml}$ , but after a number of scientific publications which reported the unreliability of the pharmacokinetics and the potential side effects, the permitted level was reduced to  $<2 \mu\text{g/ml}$ . In 1993 at the General Assembly in Rio de Janeiro it was banned completely from FEI competition and placed on the 'Prohibited Substance List'.

In the 70s and 80s the whole issue of medication control was inadequately managed. It was based on the system in racing, even using the renowned Horse Forensic Laboratory in Newmarket as the central laboratory. However, the protocol used for the collection of samples (urine and blood) was not sufficiently rigorous, and many successful legal challenges resulted - seriously impairing the validity of the programme. It was not until 1990 that an official Medication Control Program (MCP) was finally introduced, and a proactive Medication Sub-Committee appointed to assist the Veterinary and Legal Committees in managing the drugs used during competition.

#### Prohibited substances

A list was drawn up in the FEI Veterinary Regulations that included all substances deemed to be prohibited that were exogenous to the horse and capable of acting on any of the body systems, not simply the skeletal system.

It was decided, like racing, that horses could compete with the presence of certain substances in their tissues. These are referred to as 'threshold levels/ratios' and apply to only 7 substances which are:

- Substances endogenous to the horse;
- Substances arising from plants grazed or harvested as horse feed;
- Substances in horse feed arising from contamination during its preparation.

There are also a number of drugs that are permitted for use in FEI competition, including antimicrobials, anthelmintics, anti-

ulcer drugs, altrenogest for mares, rehydration fluids, electrolytes and B vitamins.

Following a disastrous Olympics in Athens in 2004 with doping and medication controlled positives in Gold medal horses, there was a major review of the MCP and the publication of new Equine Anti-Doping and Controlled Medication Regulations (EADCMR). In this, the Prohibited Substance List has been split into 'banned substances' and 'controlled medication substances'. This is a huge step forward and a major plank in the upgrade of the FEI's Medication Control Program, and its recent CleanSport Campaign.

#### FEI philosophy on drugs in sport

For all intents and purposes there was a 'zero tolerance' approach to any prohibited substance, and this inflamed many owners, riders and vets. The FEI's philosophy has always been to maintain a clean competition without the influence of any drugs affecting performance. The specific objectives are that:

1. Horses must compete on their inherent merits;
2. Competition must be safe and fair to all;
3. Prohibited substances are not permitted, but are classed as banned or controlled medication;
4. A strict system of medication control is in place for all international competitions;
5. Emergency treatment may be permitted (provided points 1–3 are not compromised).

However, in the interests of horse welfare the FEI also has a policy of authorised or permitted medication which is somewhat contrary to the philosophy on drugs in sport. Authorised medication is carefully managed and is under the control of the official veterinarians. It must:

1. Be a scheme with the best interests of the horse and its welfare;
2. Use only medication that does not affect the horse's performance;
3. Ensure that there is experienced veterinary supervision in place;
4. Be constant review of the ever changing issues in medication of horses in competition.

The system used at FEI Events during competition involves the completion of a number of authorised medication forms for Emergency Therapeutic Use Exemption (ETUE):

Form 1: Authorisation for emergency treatment; for minor conditions and requiring veterinary examination which



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is signed off by the official vet and the Chairman of the Ground Jury.

Form 2: Declaration for administration of altrenogest (Regumate) to mares.

Form 3: Authorisation for vets to give medication that is not prohibited (e.g. rehydration fluids).

Form 4: Application for elective testing for a number of drugs that may still be in the horse's system after treatment before competition.

## FEI CleanSport campaign

The FEI developed its CleanSport programme to ensure competition was 'drug free' and to improve the image of the sport. The benefits so far have been:

1. A significant reduction in the overall positive rate from 4.6% in 2004 to less than 1.0% in 2009.
2. The EADMC Rules make it much clearer for persons responsible and others on the implications of doping and controlled medication.
3. The new Prohibited Substance List is comprehensive and will be continually updated as new drugs or substances come to the notice of the FEI.
4. The Prohibited Substance Database which is freely available

to all is extremely helpful in understanding the limits of control medication and doping.

5. The production of a list of Detection Times for commonly used drugs is greatly assisting veterinarians treating horses near to the time of competition.
6. The ability to undertake Elective Testing of horses prior to competition is also of great assistance to Team Vets, riders, Chefs d'Equipe etc.
7. A clear protocol for detecting hypersensitivity at jumping competitions and the appropriate legal framework to disqualify any horse found to be hypersensitive is a very important step forward for the FEI.
8. Increased communication by the FEI on all these issues, and raising the profile by adopting the CleanSport programme is clearly having considerable benefits.

## Conclusion

Whilst the FEI Medication Control Program may still appear to be complicated, the FEI has made enormous progress in achieving a robust system of ensuring the sport is drug free and making the rules for everyone much easier to understand. They are to be congratulated on their CleanSport campaign and the results it has achieved so far.

## NOTES

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14.10–14.35

## Rationale and practicalities of drug testing

**Lynn Hillyer**

*Department of Equine Science and Welfare, British Horseracing Authority, 75 High Holborn, London WC1V 6L, UK.*

'Drug testing' has been a fundamental part of racing and competition horse regulation around the world since Daniel Dawson was executed in 1812 for doping racehorses in Newmarket with arsenic. Things have moved on somewhat since then! Although sanction remains an inevitable part of the process, it is now usually restricted to fines and disqualifications and has been progressively balanced by a shift in emphasis towards acquisition and dissemination of information to help trainers and their veterinary surgeons not fall foul of the Rules in the first place. Key to this has been national dialogue between regulators and their partner laboratories, exponential improvement in analytical ability to test for drugs and international exchange of techniques and data.

Most modern 'drug testing' programmes broadly differentiate between therapeutic medications and drugs which would be widely regarded as having little or no genuine indication ('doping' agents). This is of course a generalisation - whilst some drugs such as cocaine or erythropoietin fall fairly obviously into the 'doping' category and others such as phenylbutazone would seem to be therapeutics, there are times when seemingly straightforward drugs can be used inappropriately or drugs that may be used judiciously in the hands of some under close control may compromise a horse when given mischievously. Add to this the underlying principle signed up to by the majority of racing's regulators, that drugs should only ever be used as an aid to recovery, not as a shortcut avoiding proper rest from training and/or racing (see <http://rules.britishhorseracing.com/Orders-and-rules&staticID=126612&depth=3>, shortly to be formally incorporated into Article 6 of the International Federation of Horseracing Authorities' Agreement October 2011, <http://www.horseracingintfed.com/racingDisplay.asp?section=6>) and the complexities of differentiating between 'good' and 'bad' in an effective medication/drug control programme begin to become apparent.

Taking the starting points that the majority of those training and treating racehorses follow the principle above and that most 'positives' in British and other jurisdictions' racing involve therapeutic medications, it was clear to those who had just established the European Horserace Scientific Liaison Committee <http://ehslc.com/> in 1992 to further international harmonisation that their first and main goal was to find a way to provide information on medications to trainers through their veterinary surgeons. Whilst the principle of 'zero tolerance' with respect to 'doping' drugs was well understood and accepted, there needed to be a new approach to deal with the ever increasing ability of the laboratories to detect lower and lower concentrations that would allow proper treatment of horses in the interests of their welfare

but still enable racing authorities to pick up on patterns of use that could be inappropriate. The concept of 'Detection Times' related to no effect levels of a drug, and screening linked to that level at an internationally harmonised 'Screening Limit' was developed. In a nutshell, this is intended to ensure that through science based information, advice and prevention, if a drug is present in a horse at the time of a race it is below a level where all can be confident that it is unlikely to have a significant effect. To date, Detection Times for 20 commonly used therapeutic medications have been published <http://ehslc.com/detection/> with studies/analysis ongoing/hearing completion for a further 10–15, the majority of which are corticosteroids. The definition of a Detection Time and scientific rationale for its determination, alongside the published literature which underpins the approach, can be found on the EHSCL website. An up to date summary of the British contribution to this programme along with other work carried out by the British Horseracing Authority at its Centre for Racehorse Studies (CRS) at the British Racing School in Newmarket may be found at <http://www.britishhorseracing.com/resources/equine-science-and-welfare/medication-control-research.asp>.

Detection Times and their corresponding Screening Limits apply to samples taken on raceday, usually post race. Many racing authorities also conduct 'out of competition' testing, or 'testing in training'. In line with the shift in emphasis noted earlier, these are increasingly important opportunities to liaise with trainers and their veterinary surgeons to educate and prevent rather than penalise. Samples are analysed with full capability (i.e. below any Screening Limits) and wherever possible advice is given as to treatments that might constitute 'near misses' had they been seen in a raceday sample. It is hoped that ultimately, data from these visits can be used to add to/validate results from the CRS, for example, in assessing what difference if any is seen in drug handling by treated diseased rather than healthy horses.

The strategy behind testing in training and indeed raceday sampling, is becoming increasingly intelligence-led. The 'one size fits all' approach has moved on from sampling, for example, a horse from every race on the card to sampling variably, with targeting, increased pre-race testing and testing in training. 'Planned unpredictability', a key part of any regulator's approach, is being applied to drug testing in racing. The rationale and ensuing practicalities of drug testing today distil down to a simple message - authorities will do all they can to advise and educate those who work within the Rules on veterinary treatment and medication but will not tolerate those who cheat or attempt 'sharp practice' at the expense of racehorse welfare and/or the integrity of the sport.

### NOTES



14.35–15.00

## Managing fetlock pain in the racehorse

**Rob Pilsworth**

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Management of pain originating in the fetlock differs depending on the age of the racehorse concerned and so management strategies will be divided into 2 major categories: The yearling/2-year-old and the 3-year-old and older horse.

### **Fetlock pain in the yearling/2-year-old**

The fetlock joint is a high motion joint which is intensely loaded during rapid exercise. Horses entering training have only previously been used to cantering short distances, without carrying a rider's weight, in the open pasture. They have to undergo an intense period of bone modelling and remodelling to adapt to the excessive strains produced by continued cantering for distances beyond anything they would ever have experienced previously, and far beyond those that the horse evolved to canter over in the wild, carrying extra weight in the form of a rider, who will encourage them to go faster, for further, than they would otherwise do by choice.

### **Capsulitis/synovitis**

In early 2-year-old training, fetlock overload is often represented by capsulitis or synovitis typified by heat, synovial effusion and pain of varying degree on flexion. If the horse's use is to be maintained beyond the age of 3 years then great care should be taken in these early onset juvenile synovitis cases or irrevocable joint damage will be done. The use of intra-articular medication in the 2-year-old which is disease masking (corticosteroids) rather than disease modifying (hyaluronan and polysulphated glycosaminoglycans) should be avoided if at all possible because this will simply 'turn off' the clinical signs that are alerting the horse's connections to the fact that this horse is having problems adapting to the current exercise load.

Reduction in training to a few weeks of walking and jogging, in association with the use of oral nonsteroidal anti-inflammatory drugs, topical therapy with ice and passive flexion physiotherapy of the joint is the ideal treatment for these cases and will allow the joint to settle. This gives it further time to adapt before the commencement of further high-speed training. Several bouts of 'stop-start' training like this may be needed before the horse trains on without problems.

Minor fragmentation in the 2-year-old is common and is commonly associated with hyper-extension of the joint, causing osteochondral fragments to be displaced either on the proximal aspect of the first phalanx or on the distal aspect of the third metacarpal bone, especially the sagittal ridge. Bramlage (2009) has written very persuasively and eloquently on the need for trainers and veterinarians to address surgically the debris-shedding lesions in the fetlock joints of young racehorses, if these horses are to enjoy athletic careers beyond the immediate. In the 2-year-old which is showing great precocity and for which other factors may reduce the possibility of a successful later career (e.g. small size and scope) then intra-articular medication with hyaluronan in conjunction with a low dose of corticosteroid (3–5 mg of triamcinolone acetonide or 4 mg of dexamethasone) will usually give spectacular results in terms of resolution of soreness and effusion but one has to bear in mind that the inciting factors which produce the inflammation in the joint will still be present and degeneration of articular cartilage and changes in the subchondral bone plate will continue to progress as training

progresses. The price for this early remission of symptoms will be paid later in the horse's training career.

### **Subchondral bone injury**

The use of the term subchondral bone injury has become commonly accepted to describe the condition which all racehorse veterinarians will encounter where horses show lameness referable to the fetlock joint in the absence of any obvious swelling, synovial effusion, heat, pain on flexion or radiological change. Lameness is usually alleviated by intra-articular injection of local anaesthetic into the fetlock joint.

In the 2-year-old these lesions are often linked to marked increased radiopharmaceutical uptake (IRU) on scintigraphy, often located in the plantar lateral condyle of the hind fetlock joint and the medial condyle of the front fetlock joint as predilection sites, although both condyles of both joints can be affected. In the 2-year-old these signs should result in an immediate cessation of speed training. These lesions are often termed 'bone bruises', something of an oxymoron as bone cannot really 'bruise'. They often actually involve changes in mineralisation, vascularity, bone remodelling and with continued intense training can eventually become areas of frank bone necrosis.

The best resolution of these lesions often follows a period rest and a graded return to exercise. Not all trainers will have the patience to tolerate this recommendation. Aspirin medication by mouth is often recommended empirically (4 g daily by mouth). In this way these lesions can be reversed completely and the horse returned to athletic function as a normal skeletal specimen. Depending on the stage of the season when they occur, a period of free pasture turnout may be the ideal treatment once the acute changes have subsided with rest (Bramlage 2009).

Short cutting this rehabilitation by the use of nonsteroidal anti-inflammatory drugs as a training aid, in conjunction with intra-articular medication, will certainly allow the horse to train in the face of these acute subchondral bone injuries, but will result in increasing and progressive sclerosis of the condyles, and progression of the bone changes to an advanced degree from which there is no way back. The horse's gait becomes permanently impaired through bilateral or quadrilateral lameness. This densification of bone in the parasagittal regions of the third metacarpal bone greatly increase the risk of condylar fracture in the horse's later life (Parkin *et al.* 2009).

### **Fetlock pain in the 3-year-old and older horse**

#### **Capsulitis/synovitis**

Many older racehorses will also show a degree of increased synovial effusion and thickening of the dorsal joint capsule of the fetlock joint as training progresses. In some cases this is the result of continued training of problem horses at 2 years of age, but in other cases it does seem to represent a genuine inability of the structure of the fetlock joint to cope with training even when the increments in training have been ideally small and graduated (horses which are made of the 'wrong stuff'?).

Because of the pressure of time on the window of racing opportunity for the Thoroughbred, symptomatic treatment of synovial effusion may be more acceptable in the 3-year-old and older horse as these horses have to continue in training if at all



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possible. Intra-articular medication is often a matter of personal choice and many different agents are available. Hyaluronan and triamcinolone acetanide have been shown to have, at best, beneficial effects and at worst the least toxic effects on models of joint degeneration following osteochondral chip fragmentation than many other agents and these would be this author's treatment of choice (Frisbie *et al.* 1997). IRAP medication using the commercially available preparation kits is also available. Other treatment protocols involving various aspects of cold therapy will be discussed.

## **Subchondral bone pain**

In the 3-year-old and older horse progressive sclerosis and densification of the condyles to some degree is almost the norm in the Thoroughbred racehorse, if 'normal' is defined as the majority of any population exhibiting that trait. This stiffening of the bone causes it to lose its normal elastic shock absorbing structure, which has consequences for the wear and damage to the articular cartilage, and may well be the cause of the eventual stages of joint pathology known as osteoarthritis. It is difficult sometimes to understand the origin of the pain which these horses experience. The degree of lameness can be titrated with the degree of exercise in an almost linear fashion over only a few days. The faster these horses exercise, the more lame they will appear the following day. A couple of days of walking or jogging and they move much better. It is therefore hard to believe that the lameness is the result of permanent structural injury and is more likely to be associated with transient phenomena such as intra-osseous bone pressure or congestion of blood vessels in the vicinity causing a dull ache in the bone, which can come and go in step with the use of the joint.

Treatment strategies therefore probably have to be aimed at different sites than the articular surface of the joint. One treatment strategy is to use low-dose nonsteroidal anti-inflammatory drugs continuously throughout training at a level that will not mask the degree of lameness, but which will hopefully limit the degree of inflammatory change in the subchondral bone. Phenylbutazone has proved to be a useful drug in this respect and medication can commence at a normal loading dose but then be reduced to 1 g *per os* on alternate days for a 500 kg racing Thoroughbred. This degree of phenylbutazone medication will minimally mask lameness but appears to be useful in allowing these horses to proceed in training without undue inflammatory change and pain. Aspirin at a low dose similarly may help with limitation in vascular compromise which we know from necropsy studies is part of this syndrome. Similarly, because of the

way in which pain comes and goes with exercise, there is an art to getting horses affected by advanced subchondral bone change to the racecourse pain-free by manipulating their exercise speeds and loading in the run up to the race. This is where alternative minimally weightbearing exercise such as swimming and exercising on a horse walker or sea walker play a vital role. It is often a straight choice between getting the horse to the race completely fit, but suffering a degree of pain which will not allow it to let itself down and perform to its ability in the race, or getting it to the race slightly short in terms of fitness, but willing and able to compete to the maximum of its ability. This is a balancing act and has to be adjusted on an individual basis to every horse. Some trainers are much better at getting this balance right than others.

## **Advanced osteoarthritis**

Degenerative joint disease in the older horse is a chronic, irreversible, progressive ailment for which there is no cure. The clinical signs include lameness, joint stiffness on flexion, excessive synovial effusion with thin watery synovia, and a well documented set of radiological changes. These are difficult horses to keep sound. Strategies involve judicious but sparing use of intra-articular medication in the run up to the race, use of full dose NSAID therapy to cover full speed works, rationing of canter work, with alternate days spent walking and swimming, and careful choice of ground for both training and racing. With all of these aids, it is still often the case that these horses find it hard to compete at the level they once did, and irrevocably slip down into lower and lower grades of racing.

## **Medication, training and racing**

The role of medication during training, which is often a vital part of allowing these horses to achieve their full athletic potential, will be discussed.

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## **NOTES**



15.00–15.25

## Maintaining cartilage integrity

**Lisa A. Fortier**

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The concept of maintaining cartilage integrity is broadly approached from the viewpoint of several different disciplines. From an early age, correcting angular limb deformities with corrective shoeing/trimming or surgical procedures is aimed towards preventing overloading of a particular joint compartment and the subsequent development of osteoarthritis (OA). Foals with carpus varus present the most obvious example where midcarpal joint OA develops at a young age. It is not just the varus or valgus malalignments but correction of flexural deformities, such as acquired contracture of the deep digital flexor tendon (DDF), that lead to early onset OA of the distal interphalangeal joint, should be considered as a means to maintaining cartilage integrity.

As horses enter training, there is a natural adaptation of all musculoskeletal tissues. The well-known remodelling and potential maladaptation of the third carpal and metacarpal III bones in racehorses serve as examples. With specific respect to articular cartilage, although it is structurally fully formed, with a tidemark separating calcified from noncalcified cartilage by the age of 2, cartilage continues to remodel and dynamically responds to the mechanical stresses of exercise. For example, collagen fibril network orientation changes with exercise and this is important because the orientation of collagen fibrils impart the strength of articular cartilage to resist shear stress. There are other structural, compositional and metabolic changes that occur particularly in the trabecular region of carpal bones leading to increased cortical bone stiffness. In support of both concepts mentioned, poor conformation and early speed training have been associated with increased risk in Standardbred racehorses for developing lameness associated with the middle carpal joint. Collectively, this body of work indicates that training methods clearly influence bone and cartilage development and need to be considered in the general scope of maintaining cartilage integrity.

Biomarkers of pending or very early signs of cartilage 'stress', prior to degeneration of articular cartilage would be the ideal means for practitioners to monitor pending cartilage damage and maintain the articular environment. To be truly practical, the ideal biomarker would be detectable in serum and not only in synovial fluid or urine. In a recent longitudinal study trying to identify markers or pending musculoskeletal injury, several serum biomarkers were found to change during training and with injury including osteocalcin, aggrecan synthase and the c-terminal telopeptide of collagen *type I*. The c-terminal telopeptide has also been shown to be a marker of cartilage degradation in horses with joint injury. Computer modelling suggests that these markers could be used to predict musculoskeletal injury. Another type of biomarker is the use of imaging techniques. Methods such as

optical coherence tomography, multiphoton microscopy, and new MRI pulse sequences to detect early articular cartilage injury prior to cartilage degradation are under investigation.

Once cartilage injury has occurred, there are still practical means to maintain cartilage or at least minimise degradation. For example, all carpal osteochondral fragments are not alike. The radiocarpal joint is far more forgiving of the presence of an osteochondral fragment than the middle carpal joint. To preserve a horse's cartilage and athletic career, in general, osteochondral fragments should be removed but those in the middle carpal joint require prompt attention.

Another surgical method to maintain cartilage integrity is through pinning of cartilage flaps using PDS (polydioxanone) pins rather than simply remove large cartilage flaps. PDS pins have been successfully used to reattach large cartilage flaps covering medial femoral condylar cysts and to reattach osteochondrosis (OCD) lesions of the femoral and talar trochlear ridges. PDS pinning is not necessary for all OCD fragments or for all medial femoral condylar cysts and is reserved only for the severe cases due to the added operating room time, surgical expertise required to perform the procedure and cost.

For any of the above mentioned modalities to be successful in preserving articular cartilage, the most important aspect is early diagnosis and intervention prior to the onset of OA.

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### NOTES



## Lameness: Medicating/Treating the Competition Horse

Chaired by Leo Jeffcott

*Sponsored by Boehringer Ingelheim*



16.00–16.25

### Practical chondroprotective drug use

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When confronted with a horse suffering from acute or chronic joint disease, your therapeutic goals should be aimed at both the soft tissue supportive structures and cartilage within the joint. The joint should not be thought of as simply articular cartilage, but rather as an organ consisting of cartilage, joint capsule, ligaments, synovial fluid and subchondral bone. Your therapeutic goals may be to decrease inflammation, alleviate pain or restore the articular environment to slow progression of disease. Your recommended therapy will likely include a combination of supportive care methods, as well as administration of pharmaceuticals and nutraceuticals.

#### Hyaluronan

Hyaluronan (HA) is present within articular cartilage where it is synthesised by chondrocytes and in synovial fluid where it is synthesised by *type B* synoviocytes. Hyaluronan can exist as hyaluronic acid, sodium hyaluronate or as hyaluronate depending on the environment in which it is found, and all terms are used interchangeably. It has been recognised for many years and in several species that in osteoarthritis (OA) the molecular weight and concentration of HA were diminished by one-half to one-third of their normal values, giving rise to the concept of viscosupplementation.

Hyaluronan imparts the viscoelastic nature to synovial fluid, which means it behaves as a viscous solution at low shear rates and is elastic in nature at high shear rates. In synovial fluid HA also lubricates the synovial membrane/cartilage interface (boundary lubrication) and physically excludes active inflammatory components and leucocytes from the joint cavity, a mechanism known as steric exclusion. Hyaluronan has additional direct anti-inflammatory effects and has been shown to decrease fibroblastic pannus formation in osteoarthritic joints.

The functional mechanisms of HA are directly dependent on the molecular weight and concentration of HA. This concept should be kept in mind when choosing from the assorted preparations of HA available for use. The molecular weight of equine synovial fluid HA has been reported to range between 2–3 million daltons, while the reported concentration of HA ranges between 0.33–1.5 mg/ml. For the available HA products, molecular weight and price are typically directly and positively correlated. Therefore, the high molecular weight preparations are recommended due to their increased efficacy and longer duration of action.

The various HA products have excellent safety profiles. Joint flares have been reported to occur in approximately 5% of injections. Joint flares can be difficult to distinguish from joint infection in the first 24 h and may require active treatment such as joint lavage, analgesics, NSAIDs and precautionary antibiotic administration. The clinical presentation of joint flares is typically milder than a joint infection with regard to joint swelling;

lameness, synovial white blood cell count and joint flares are self-limiting. Joint flare also occurs subsequent to the administration of steroids.

The dosing routine for hyaluronan in horses has been arrived at based on clinical impressions, and there is wide variation in how horses respond clinically to HA administration. When administered for idiopathic synovitis, HA would typically be injected intra-articularly (i.a.) every 3–6 weeks for 3 injections. There is no rest period required after HA administration. It is common practice to administer corticosteroids with HA. Combinational therapy of HA/corticosteroid is recommended when treating synovitis that is minimally responsive to HA alone or when treating the coffin joint, which does not appear to respond clinically as well to HA therapy as other joints. The manufacturer recommended doses are based on use in a fetlock or carpus, so when using HA in a large joint such as a stifle, one should probably administer a double dose.

#### Polysulphated glycosaminoglycan

Polysulphated glycosaminoglycans (PSGAGs) are capable of stimulating chondrocyte metabolic activity while concurrently inhibiting the effects of many enzymes involved in cartilage breakdown. PSGAGs also stimulate HA synthesis by the synovial membrane, and have anti-inflammatory and analgesic properties. These beneficial effects on cartilage metabolism have been demonstrated in numerous species in both *ex vivo* and *in vivo* studies and in multiple types of naturally occurring and experimental joint diseases. Despite extensive research, the exact mechanisms of action of PSGAG remain unknown.

Originally, PSGAG was designed and evaluated for i.a. administration. When used i.a., PSGAG was administered at a dose of 250 mg weekly for a minimum of 3 weeks with good clinical results. However, it is well recognised that i.a. infection is potentiated by the administration of PSGAG. In order to circumvent potentially devastating iatrogenic i.a. infections, i.m. administration of PSGAG was evaluated. Following administration of 500 mg PSGAG i.m., therapeutic levels of PSGAG were found in multiple joints for up to 12 h. It is currently recommended that PSGAG be administered i.m. at 500 mg every 3–5 days for a minimum of 5 treatments. It is certainly safest to administer PSGAG i.m., however if one is going to administer PSGAG i.a., then an aminoglycoside (e.g. 250 mg amikacin) should be concurrently injected. While no studies have been performed to determine the effects of amikacin injection on PSGAG activity, there does not seem to be any decline in clinical responses.

#### Pentosan polysulphate

Pentosan polysulphate (PPS) was initially used in humans as an anticoagulant, then as an anti-inflammatory, and most recently



as the major treatment for interstitial cystitis. PPS is made from beechwood shavings and is inexpensive. Both the sodium (NaPPS) and calcium (CaPPS) forms exhibit a wide range of pharmacological activities, with CaPPS reported to have greater bioavailability than NaPPS. PPS stimulates chondrocytes to synthesise new cartilage matrix and inhibits multiple degradative enzymes and inflammatory mediators, thereby attenuating catabolic events responsible for the loss of cartilage matrix in OA joints. There is substantial evidence that PPS can stimulate the synthesis of HA by synoviocytes and stimulate the release of tissue plasminogen activator, consequently increasing fibrinolysis with resulting improvement in synovial membrane and subchondral blood flow.

## Nutriceuticals

There are numerous components recommended in the treatment of joint disease including: chondroitin, glucosamine, Perna mussel, ascorbic acid and omega-3 fatty acids. Few have been investigated in the laboratory and in thorough clinical trials. Because nutraceuticals are not drugs, they are not regulated by government agencies in most countries. When tested, the label claims and actual components of most joint supplements do not match. The equine formulation that has been most extensively studied is Cosequin (Nutramax). That is not to say that none of the other joint supplements work, but there is no way to determine if they will. One way to determine if a nutraceutical is efficacious is to suggest that a client start their animal on Cosequin to see the maximal effect that might be obtained. They can then change to a less expensive brand and use their judgement to see if it works as well. Cosequin does have 2 disadvantages: 1) it is expensive and 2) is only obtainable through a veterinarian.

## Tetracycline family antimicrobials

Tetracycline antimicrobials such as minocycline and doxycycline have long been advocated as treatments for rheumatoid and OA in humans. Clinical signs of improvement attributed to tetracycline therapy include decreased joint pain and suppressed progression of articular cartilage erosion. In equine practice, horse owners frequently report that their lame horse became sound and 'never went better' when placed on doxycycline pending test

results for Lyme Disease, despite the fact that the vast majority of those horses tested negative for Lyme Disease. The effectiveness of oral doxycycline and minocycline in the treatment of OA is due at least in part to the ability of tetracyclines to reduce matrix metalloproteinase (MMP) activity with joints through binding of the divalent cation zinc which is required to convert pro-MMP to active MMP.

In our laboratory, *in vitro* and *in vivo* studies were performed to assess the capacity for doxycycline and minocycline to alleviate cartilage degradation associated with treatment of catabolic mediators interleukin-1 (IL-1) and matrixmetalloproteinase-13 (MMP-13). Our studies indicate that both doxycycline and minocycline exert their primary effect on the synovium which in turn results in protection of the articular cartilage from the degradative effects both catabolic mediators IL-1 and MMP-13. Interestingly, our *in vivo* studies show that doxycycline accumulates in the synovial fluid to a greater extent than minocycline. Current studies are being performed to determine the minimal dosing regimen needed to achieve anti-inflammatory, but not antimicrobial levels in synovial fluid. The long-term effects of low-dose doxycycline on antimicrobial susceptibility and photosensitisation are unknown, but there are studies of people on long-term, sub-antimicrobial doses of doxycycline and minocycline with rare side effects reported. Tetracycline antibiotics are highly plasma protein bound and should not be administered in conjunction with other highly protein bound drugs such as phenylbutazone.

With any of the chondroprotection medications, it is important to assign a rehabilitation programme that includes corrective trimming/shoeing, a detailed exercise programme and weight loss if necessary in order to achieve maximal success.

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## NOTES



# Hall 1A ■ Friday 9th September

16.25–16.50

## Practical use of nonsteroidal anti-inflammatory drugs in competition horses

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The nonsteroidal anti-inflammatory drugs (NSAIDs) phenylbutazone (PBZ), flunixin meglumine (FLU), ketoprofen (KET), dipyrone (DIPY) and meloxicam (MEL) are reliable analgesics frequently employed in the appropriate treatment of horses with musculoskeletal and/or visceral pain. Legitimate uses of these drugs include reducing pain during healing of musculoskeletal injuries, for management of chronic musculoskeletal ailments and transient relief of abdominal pain. Concurrent use of 2 NSAIDs enhances the likelihood of toxic effects. The practice of 'stacking NSAIDs' is attractive because the analgesic effects of 2 NSAIDs may be synergistic (Keegan *et al.* 2009).

National laws in 9 countries in Europe regulate veterinary use of NSAIDs in horses. Legislation in France and Sweden clearly forbids the use of NSAIDs in sport horses. Laws are less clear in the other 7 countries and the legality of using NSAIDs in competition horses must be explored on a country-by-country basis (Lazarus 2010). European Union regulations prohibit giving PBZ to any horse that may enter the food chain. Horses in the EU, including Great Britain, that are not intended for human consumption may be treated with PBZ.

Analgesia induced by NSAIDs may improve the ability of performance and racing horses to excel in their disciplines and may confer an unfair advantage in competition. Administration of NSAIDs to horses with musculoskeletal injuries or abdominal pain during competition may mask pain and result in further injury or illness. Most racing jurisdictions in Europe and Asia forbid horses to compete under the influence of NSAIDs. The FEI has developed a list of 1103 medications that are forbidden in horses competing in FEI-sanctioned events. Of these drugs, 137 are considered controlled medications that have a legitimate use in modern veterinary therapeutics, but that are forbidden during competition. Clearly, veterinarians must know how to manage horses during the precompetition period so as not to allow horses to have unacceptable levels of PBZ, FLU, KET, DIPY or MEL, all NSAIDs on the FEI controlled substance list.

Administration of medications during competition or on race day is controlled by invoking Screening Limits of Detection - SLOD (FEI) or International Screening Limits - ISL (EHSLC) respectively. SLOD and ISL are determined by administering the drugs of interest to healthy, resting horses and measuring plasma concentrations of drugs during the post administration period. The governing body sets the SLOD or ISL at a value designed to preclude use of a drug so close to competition that the drug is still effective, but to allow judicious 'best practice' use of needed medications in the precompetition period. All drug detection laboratories sanctioned by the governing bodies adopt the prescribed SLOD or ISL so that results of testing will be comparable among them. The SLOD or ISL is then translated into a 'Detection Time' that corresponds to the number of hours after administration required for mean plasma drug concentrations to reach the SLOD/ISL. Detection times are published so that veterinarians responsible for the care of racing and competition horses can determine a safe interval before the competition (Withdrawal Time) during which horses will not be treated with forbidden substances. The FEI has published detection times for 13 drugs including these 5 NSAIDs.

Drug	Dosage/route	Detection time
PBZ: (phenylbutazone)	4.4 mg/kg bwt, q. 12 h for 5 days <i>per os</i>	168 h (7 days)
Equipalazone	8.8 mg/kg bwt i.v.	168 h (7 days)
Phenylarthrite	8.8 mg/kg q. 12 h <i>per os</i>	168 h (7 days)
FLU* (flunixin meglumine)	1 mg/kg bwt i.v.	144 h (6 days)
Finadyne		
KET** (ketoprofen)	2.2 mg/kg bwt q. 24 h i.v.	96 h (4 days)
Ketofen		
DIPY (dipyrone)	30 mg/kg bwt i.v.	72 h (3 days)
Metamizole		
MEL (meloxicam)	0.6 mg/kg bwt i.v.	72 h (3 days)
Metacam	for 14 days	

\*Re-uptake of oral drugs through ingestion of manure or contaminated bedding can result in prolonged detection times. \*\*Topical use of ketoprofen has resulted in prolonged detection times.

The selection of the SLOD/ISL directly determines the dosage, frequency and duration of treatment that will result in detection of drug concentrations below the prescribed levels. The difficulty for regulatory bodies and consequently for veterinary practitioners is distinguishing between physiologically relevant drug concentrations that might enhance performance or jeopardise horse welfare and irrelevantly small but detectable concentrations of the same drugs. Unquestionably, shorter withdrawal times allow veterinarians to use drugs in the best interests of their equine patients, closer to the competition.

The potentially negative effects of NSAID therapy on the welfare of competition and racing horses are difficult to quantify. Currently the only outcome data available are rates of catastrophic injuries, and most of the information is based on racehorses. At the FEI NSAID summit in Lausanne, Lynn Hillyer, Chair of the EHSLC, reported data from one racing jurisdiction in North America that showed an increasing rate of catastrophic injury during racing as the SLOD for PBZ increased, and a decreasing rate when the SLOD was reduced. Statistical significance of these comparisons was not provided. During 2009, in horseracing in Canada and the USA, the fatality rate was 2 per 1000 starts.

SLOD for NSAIDs used in horses competing under USEF Drug and Medication rules are more permissive than those used by racing jurisdictions and the FEI. Among horses competing under USEF Drug and Medication Rules in the USA in 2009, there were 0.01 catastrophic injuries per 1000 starts.

What can we conclude, in the context of horse welfare and use of NSAIDs, from this comparison between fatality rates of North American racing and performance (nonracing) horses? Firstly, the profiles of racing and performance horses are very different with respect to average age, number of starts per year and average years in competition. Secondly, the speed required of racehorses is much greater than that needed by most other performance horses. Three-day event horses do perform at speed and the risk of catastrophic injury is probably higher in this discipline. With respect to welfare, it is likely that the use of NSAIDs in horses that perform at speed will pose a greater risk than for horses that compete at a substantially slower pace.



# Friday 9th September ■ Hall 1A

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Veterinarians and regulatory bodies must continue to look for a balance between maintaining the level playing field and protecting horse welfare during competition, and allowing 'best veterinary practices' for treatment of horses before competition.

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## NOTES

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# Hall 1A ■ Friday 9th September

16.50–17.15

## Practical autologous product use - IRAP/PRP/Stem cells

**Lisa A. Fortier**

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Autologous products are aimed at using the body's own resources to enhance functional tissue regenerative. The main autologous products currently used in equine practice include autologous conditioned serum (ACS)/interleukin-1 receptor antagonist protein (IRAP), platelet rich plasma (PRP) and stem cells of several varieties. Each of these therapies is relatively new so there is very limited clinical data accumulated to date.

### Autologous conditioned serum (ACS)

ACS is generated by incubation of venous blood using the same process as IRAP, but for primarily legal reasons, it is called ACS. It is thought to act by blocking the receptor to the inflammatory cytokine interleukin-1 (IL-1). When injected intra-articular into horses with surgically created synovitis/early arthritis, ACS resulted in decreased synovial hyperplasia and lameness compared to placebo treated groups. There is a newer generation of ACS termed IRAP II which boasts increased IRAP levels and is presently being tested by the equine group at Colorado State University.

### Platelet rich plasma (PRP)

PRP is defined as plasma with a 2- or more fold increase in platelet concentration above baseline levels or  $>1.1 \times 10^6$  platelets/ $\mu$ l. PRP is generated primarily by centrifugation or gravity filtration. There are differences in the volume of blood required, time and speed of centrifugation, addition of an activating agent, leucocyte concentration, method of delivery, and qualitative/quantitative differences with respect to final PRP volume and final platelet and growth factor concentrations between the available systems. Overall, the final PRP platelet concentration is 2–8 times over baseline. A key concept to understand is that PRP is a milieu of growth factors, cytokines, perhaps some RBC, and all the proteins, electrolytes, hormones, etc. that are present in plasma.

The concept that PRP would improve joint or tendon disease is based on the physiological role of platelets in wound healing. Through a modulation of the inflammatory response, promotion of local angiogenesis, attraction of fibroblasts and local stem cells to the site of injury and an induction of autocrine growth factor production by uninjured adjacent cells, platelets and their products are instrumental in normal tissue repair and regeneration.

Once isolated, the PRP can be injected into a joint or tendon with or without an activating (clotting) agent. When exposed to collagen or cells, platelets will be naturally activated and the clotting cascade will begin. A PRP clot serves as a fibrin matrix which serves as a scaffold for tissue repair and a reservoir for retention and slow release of growth factors.

The application of PRP in joints and tendons is relatively new and therefore there are limited clinical publications investigating its use. Chondrocytes and MSCs exposed to PRP both have significantly increased cell proliferation and cartilage extracellular matrix synthesis of proteoglycans and collagen *type II* compared to controls. Synoviocytes from OA patients cultured in PRP demonstrated significantly increased hyaluronic acid production and secretion, suggesting that PRP could potentially serve as an endogenous source of chondroprotection and joint lubrication following intra-articular application.

### Stem cells

Stem cells hold tremendous promise for the treatment of musculoskeletal injuries. There are several therapies currently being employed or marketed under the guise of stem cell therapy; therefore understanding the fundamentals of stem cell biology is important for choosing the appropriate treatment type and application protocol.

Stem cells are broadly defined as undifferentiated cells that possess the ability to divide for indefinite periods in culture and may give rise to highly specialised cells of each tissue type (mesoderm, ectoderm, endoderm). There are 2 broad categories of stem cells, embryonic and adult-derived. Embryonic stem cells (ES cells) used to be defined as those derived from preimplantation embryos. More recently advances have been made so that ES cells can be generated from adult fibroblasts using many of the same technologies that were used to clone Dolly the sheep. These cells are known as iPS (induced pluripotent stem) cells. Adult-derived mesenchymal stem cells (MSC) can be obtained from bone marrow, fat, umbilical cord blood, muscle, and many other tissues including cartilage, trabecular bone and tendon. Two techniques are currently available for the treatment of cartilage, tendon and ligament injuries with MSCs and they include bone marrow-derived MSCs and adipose derived MSCs. These are the only stem cells that are being practically used for equine lameness at the present time. Each technique has its strengths and weaknesses.

### Stem cells in cartilage regeneration

Our laboratory generated a stem cell concentrate from sternal bone marrow aspirate and tested it in an equine model of articular cartilage loss. Compared to other cartilage repair procedures, this method has the advantages of being a point-of-care technique (no laboratory culture period is necessary) that is autogenous, arthroscopically applicable, and delivers all 3 components believed to be important for cartilage regeneration; cells, growth factors and a scaffold. We treated 10 horses in which 15 mm full thickness defects were made on the lateral trochlear ridge of the femur and 18 clinical cases. No animal had post operative synovitis of other detectable adverse reaction. Results indicate significant improvement in cartilage repair at 4 and 8 months post operatively. There are no comparative studies for practical cartilage repair in horses using other types of stem cells.

In a study comparing MSCs to adipose-derived cells (A-MSCs) in horses with surgically created carpal osteochondral fragments which mimics synovitis or early OA, free i.a. injection of MSCs were superior to A-MSCs in restoring the normal articular environment.

### Stem cells for tendon injuries

For BM-MSCs, bone marrow aspirate-tissue culture adherent cells are expanded in the laboratory. A 2–4 week culture period is needed to expand these selected cells until they are available in sufficient quantity for implantation under standing sedation into the tendon core lesion using ultrasound guidance. Data in press from Roger Smith's group with an impressive minimal 2 year follow-up period indicates that for Thoroughbred racehorses treated with autologous cultured, bone marrow derived MSCs that the re-injury percentage of all racehorses with follow-up



(n = 113) undergoing MPC treatment was 27.4%, with the rate for National Hunt racehorses being 25.7% (n = 105) and for flat racehorses 50% (n = 8), which was significantly less than published data for horses treated in other ways. No relationship between age, discipline, number of MPCs injected, or interval between injury and implantation was found.

For A-MSCs, the technique is based on data which suggested that adipose-derived MSCs exhibited a similar degree of multipotentiality to BM-MSCs although in many studies they performed less well than BM-MSCs in differentiation assays. No references regarding the application of A-MSCs in naturally occurring equine tendonitis are presently available despite the reported treatment of thousands of racehorses.

There are encouraging aspects to all the above mentioned technologies although definitive proof of efficacy is still lacking. Furthermore there have been no direct comparisons between the autologous products so it is very difficult to recommend one treatment over the other or to outline specific treatment modalities for different types of injuries and tissues. It must be remembered that there are still considerable gaps in our knowledge although the technology is developing rapidly.

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### NOTES



# Hall 1A ■ Friday 9th September

17.15–17.40

## Practical corticosteroid use

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Corticosteroids remain one of the most frequently used drugs for the treatment of orthopaedic conditions in competition horses. This presentation will concentrate on the intra-articular use rather than the less frequently employed systemic and topical routes.

Corticosteroids have traditionally received a rather 'bad press' with respect to their potential for causing cartilage damage. Certainly the drugs can be over-used but if the right products are used in appropriate dosages, in horses with actual joint disease they can be most helpful drugs. The majority of research work suggests that triamcinolone is more chondroprotective than betamethasone or methylprednisolone acetate (MPA), and is generally considered the drug of choice in our practice. MPA has been shown to have deleterious effects on cartilage and its use is not encouraged. With appropriate aseptic technique for injection the risk of infection is very low in practical terms. I would not normally administer intra-articular antibiotics as a routine, but would inject 100 mg of amikacin along with corticosteroids if the joint had been blocked in the previous week.

Injection with corticosteroids is often combined with hyaluronic acid. There is no firm evidence that this is beneficial but it may possibly ameliorate some of the deleterious effects of corticosteroids as well as having a beneficial effect in its own right and many clinicians comment that they may get a longer duration of positive effects after medication with steroids if they are combined with hyaluronic acid. Because of the risk of laminitis I would normally use a maximum dose of 20 mg of triamcinolone in a sport horse. Racing Thoroughbreds seem more

resistant to developing laminitis and a dose of up to 40 mg would commonly be used in our practice. A dose of 5–10 mg per joint would be used generally, depending on the number of joints to be treated. Methylprednisolone acetate still tends to be used more in low-motion joints and for infiltration into the back and sacroiliac region. I have used combined injections of up to 200 mg of methylprednisolone acetate with the simultaneous injection of 20 mg of triamcinolone in cases needing medication of multiple regions.

In a retrospective study in our practice, only 3 horses out of 2000 receiving joint injections developed signs of laminitis and 2 of these were transient bouts in ponies which had previously had laminitis. Thus the owners must be warned of the risk of laminitis, albeit a very low risk if the products are used appropriately. The limbs are routinely bandaged for 24 h after injection. My standard routine is to box rest the horse for 48 h from the time of injection, followed by 2 days of box rest and hand walking exercise, followed by a gradual return to exercise, depending upon the degree of lameness and the pathology being treated. I prefer not to treat a joint more than 2–3 times annually and if there is a diminishing response to medication, then the diagnosis should be reviewed and alternative treatments considered.

Withdrawal periods for medication control at competitions may also influence the choice of corticosteroid. MPA has a long and variable detection time, and its use during the competition season is discouraged. I use a withdrawal period of 13 days for triamcinolone and 3 days for dexamethasone.

### NOTES

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