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1. Introduction

When I graduated, it was accepted wisdom that lameness was assessed by watching horses move in hand. As an experienced rider, I became increasingly aware of the value of ridden exercise to assess lameness and performance. This has become an essential part of lameness and poor performance diagnosis. Diagnostic analgesia is now commonplace, but it is only comparatively recently that we have become aware of some of its limitations. Since I graduated, diagnostic imaging has evolved from radiography to include ultrasonography, nuclear scintigraphy, magnetic resonance imaging (MRI), and computed tomography. We are still learning how to interpret what we find and to integrate this with clinical findings. I aim to discuss the science and art of lameness diagnosis.

Accurate lameness diagnosis requires recognition of the lame limb(s), identification of any significant palpable abnormalities, determination of the source(s) of pain, and appropriate diagnostic imaging to identify the potential cause(s) of pain, with high quality images, correctly interpreted. This is an acquired art requiring careful observation, pattern recognition, a logical deductive process, and a questioning mind. By continuing to look, I believe that we can all continue to learn and to make new discoveries. My aim is to review some of the advances in lameness recognition, diagnostic analgesia, and advanced imaging, especially MRI. With respect to MRI, my focus is on the foot, because this is the area about which we have learned most and that has the best validation by comparison of imaging findings with postmortem examinations. However, throughout, I aim to emphasize the complexities and the many unanswered questions that remain. This is a challenging field.

2. Looking at the Horse and Learning to Read the Horse

The identification of lameness is an art, a skill that with practice and guidance can be enhanced. It requires an understanding of normal gaits and how they may be modified under a variety of circumstances. I use a simple grading system (0–8) that is applied independently at a walk and a trot, in straight lines and in circles (on both soft and firm and/or hard surfaces and both to the left and to the right), and ridden (at rising trot [sitting on either the left or right diagonals] and sitting trot, and both to the left and the right, and if necessary when performing specific movements, eg, a 10-m diameter circle to the left, or half pass to the right). In my view, this gives a much more accurate picture of the lameness than the American Association of Equine Practitioners (AAEP) 0 to 5 scale, especially if also embellished by some verbal description of the gait, for example, “a shortened cranial phase of the stride,” “marked irregularity of rhythm” (appreciated by both visual assessment and listening to the gait), and “toe drag,” or an “intermittent, hopping lameness.” The system is effectively a numerical rating scale, because the grades have no definitions other than mild, moderate, and severe (0 = sound; 2 = mild; 4 = moderate; 6 = severe; 8 = non-weight-bearing). Thus, a subtle lameness may be grade 1, whereas an obvious lameness may be grade 5. This requires a good understanding of the breadth of gait abnormalities that may be encountered. I find it a workable system, because the manifestations of lameness vary so much among horses, depending on the source of pain and its severity, and each individual horse’s response to that pain. Some features, such as extension of the fetlock, are more accurately assessed at a walk than at a trot because of the slower foot-fall. The longer stance phase at a walk,
and therefore the greater extension of the distal interphalangeal (DIP) joint, may make some foot-related lameness (e.g., injuries of the deep digital flexor tendon [DDFT] or a collateral ligament of the DIP joint) more obvious at a walk than at a trot. In my opinion, a 0 to 8 system applied independently for each situation under which the horse is examined, supplemented by verbal qualifications, offers a far more flexible system than that of the AAEP grading scale and provides sufficient grades to cover a wide spectrum of gait abnormalities and facilitates grading of change in lameness after local analgesia.

With both forelimb and hindlimb lameness, it is useful to assess the horse walking in small circles, which often accentuates foot-related lameness but is not specific for foot pain. This also gives the opportunity to assess the flexibility of the horse’s neck and back, the ease with which it crosses over its hindlimbs, and to detect any gait abnormalities suggestive of a component of ataxia, such as toe drag, hindlimb circumduction, leaving limbs in abnormal positions, irregularly sized steps, and interference between limbs. Evaluating the horse step backward can also give additional information about the presence of shivering and flexibility of the lumbosacral region.

Whichever lameness grading system is used, it is important to recognize that for consistency of results, the circumstances in which a horse is examined must remain the same. The speed of trot may influence the degree of lameness; some horses may try to slow the speed to protect themselves. Lameness may be accentuated on a slight downward slope compared with a horizontal surface or on a deeper sand surface than on a firm, waxed rubber and sand arena. Detection of these variations under different conditions may give an indication of the likely source of pain causing lameness. For example, hindlimb lameness that is worse on a circle on a hard surface is most likely to reflect foot pain.

Assigning a grade is not always straightforward because there are so many ways in which the gait may be modified. When a horse with unilateral hindlimb lameness is moving in straight lines, there is usually some degree of asymmetry of movement of the hindquarters. There may be reduced extension of the fetlock joint, reduced flexion of the hock, toe drag, or alteration in stride length; the horse may move on three tracks, usually drifting away from the lame limb; alternatively, the lame hindlimb may deviate axially during protraction or less commonly is swung outward during protraction. The rhythm may be altered both audibly and visually. Irrespective of the degree of hindquarter asymmetry, there may be a head-nod mimicking ipsilateral forelimb lameness.

Our ability to detect asymmetry of movement of the hindquarters is limited. A computer model was devised to determine how capable we are of assessing hindlimb lameness, on the basis of evaluation of movement of the tubera coxae. The model had two forms, one in which there was random asymmetry of movement between two objects and the other that simulated the patterns of movement seen in a lame horse. In the first model, no differences were seen in the skill of inexperienced assessors compared with experienced clinicians, suggesting that there are no innate differences in the ability to detect asymmetry. However, the experienced clinicians performed best with the real lameness-based data. Nonetheless, even with lameness-based data the accuracy of detecting asymmetry of movement simulating low-grade lameness was poor: asymmetries in movement of <25% could not be detected. In a more recent study of so-called normal horses, assessed by veterinarians with a range of experience, and also objectively with the use of inertial measurement units, an expert was able to detect lameness manifest as asymmetries of 10%. However, the expert was not confined to assessing asymmetry alone and could use any technique to determine whether the horse was lame or sound.

I focus on both the tubera coxae and the tubera sacrale, although it may be impossible with a horse with a naturally high tail carriage, such as Arabians, some Warmbloods, and gaited horses, when the tail conceals the tubera sacrale. A skewbald or piebald horse with one white hindquarter and one colored hindquarter, or a horse with unilateral gluteal muscle atrophy in either the lame or the non-lame limb, or a horse with asymmetry in height of the tubera sacrale, or an excitable horse that will not trot straight potentially confound our interpretation. A bilaterally symmetrical hindlimb lameness may manifest merely as a short stride, stiffness, and lack of hindlimb impulsion. All of these factors potentially compromise our ability to detect the lame limb(s). Some can be solved—an excitable horse can either be worked or sedated, but you cannot change the markings of a colored horse or the positions of the tubera sacrale. The use of markers placed on the tubera coxae may facilitate assessment.

With forelimb lameness, there is usually abnormal movement of the head and neck, although in a horse with a naturally short-striding gait, with a tendency to roll from side to side, this may be difficult to discern. Assessment of head and neck posture is also important for detection of neck pain that can result in alterations in gait.

I believe that it is important to assess the horse moving from behind, from the front, and from the side to assess all aspects of the gait and to both watch and listen to the horse. Irregularities of gait may be emphasized by listening to the limbs striking the ground or hearing a toe drag during protraction. An abnormally deliberate placement of hindlimbs to the ground may suggest a neurological component to the gait abnormality or a mechanical component such as stringhalt or fibrotic myopathy. Assessment of foot placement—toe first or flat-footed, lateral side first or flat, in line with the ipsilateral foot.
or to one side—and breakover may reflect the site of pain or help to explain why lameness may have developed. Viewing the horse from behind may reveal that a hind foot is placed axially and the horse appears to collapse laterally over the fetlock, with an unusual wobble of the hock. This may be normal for the horse but may be a predisposing cause for lameness. Abaxial sliding of a hind foot during landing may reflect ataxia. Axial deviation of a hindlimb during protraction may be a way of reducing proximal limb flexion associated with lameness.

Careful observation of the horse from the front at the walk may reveal slight bulging of a shoulder: “shoulder-slip,” usually a manifestation of brachial plexus injury. Watching the hocks carefully from behind may reveal intermittent, slight movement of the superficial digital flexor tendon (SDFT) reflecting subluxation secondary to a partial tear of the retinaculum. Unequal height of the hocks may reflect partial disruption of gastrocnemius or coxofemoral joint subluxation.

No grading system can take into account a bilaterally symmetrical lameness. A lameness grade ascribed to the lamer limb or lamest lameness in the presence of an asymmetrical bilateral lameness or concurrent forelimb and hindlimb lameness can be potentially highly misleading. With a bilaterally symmetrical forelimb lameness, the horse may show only a subtle shortening of stride. Abolition of pain in one limb by local analgesia may reveal either a low-grade lameness in the contralateral limb or a moderate to severe lameness, but this can be highly unpredictable on the basis of the initial clinical assessment.

Many but not all forelimb lamenesses are accentuated on a circle. I prefer to see a horse lunged on a circle rather than led, because it is easier to assess any adaptations of the horse’s balance, posture, and rhythm. Although historically it has been assumed that most horses with foot pain have lameness accentuated with the lame(r) limb on the inside of a circle, approximately one-fifth of 718 horses were lamest with the lame(r) limb on the inside of a circle.

Forelimb lameness associated with proximal suspensory desmitis is usually worse with the lame(r) limb on the outside of a circle on a soft surface. Bilateral forelimb lameness sometimes manifests as a shortness of stride, hurried rhythm, loss of balance, and a tendency to look out of a circle. Foot-related pain and some other sources of pain causing lameness may be worse on a firm or hard surface compared with a soft surface. However, selection of an appropriate hard surface is crucial because the horse must move confidently; a slippery surface may result in marked shortening of stride and apparent loss of balance in a clinically normal horse, especially those that move extravagantly. I use a gravel surface on an incline immediately adjacent to purpose-built modified tarmac; the gravel provides excellent grip, and the downward slope often accentuates lameness. A horse that trots sensibly can be assessed moving from the gravel to the harder tarmac surface and back onto the gravel. A horse may move with a rather restricted stride, and, if asked to move forward more freely, may break to canter rather than increase the stride length. This is usually a manifestation of hindlimb lameness but can also reflect forelimb lameness.

Evaluation of the horse moving in circles on the lunge can also be helpful for assessment of hindlimb lameness, with some lamenesses becoming more evident with the lame hindlimb on the inside of a circle and some with the lame hindlimb on the outside of a circle, and may change depending on the circumstances under which the horse is assessed. In my experience, this is not necessarily related to the source of pain causing lameness. However, with mild lameness that is not modified by being on a circle, detection of hindlimb lameness may be more difficult than in straight lines. The pelvis tilts inward on the lunge, therefore comparison of movement of the tubera coxae is more difficult than in straight lines. Lameness may modify the horse’s body posture, with a tendency to lean in so that the body is no longer perpendicular to the ground (Fig. 1). This makes evaluation of pelvic symmetry even more difficult, but alterations in rhythm may make it easier to detect the lame limb. The lame hindlimb may cross in under the horse’s body when on the inside of a circle and may have an accentuated toe drag. When on the outside of a circle, a
shortened cranial phase of the stride of the lame hindlimb may be exaggerated.

It is also crucial to recognize the influence that forelimb lameness can have on the hindlimb gait and the effect that hindlimb lameness can have on forelimb gait. It is well recognized that hindlimb lameness can induce a head-nod mimicking ipsilateral forelimb lameness. It is less well recognized that forelimb lameness may induce asymmetry of the hindlimb gait. By observing the horse under a variety of circumstances, it may be possible to differentiate between genuine concurrent forelimb and hindlimb lameness and an apparent lameness that is secondary. However, it may or may not be possible to determine whether there is concurrent forelimb and hindlimb lameness without using local analgesia. Thus, each lameness must be graded independently initially, in the knowledge that abolition of pain in, for example, the hindlimb may abolish both the hindlimb lameness and the apparent (secondary) forelimb lameness.

Assessment of a horse moving on the lunge can help to determine if a head-nod present in conjunction with a hindlimb lameness is related to the hindlimb lameness or reflects a forelimb lameness. If the hindlimb lameness deteriorates on the lunge but the head-nod remains unchanged, or if the head-nod is worse but the hindlimb lameness stays the same, it can probably be concluded that there is coexistent forelimb and hindlimb lameness. This requires verification with the use of local analgesic techniques, further discussion of which is beyond the scope of this commentary.

Although overt lameness is rarely seen on the lunge in canter, assessment of canter and transitions from trot to canter and canter to trot can give useful additional information. A bilateral forelimb lameness may manifest as a short-striding, jarry canter. Hindlimb lameness may result in the hindlimbs being placed closer together than normal, so-called “bunny-hopping” when extreme. There may be reduced extension of the distal limb joints of the lame hindlimb, especially with the lame limb on the inside of a circle. The fetlock may appear to knuckle forward slightly during the stance phase, with incomplete loading of the heel. Alternatively the horse may be on the forehand and croup high in canter (Fig. 2). Curiously this gait adaptation is sometimes also seen in a horse with bilateral forelimb lameness. The horse may repeatedly become disunited (ie, change legs behind, while maintaining the correct forelimb lead); however, this may be a normal feature of a young unbalanced horse that lacks hindlimb strength and coordination. Irregular steps behind in a transition from trot to canter or from canter to trot are a manifestation of hindlimb lameness. Stepping short behind and maintaining a croup-high posture in a downward transition may reflect bilateral hindlimb lameness.

Mild hindlimb lameness may still be barely detectable, and assessment of the horse ridden may be crucial. It is important to recognize the influence of the weight of a rider on hindlimb gait, especially when in rising trot. The lameness is usually accentuated when the rider sits on the diagonal of the lame hindlimb and the horse feels more uncomfortable to the rider on this diagonal. The horse may adjust its rhythm to try to shift the rider back to the opposite diagonal. A heavier rider is more likely to accentuate lameness than a lighter rider. A subtle lameness may still be unapparent and riding consecutive 8- to 10-meter diameter circles to the left and to the right may induce irregularities of rhythm reflecting lameness and a change in the speed, with the horse slowing down when uncomfortable. Likewise, performing so-called lateral movements such as shoulder-in, travers, and half-pass may induce gait irregularities that are otherwise not apparent. In some horses, the only manifestation of a hindlimb gait abnormality may be a tendency to change limbs.
behind in canter, to become disunited. Alternatively, the horse may have difficulties in performing canter pirouettes in one direction, or flying changes either from left to right, or from right to left. It is important to be aware of the influence of a rider; an unskilled rider who lacks balance may induce lameness, whereas a highly skilled rider may conceal lameness. A rider who is constantly moving their hands may cause irregular movement of the horse’s head, which may mimic forelimb lameness. A rider who restricts the horse with their hands may cause a loss of hindlimb propulsion and/or irregular steps. Unless the rider is sufficiently skilled, lameness that is only apparent when a horse is working in maximum collection may not be apparent. Evaluation of the horse working to a contact, “on the bit” compared with on a long rein, may reveal that lameness is apparent under one circumstance but not the other.

The tendency of the saddle to consistently slip to one side may be a manifestation of hindlimb lameness, with the saddle usually slipping to the side of the lame(r) limb but sometimes slipping to the side of the non-lame or less lame limb (Fig. 3). This presumably reflects the different ways that horses modify their gait in the face of hindlimb lameness. Saddle slip with two riders was present in 38 of 71 (54%) horses with hindlimb lameness and was abolished when lameness was resolved with the use of diagnostic analgesia in 37 of 38 (97%) horses, verifying a causal relationship. The saddle slipped to

Fig. 3. A, Rear view of a 9 year old Prix St Georges Warmblood dressage horse, with a low-grade left hindlimb lameness, most apparent in left half-pass. The saddle has slipped to the left (toward the side of the lame limb), a persistent feature on the left rein in both trot and canter. Saddle slip was resolved when the lameness was abolished by diagnostic analgesia. B, Rear view of a small riding horse, with left hindlimb lameness. There is saddle slip to the right on the left rein, which was worse in circles than in straight lines. Abolition of the lameness by diagnostic analgesia resolved the saddle slip.
the side of the lamest limb in most horses (32/37 [86%]). Saddle-slip persisted in one horse in which the flocking of the saddle was asymmetrical. When ridden in a better-fitting saddle, no saddle-slip was observed. Saddle-slip associated with hindlimb lameness was not related to the degree of lameness. The observation of saddle-slip may actually highlight the presence of lameness. Saddle-slip was usually worse in rising trot than in sitting trot, in circles compared with straight lines (irrespective of the appearance of the lameness), in canter than in trot, and with a lighter rider compared with a heavier rider. No horse had significant left-right asymmetry of the shape of the thoracic region. However, it is important to recognize that saddle slip may also be induced by an ill-fitting saddle, crookedness of the rider, or asymmetry of the horse’s back.

The way in which a multilimb lameness may influence a horse’s performance when ridden must also be understood. There may be no overt lameness; therefore grading the degree of lameness may be impossible, although the gait of the horse is altered and performance compromised. For example, there may be no detectable lameness, but on the lunge a horse may lean the body inward and look to the outside, while the inside hindlimb crosses in under the body toward the contralateral forelimb during protraction. This may well be a manifestation of lameness, but is not quantifiable and can only be described. It is also not specific for lameness, because a young unbalanced horse, lacking musculoskeletal strength and coordination, may show a similar gait. When ridden, the same horse with poor performance may lack hindlimb engagement and impulsion and tend to be croup-high in downward transitions. Superficially, to an inexperienced eye, the horse may appear normal and the appearance may markedly underplay the discomfort experienced by the horse. Leaning on the bit, taking an uneven contact, tilting the head (Fig. 4), opening the mouth, raising the head, becoming overbent, stiffness in the neck or back, crookedness, difficulties to turn in one direction, reluctance to go forward or undue hurrying, evasiveness, and spoikiness can all be manifestations of lameness. The complete transformation of balance, impulsion, engagement, quality of contact with the bit, and willingness to work after appropriate local analgesia to abolish subclinical lameness will emphasize the degree of pain that the horse is experiencing.

Problems that are only evident ridden have frequently been attributed to pathology in the thoracolumbar-sacral spine. Primary back pain may induce back stiffness and limited hindlimb impulsion or a restricted gait all round. However, lameness can induce similar symptoms. Likewise, neck stiffness is often attributed to neck pain but can also be a manifestation of lameness. Comparison of a horse ridden in rising and sitting trot can help to identify a component of primary back pain. In sitting trot, a horse with back pain is more likely to alter its rhythm and/or speed and alter the position of the head and neck, becoming slightly above the bit, compared with a lame horse.

Some lameness may only be apparent under specific circumstances. For example, with forelimb lameness, the horse may be unwilling to land with the left forelimb leading in canter. The ground reaction force is greater in the trailing (non-lead) forelimb on landing, so unwillingness to land with the left forelimb leading is protecting the right forelimb from concussion. However, there is greater stress on the suspensory apparatus of the leading forelimb, so unwillingness to land with the left forelimb leading could reflect suspensory injury in the left forelimb. Pushing off the hindlimbs unevenly can cause a horse to jump crookedly across a fence, with the hindlimbs drifting toward the lame limb. With low-grade hindlimb problems, lameness may manifest as unwillingness to perform flying changes from left to right in canter (or vice versa) or inability to maintain a regular rhythm in piaffe, passage, or pirouettes. The hindlimbs may be unable to sup-
port weight normally, so there may be a tendency to place them more closely together in canter. Particularly when the lame limb is on the inside of a circle, it will not be protracted as far as normal and will therefore be placed closer than normal to the outside hindlimb. A stiff, stilted canter or poor hindlimb propulsion in canter, with the hindlimbs trailing, may be the most obvious manifestations of a bilateral hindlimb lameness.

There are also some specific lamenesses that are generally only evident when a horse is ridden. There is a hopping-type forelimb lameness, characterized by a shortened cranial phase of the stride and a marked lift of the head and neck as the lame forelimb is protracted. It can vary in degree within a work period and may disappear if the horse is ridden on a long rein. It is often but not invariably worse with the affected limb on the outside of a circle. It may be sensitive to the diagonal on which the rider sits. Such lameness is often refractory to any diagnostic analgesic technique, is non-responsive to non-steroidal anti-inflammatory drugs (NSAIDs), and comprehensive investigation with the use of diagnostic imaging usually fails to identify a cause.

Unilateral primary strain of a brachiocephalicus muscle may manifest as lameness only discernible when the horse is ridden at walk. It is characterized by lifting of the head and neck as the limb is protracted and a shortened cranial phase of the stride. Some horses with injuries of the tendon of biceps brachii have also shown lameness only when ridden, which was markedly more severe at the walk than at the trot. Low-grade shoulder-slip may be more obvious at a walk than at faster gaits and when ridden compared with in-hand.

There is a lameness typified by a shortened cranial phase of the stride of one hindlimb at the walk, when ridden “on the bit” (or driven) but not when ridden (or driven) on a loose rein, nor when lunged, even with side reins adjusted to simulate the position of the horse’s head and neck when working “on the bit.” No lameness is detectable at other gaits. This lameness is also refractory to any diagnostic analgesic technique, is non-responsive to NSAIDs, and comprehensive investigation with the use of diagnostic imaging usually fails to identify a cause. Prolonged rest does not alter the problem, nor does physiotherapy, acupuncture, chiropractic treatment, or osteopathic treatment.

There remains a small but important group of horses in which even a highly skilled and experienced lameness clinician cannot see lameness, but a skilful rider can feel lameness and/or compromise in performance. I never cease to be amazed by the large degree of apparent asymmetry of movement I can feel when riding a horse that I cannot detect by visual appraisal. However, it must also be recognized that some highly successful competition riders are apparently unable to feel even quite obvious lameness, whereas some far less talented riders do have the ability to feel even low-grade lameness.

Ridden exercise is not, however, a substitute for assessment in hand and on the lunge, because there are some lamenesses, especially forelimb lameness, which may only be apparent in hand and/or on the lunge. It is curious that even when ridden on a long rein under identical circumstances, such lameness may be concealed. It should also be borne in mind that a horse that shows lameness that is evident when trotted in hand, which can be abolished with the use of diagnostic analgesia, may also demonstrate another lameness due to an unrelated cause when ridden.

Low-grade hindlimb ataxia can mimic hindlimb lameness, and hindlimb lameness and ataxia may coexist. Concurrent existence of hindlimb lameness and low-grade ataxia can confound interpretation. Horses with mild ataxia may have a bouncy, croup-high, and stiff-legged hindlimb gait when decelerating from trot to walk, with steps of irregular height. There may be more deliberate placement of the hind feet to the ground, with sideways movement of the feet. There may be a toe-drag and lack of hindlimb propulsion. If turned in small circles, there may be a toe-drag, abnormal limb placement, delayed movement of limbs, circumduction of the outside hindlimb, and sometimes one limb may collide with another. Using a tail-pull test at the walk may highlight the presence of weakness. An affected horse may have a croup-high canter on the lunge.

There are some gait characteristics that can be highly suggestive of the primary source of pain causing lameness. For example, a short pottery forelimb gait, soreness when turning, and accentuation of the lameness on a firm surface on the lunge are features highly suggestive of foot pain. A forelimb lameness that is worse with the affected limb on the outside of a circle on a soft surface is suggestive of proximal suspensory desmitis. A forelimb lameness that is markedly accentuated by carpal flexion is likely to reflect carpal pain. However, many gait modifications are non-specific, and horses may adapt their gaits differently despite similar sources of pain. Flexion tests are also non-specific. Whereas exacerbation of lameness after fetlock flexion may reflect fetlock region pain, lameness associated with proximal suspensory desmitis may also be accentuated, presumably because of release of tension on the suspensory apparatus during flexion and then sudden loading when the limb is placed to the ground.

The importance of clinical observation cannot be over-emphasized—looking and seeing; assessment of stance at rest, conformation, balance and symmetry; systematic palpation; determination of the baseline lameness; and the response to flexion tests. At this stage in an investigation, it may be possible to determine accurately a potential source of pain causing lameness, but in many cases local analgesia...
will be required. Although careful assessment of gait may be suggestive of the primary source of pain causing lameness, in my experience, many of the findings are non-specific and the same injury may be manifest differently among horses. Moreover, more than one source of pain may coexist in one limb, therefore accurate diagnosis can only be achieved with the use of diagnostic analgesia. The gait characteristics may change after abolition of one source of pain if another source coexists. For example, a horse had a low-grade bilateral forelimb lameness evident only on the lunge on a firm surface or ridden, as mild (grade 2/8) left forelimb lameness on the left rein and subtle (grade 1/8) right forelimb lameness on the right rein. After palmar digital nerve blocks of the left forelimb, right forelimb lameness was accentuated (grade 3) on the right rein, but only when ridden. Palmar digital nerve blocks of the right forelimb abolished this lameness, but then a more severe right forelimb lameness (grade 4) became apparent on the left rein when ridden. This was ultimately abolished by palmar metacarpal (subcarpal) nerve blocks. Foot pain and proximal suspensory desmitis coexisted.

There are differences of opinion, reflecting personal experiences about whether a horse is lame enough to block, for example, is it likely that the observer would be able to detect an improvement if lameness was abolished? This can certainly be tough if a horse with subtle lameness is only assessed in-hand and on the lunge. However, it must always be borne in mind that subtle lameness may reflect bilateral lameness and if pain is abolished in one limb a much more obvious lameness may become apparent in the contralateral limb. If a horse is also assessed ridden and other aspects of performance are considered together with lameness, I believe many horses with subtle lameness can be nerve-blocked with meaningful results. This is likely to be much more rewarding than resorting to survey radiography or nuclear scintigraphic examination, which often yield false-negative or false-positive results.

Nerve blocks must be performed in a systematic way; there are few shortcuts, and this time-consuming procedure cannot be rushed without risks of misinterpretation of the results. However, on the basis of the findings of an initial clinical assessment, a logical decision can be made about where to start. For example, if there is a markedly positive response to distal limb flexion, then intra-articular analgesia of the fetlock may be performed, bearing in mind the potential to influence closely related anatomical structures, such as the suspensory ligament branches. In a hindlimb, in the absence of clinical signs related to the fetlock and more distal aspects of the limb, it would be reasonable to start by perineural analgesia of the plantar (at the junction of the proximal three-quarters and distal one-quarter of the metatarsus) and plantar metatarsal (distal to the “button” of the second and fourth metatarsal bones) nerves (a “low four-point-block”). If a horse showed lack of hindlimb propulsion but no detectable lameness, bilateral perineural analgesia of the deep branch of the lateral plantar nerve may result in substantial improvement in the horse’s performance when ridden, whereas a unilateral block may confusingly result in little change. However, the distal aspect of the limb should first be excluded as a potential source of pain by use of a “low four-point-block.”

Nerve blocks can paradoxically result in an increase in lameness severity. If the foot is desensitized and it is not the source of pain causing lameness, lameness may deteriorate. This is a non-specific finding but is often seen in association with suspensory ligament pain. I believe that the foot serves a proprioceptive function and the horse can modify its gait to reduce pain. Desensitization of the foot reduces its proprioceptive function, and the horse is less able to adapt its gait to reduce loading and therefore increased strain is placed on the suspensory apparatus, resulting in accentuation of lameness.

Thus, interpretation of the response to local analgesia is not always straightforward. How much improvement is expected after apparent desensitization of a single source of pain? This depends on both the severity of the pain, the cause(s) of pain, and how that pain is mediated and whether there may be a mechanical component to the lameness. For example, severe foot pain associated with a sub-solar abscess, a fracture of the distal phalanx or navicular bone, laminitis, or adhesions of the DDFT may be unaffected or only partially improved by palmar nerve blocks performed at the base of the proximal sesamoid bones. Pain associated with a neuroma may be minimally influenced by perineural analgesia. However, it must also be borne in mind that more than one source of pain may coexist, and more proximal nerve blocks may be required. This is where the art and science of lameness diagnosis must be combined.

3. Some Further Observations Concerning Local Analgesia

Diagnostic analgesia is frequently required to localize the site(s) of pain causing lameness, but it is crucial to be aware of the limitations of the techniques used and how confusion may arise, a problem I first addressed in 1986.9 It is well-recognized that perineural analgesia may abolish pain distal to the sites of injection, but at some sites proximal diffusion of local anesthetic solution may result in abolition of pain at sites considerably proximal to the site of injection. Pain associated with the proximal interphalangeal (PIP) joint was induced by injection of bacterial lipopolysaccharide, and baseline lameness was recorded.10 Local anesthetic solution (1.5 mL per site) was injected around the palmar digital nerves at sites 1, 2, and 3 cm proximal to the cartilages of the foot (ungular cartilages). The
median lameness score improved after injection at
the two most proximal sites. I have seen 80% im-
provement in lameness associated with severe sub-
chondral bone trauma of the proximal aspect of the
middle phalanx with the lesion communicating with
the PIP joint, or in association with advanced osteo-
arthritis (OA) of the PIP joint, within 10 minutes of
perineural analgesia of the palmar digital nerves
immediately proximal to the cartilages of the foot.
Moreover, it has been reported that perineural an-
algesia of the palmar digital nerves may resolve
lameness associated with lesions of the metacarpo-
phalangeal joint.11

After perineural injection of 2 mL of a radiodense
contrast medium (iohexol) around the palmar
nerves at the base of the proximal sesamoid bones,
there was immediate proximal spread of the con-
trast medium detected radiologically, and, within 10
minutes, the mean proximal spread was 16.9 ± 13.7
mm and further spread up to 30 minutes after in-
jection (mean, 20.8 ± 15.1 mm) (Fig. 5A).12 Spread
was not significantly different in horses that stood
still or those that were walked after injection. Di-
fusion is influenced by molecular weight, and io-
hexol has a larger molecular weight than
mepivacaine; thus, mepivacaine may diffuse fur-
ther. This explains clinical observations that
horses with incomplete fractures of the sagittal
groove of the proximal phalanx often have lameness
that is abolished by perineural analgesia of the pal-
mar nerves performed at the base of the proximal
sesamoid bones (Fig. 6).13 Lameness associated
with injuries of the palmar annular ligament or the
insertion of the suspensory ligament branches, the
fetlock joint, and the digital flexor tendon sheath
may be similarly abolished.

Distribution of contrast medium in a patch out-
side the neurovascular bundle occurred occasionally
(Fig. 5B), or proximal spread occurred in a lym-
phatic vessel.12 These observations may explain in
a clinical setting either a delayed onset of analgesia
or false-negative results of palmar nerve blocks per-
formed at the base of the proximal sesamoid bones.
Perineural injection of iohexol around the medial
and lateral palmar nerves at the junction of the
proximal three-quarters and distal one-quarter of
the metacarpus and over the palmar metacarpal
nerves immediately distal to the second or fourth
metacarpal bones, simulating a “low four-point
block,” resulted in significant proximal diffusion

Fig. 5. A, Dorsopalmar radiographic image of a left forelimb, obtained immediately after subcutaneous injection of 2 ml radiodense
contrast medium over the lateral palmar nerve at the base of the lateral proximal sesamoid bone. There is an elongated distribution
of the contrast medium and marked proximal and distal diffusion, within the fascia surrounding the neurovascular bundle. The
extents of proximal (A) and distal (B) diffusion were measured from a line drawn perpendicular to the medial/lateral aspect of the
proximal articular surface of the proximal phalanx, to the most proximal/distal aspect of the contrast ‘patch’. B, Dorsomedial-
palmarolateral radiographic image of a right forelimb, obtained immediately after subcutaneous injection of 2 ml radiodense contrast
medium over the medial and lateral palmar nerves at the base of the medial and lateral proximal sesamoid bones. The needles were
left in position. Note the elongated distribution of the contrast medium and the marked proximal diffusion from the tip of the needle
(white arrowhead) within seconds after injection at the medial injection site (right side of the image, white arrow). At the lateral
injection site (left side of the image), the distribution of the contrast medium was more diffuse (black arrowheads), compared with the
elongated pattern laterally, suggesting that it was outside the fascia surrounding the neurovascular bundle. (Reproduced from
over time but never proximal to the mid-metacarpal region.\textsuperscript{14} Distribution of the contrast medium within the neurovascular bundle was achieved in only 77.5\% of sites compared with 89\% when injecting at the base of the proximal sesamoid bones (Fig. 7).\textsuperscript{12} There was no difference in successful injection into the neurovascular bundle with injections performed in a weight-bearing or a non–weight-bearing position. Delayed desensitization or failure to achieve analgesia may occur if injection is not into the neurovascular bundle. The most proximal extent of the contrast medium, measured from the tip of the needle in the radiograph obtained immediately after performing the palmar injection, was 80.9 mm.\textsuperscript{14} The most proximal extent of a palmar patch of contrast medium after injection of the palmar metacarpal nerves was sometimes seen between the distal one-third and one half of the metacarpal region. It is therefore unlikely that after a palmar metacarpal injection immediately distal to the distal aspect of the second and fourth metacarpal bones, diffusion would result in improvement of pain in the proximal metacarpal region. The explanation for a low four-point block improving lameness associated with proximal suspensory desmitis\textsuperscript{15} may be caused by painful lesions in the suspensory ligament extending further distally than can be appreciated ultrasonographically or by the presence of a concurrent site of pain in the fetlock region.

Paradoxically, in some horses with subchondral bone pain in the palmar aspect of the condyles of the third metacarpal bone associated with extensive mineralization, lameness is improved but not abolished by a “low four-point block.”\textsuperscript{b,c} Palmar metacarpal nerve blocks performed just distal to the carpus are required to resolve the lameness, although no lesions in the carpus or proximal metacarpal region have been identified through the use of radiography, ultrasonography, and MRI. It is presumed that there are nerves that enter the third metacarpal bone proximal to the sites of injection for the “low four-point block,” which require anesthesia to abolish pain and lameness. Similar observations have been made in hindlimbs.

In two of 40 limbs in the “low four-point block” study, there was radiodense contrast medium within the digital flexor tendon sheath (DFTS) (Fig. 7), which raises concerns about potential iatrogenic synovial sepsis.\textsuperscript{14} The injection sites were not clipped before injection, and, although the sites were thoroughly cleaned, the limbs were not prepared for aseptic injection. Neither of the two horses whose DFTS was penetrated developed effusion of the DFTS or lameness after the study, but we recommend extra care (thorough cleaning and clipping if the hair does not permit adequate cleaning) when preparing for blocking the palmar nerves when performing a low four-point nerve block. To minimize the risk of iatrogenic infection of the DFTS, the palmar injection should be performed proximal to the proximal extent of the DFTS. This, depending on the degree of DFTS effusion may, however, run the risk of desensitizing more proximal structures. Since performing this study, I saw one horse with no palpable distension of the DFTS that developed sepsis of the DFTS within 15 hours of a low four-point block.
Intra-articular analgesia of the fetlock joint or intrathecal analgesia of the DFTS might be considered to be potentially more specific than perineural analgesia. However, there is the potential to influence structures in close proximity to these synovial structures resulting in false-positive results. Intra-articular analgesia of the fetlock joint may abolish pain associated with lesions of the suspensory ligament branches or the origin of the oblique or straight sesamoidean ligaments. Intrathecal analgesia of the DFTS can also alleviate pain associated with injuries of the straight or oblique sesamoidean ligaments or branches of the SDFT.

Intra-articular analgesia of the fetlock joint can also result in false-negative results in the presence of subchondral or trabecular bone pain. Clinical observations and cadaver studies have demonstrated the potential lack of specificity of a variety of techniques for analgesia of the proximal palmar aspect of the metacarpus. This has been further highlighted by an in vivo study that used the radiodense contrast medium model. After perineural injection of the medial and lateral palmar metacarpal nerves distal to the carpus from either the medial and lateral aspects, or from the lateral aspect alone, there was radiodense contrast medium in the carpal metacarpal and middle carpal joints in 50% of limbs and 12.5% of limbs, respectively. Thus, it is important to evaluate both the proximal metacarpal region and the carpus with the use of diagnostic imaging after a positive response to subcarpal analgesia of the palmar metacarpal nerves. Although this lack of specificity is most important in racehorses, which have a high incidence of carpal disease, there is increasing recognition of the importance of carpal pain in sports horses as well. With a lateral approach to the lateral palmar nerve, there is a small but significant risk of inadvertent penetration of the carpal sheath, which may result in false-negative responses to diagnostic analgesia. With a medial approach to the lateral palmar nerve, there is a risk of proximal diffusion of local anesthetic solution into the distal one-third of the antebrachium and spread around the median nerve and the caudal branch of the ulnar nerve (Fig. 8). This may result in desensitization
of the entire palmar carpal region and caudodistal aspect of the radius.

It is clear that no method of anesthesia of the proximal metacarpus is either entirely specific or reliable. It is therefore important to recognize the limitations of each technique, including the risks of synovial infection. Interpretation is further complicated by the clinical recognition that carpal and proximal metacarpal lesions may coexist.\textsuperscript{21} Moreover, although proximal diffusion from a subcarpal injection site was not seen in the contrast-medium study, palmar and palmar metacarpal nerve blocks have abolished pain associated with a parasagittal fracture of the third carpal bone and rupture of the medial palmar intercarpal ligament\textsuperscript{d} or lesions at the origin of the ALDDFT on the palmar aspect of the third carpal bone.\textsuperscript{22}

The medial approach to the lateral palmar nerve is potentially perhaps the least specific technique for analgesia of the proximal palmar aspect of the metacarpus. Some difficult horses are easier to inject with the limb on the ground; a lateral approach to the lateral palmar nerve may be preferable in such a horse. This approach also is unlikely to interfere with subsequent ultrasonographic examination, whereas with the subcarpal techniques there is a risk of creating air artifacts. Other horses are more easily restrained with the limb to be injected maximally flexed; in such horses either approach to the palmar metacarpal nerves may be the preferred technique. Proficiency in all techniques and adaptability are desirable.

It is a common clinical observation that lameness associated with proximal suspensory desmopathy (PSD) in hindlimbs may be improved by intra-articular analgesia of the tarsometatarsal (TMT) joint, particularly if assessment of the block is delayed >10 minutes or if >3 mL of local anesthetic solution is injected. There is close proximity between the distal plantar outpunchings of the joint capsule and the suspensory ligament.\textsuperscript{23} If >3 mL of mepivacaine is injected into the TMT joint, increased pressure within the joint is likely to encourage plantar leakage through the needle tract. Injection of radiodense contrast medium into the TMT joint resulted in spread to the centrodistal joint in 35% of limbs\textsuperscript{23} and frequent diffusion (90% of limbs) around the insertions of tibias cranialis and fibularis teriatus, as far proximally as the tarsocural joint. There is therefore the potential for mepivacaine to influence the dorsal metatarsal nerves. There was also diffusion into the tarsal sheath, with traces present in 35% of limbs by 15 minutes after injection of the TMT joint. In one limb, contrast medium was distributed in the tarsocural and talocalcaneal-centroquartal joints and not in the TMT joint. It is conceivable that the needle was inadvertently positioned proximal to the fourth tarsal bone.

After subtarsal injection of contrast medium around the plantar metatarsal nerves with/without the plantar nerves, proximal distribution of contrast medium within the tarsal sheath was seen in 40% of limbs; no proximal diffusion was identified outside the tarsal sheath. Contrast medium was only seen in one TMT joint, suggesting that the likelihood of subtarsal analgesia influencing the tarsus is relatively small. However, even with the use of the potentially more specific technique of perineural analgesia of the deep branch of the lateral plantar nerve (3 mL mepivacaine evaluated 10 minutes after injection), pain associated with the central tarsal (CT) bone has been substantially improved in two horses, one with abnormal mineralization of the CT bone and one with a fracture (Fig. 9).\textsuperscript{24} Highlighting the lack of specificity of diagnostic analgesia and confirming previous observations.\textsuperscript{25}

We have previously documented false-negative responses to intra-articular analgesia of the tarsocural joint associated with lesions in the subchondral bone\textsuperscript{26} and false-negative responses to intra-articular analgesia of the CD and TMT joints in associated with advanced osteoarthritis (OA).\textsuperscript{27} Perineural analgesia of the tibial and fibular nerves should abolish pain from the tarsus. However, local diffusion from the tibial injection site may result in resolution of lameness associated with gastrocnemius tendonitis.\textsuperscript{28} Proximal diffusion may result in im-

Fig. 8. Lateromedial radiographic image acquired immediately after injecting radiodense contrast medium using a medial approach to the lateral palmar nerve at the level of the accessory carpal bone. There is proximal spread of the contrast medium in a Y-shaped pattern in the distal third of the antebrachium, around the median nerve and the caudal branch of the ulnar nerve. Such spread of local anaesthetic solution could result in densitisation of the palmar aspect of the carpus.
Magnetic resonance imaging has greatly enhanced our ability to make more specific diagnoses, most particularly in the foot, but also in the pastern, fetlock, metacarpus and metatarsus, carpus and tarsus, and stifle. However, as with any imaging modality, acquisition of high-quality images and accurate interpretation are of paramount importance. Selection of the correct region to examine is crucial; the previous discussion about the lack of specificity of local analgesic techniques highlights this. If lameness is abolished by palmar (plantar) nerve blocks performed at the base of the proximal sesamoid bones, it may be necessary to examine not only the foot and pastern but also the fetlock.

The use of MRI has also highlighted our lack of understanding about what may cause pain. A horse with unilateral lameness may show similar abnormalities of the navicular bone and deep digital flexor tendon bilaterally. Why did the horse not show bilateral lameness? Is the non-lame limb at an earlier subclinical phase? How likely is it that the horse will become lame on this limb? If a combination of structures is abnormal, which is most likely to be the current major source of pain? Does the presence of increased radiopharmaceutical uptake make it more likely that a structure is a source of pain? These and many other questions remain currently unanswered. The following discussion reviews some of our advances in knowledge related to a variety of conditions of the foot, including pertinent clinical features. It is crucial to recognize that not all horses require MRI. Careful clinical appraisal and acquisition of high-quality radiographs and ultrasonographic images, correctly interpreted, can often lead to an accurate diagnosis. Since the advent of MRI, I believe that we are in a better position to understand the clinical significance of some subtle radiological abnormalities.

Collateral Ligaments of the DIP Joint

**Biomechanical Function**

The collateral ligaments (CLs) of the DIP joint originate from depressions on the distal medial and lateral aspects of the middle phalanx and insert both in small depressions on the dorsomedial and dorsolateral aspects of the distal phalanx, close to the joint margins, and to the dorsal aspect of the medial and lateral ungular cartilages. Their function is to support the DIP joint in its movements in sagittal, frontal, and transverse planes. Asymmetric foot placement, with the quarters at different heights, results in lateral or medial rotation and sliding of the distal phalanx relative to the middle phalanx (frontal movement). It also results in the middle phalanx rotating and the elevated side of the middle phalanx moving in a palmar direction (transverse movement). These movements are passive but place particular stress on the CLs of the DIP joint. Landing on the lateral aspect of the foot first, a consistent feature in Warmblood horses (although not necessarily detectable by gross observation), may result in asymmetric forces in the CLs of the DIP joint. A recent in vitro study with the use of three-dimensional bone and ligament reconstruction...
demonstrated that strain in the CLs of the DIP joint was increased on sharp turns compared with straight lines; the highest strain increase was in the medial CL. The medial CL is placed under particular strain by the outward rotation of the DIP joint created by the rapid deceleration of the lateral side of the foot as it lands first.

**CL Injury**

CL injury has been recognized as an important cause of foot pain either alone or in association with other osseous or soft tissue injuries. There are frequently no localizing clinical signs, although occasionally there is mild swelling just proximal to the coronary band and/or heat, which may only be detectable after clipping preparatory to ultrasonography. To date, no conformational abnormalities of the digit have been associated with CL injury. Horses may be uncomfortable standing on a wedge that tilts the foot mediolaterally or standing on a board that elevates the toe. Lameness may be detectable at walk but be much less apparent at trot in straight lines. There is a longer stance phase at the walk than at the trot, so there is greater extension of the DIP joint and thus more stress on the CLs of the DIP joint. In a histological study, lesions at the insertion were worse toward the palmar aspect of the CL. This concurs with both scintigraphic observations that increased radiopharmaceutical uptake (IRU) often extends palmar to the coronary band and radiological observations that osseous cyst-like lesions (OCLLs) tend to occur at the palmar aspect of the insertion site. Extension of the DIP joint results in increased tension in the palmar aspect of these ligaments. Characteristically lameness is often two grades or higher more severe at trot in circles compared with straight lines, especially on a firm surface when the foot does not sink into the ground.

**Response to Diagnostic Analgesia**

In 114 horses with primary CL desmopathy of the DIP joint and no other cause of lameness, 35.6% were sound after perineural analgesia of the palmar digital nerves; 44.5% improved ≥50%; in 30% there was no change or <50% improvement. Lameness was abolished in all horses by palmar (at the base of the proximal sesamoid bones) nerve blocks. Sixty-eight percent of horses did not respond to intra-articular analgesia of the DIP joint within 5 minutes of injection; 30% improved ≥50%, and 2% were sound. Lesions at the distal aspect of the CLs identified histologically occurred principally adjacent to the synovium of the DIP joint. This may have implications for pain associated with injury and the potential response to intra-articular analgesia. Local anesthetic solution may desensitize the superficial nerves in the synovium within seconds after intra-articular injection. It was presumed that the horses that did respond to intra-articular analgesia may have secondary synovitis or OA caused by mild joint instability, the result of CL injury. However, it is possible that pain associated with a focal lesion on the axial, synovial side of the CL insertion could be alleviated, especially if the assessment of the response to intra-articular analgesia was delayed. We have observed abnormal accumulation of synovial fluid axial to an abnormal CL using MRI, and, in association with an OCLL there may in some horses be communication between the lesion and the joint. None of 65 horses with primary CL injury of the DIP joint showed improvement in lameness after intrathecal analgesia of the navicular bursa. A uniaxial palmar block usually had no effect in horses with uniaxial lesions, perhaps because both CLs were painful but only one had structural change.

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*Fig. 10. A, Transverse ultrasonographic image of the medial collateral ligament (CL) of the distal interphalangeal (DIP) joint. The CL is enlarged, with poorly demarcated margins and heterogeneous echogenicity, consistent with desmitis. B, Transverse fast spin echo low-field magnetic resonance image of the same limb as Fig. 10A. Medial is to the right. The medial CL of the DIP is enlarged close to its insertion on the distal phalanx and has increased signal intensity. The lesion extended the entire length of the ligament and also had increased signal intensity in fat suppressed images.*
Lesions may be identified ultrasonographically, but only a limited length of the ligament can be assessed. Ultrasonographic assessment revealed lesions consistent with desmitis characterized by enlargement of the ligament, and areas of reduced echogenicity were identified in 85 of 313 horses (27%), with lesions confirmed with the use of MRI. On MRI, CL injury was defined as increased signal intensity in T1 and T2*-weighted (W) gradient echo (GRE) or fast spin echo (FSE) images in part or all of the ligament, with enlargement or change in shape, or altered definition of the margins, with or without alteration in signal intensity in the periligamentar tissues. Some horses also had increased signal intensity in fat-suppressed images. In all studies, medial lesions were identified more frequently than lateral injuries; lesions were sometimes bilateral. Interpretation of MR images is potentially confounded by the magic angle effect (MAE) in images acquired in both low-field and high-field magnets related to the orientation of the fibers, especially at the origin; the lateral CL is especially susceptible in images acquired standing because of its more sloping orientation. Use of sequences with long echo times is useful but does not abolish the MAE. Concurrent osseous pathology is discussed below.

**Diagnostic Imaging**

**Verification of MRI by Comparison With Histology**

To verify the interpretation of MR images, a comparative high-field MRI and histological study was performed in horses both with and without suspected CL injury. It was hypothesized that abnormal signal intensity and tissue contour would represent change in tissue structure detected through histological examination. The aims were to compare results in horses free from lameness and those with chronic lameness and to describe possible progression of lesions. One or both feet from 12 horses free from lameness (Group N) and 25 horses with foot-related lameness (Group L) were examined with the use of MRI and by gross postmortem examination. The MR images were graded (0–3) by one analyst. Sagittal histological sections from the proximal and distal aspect of each CL were examined histologically and were assigned a histological grade: 0 = normal or the presence of transitional fibrocartilaginous metaplasia, considered to be within normal limits, near the origin and/or insertion; 1 = localized pallor; 2 = diffuse, extensive, or multifocal fibrocartilaginous metaplasia; 3 = extensive fissuring degeneration and any associated osseous pathology (Fig. 11A,B). The overall score assigned to each CL was the sum of the proximal and distal grades. Scintigraphic images from lame horses were also evaluated.

In horses from Group N, 25 CLs were graded normal on both MR images and histology. The majority of CLs had a histology score of 0. Two CLs were graded 1 on MR images but were histologically normal. Two CLs had MR abnormalities verified histologically. However, two CLs appeared normal on MR images but were histologically abnormal.

In Group L, in 72 of 89 CLs the results of MRI and histopathology concurred. Eighteen CLs were...
deemed normal on both MR images and histology. Fifty-four CLs had MR abnormalities verified histologically. Of the 13 CLs from 10 horses that were graded as normal with the use of MRI but were abnormal histologically, the overall histology score ranged from 1 to 5 of 6 (mean, 3.33). Histological abnormalities of the CLs of the DIP joint were identified in 37 medial CLs and 30 lateral CLs. When the score was compared between medial and lateral, there was a significantly more severe histological score in the medial than lateral ligaments ($P = 0.007$). The grade of lesion was greater distally than proximally in 44 CLs, of equal grade in 16 CLs, and was less distally than proximally in seven CLs, with similar proximodistal distribution of lesions medially and laterally.

The distal insertion of the CLs was the region in which the most severe degenerative abnormalities were seen histologically, especially axially (ie, adjacent to the synovium of the DIP joint) and toward the palmar aspect. Early changes were characterized by linear areas of pallor and hyalinization of the collagen fibers, followed by transformation of fibroblasts into chondrocytes and the development of diffuse areas or focally dense areas of fibrocartilaginous metaplasia (Figs. 11A,B). More severe lesions showed a very typical pattern characterized by tortuous, intercommunicating fissures, with smooth edges within the degenerate, hyalinized collagen, containing chondrocytes and chondrones. The pattern was very distinct from ordinary splitting artifacts as the result of histological processing. Several horses showed evidence of lesion-associated blood vessel occlusion and attempted revascularization. Naturally occurring clefts were seen at the bone-ligament interface in association with severe ligament degeneration. Osseous changes were seen immediately distal and palmar to the insertion of the CL on the distal phalanx, with early changes characterized by bone loss on the abaxial cortical surface of the distal phalanx and replacement by fibrous tissue. Other horses had mild interstitial edema in the marrow fat. Lesions progressed to the formation of bone spaces and ultimately an OCLL.

In the majority of horses, histological lesions in both the medial and lateral CLs were similar (23.9%) or worse (65.7%), distally compared with proximally. We have previously suggested that the insertion site is a major stress point, irrespective of the site of injury within the ligament. Concurrent Osseous Pathology

The relationship between CL injury and osseous abnormalities was investigated in 39 horses (Fig. 12). Focal or diffuse areas of IRU in the distal phalanx at the insertion of the CL had previously been identified scintigraphically,33,34,43 and it had been suggested that this site is susceptible to significant biomechanical loading, irrespective of the site of injury within the ligament.43 It was hypothesized that there would be (1) a higher incidence of osseous abnormality at the insertion of an injured CL than at the origin; and (2) a relationship between the presence of osseous abnormality and duration of lameness.

Magnetic resonance images of 313 feet of 289 horses with foot pain and a definitive diagnosis of collateral desmopathy of the DIP joint were retrospectively analyzed for presence and type of osseous abnormality in the middle and distal phalanges. Scintigraphic images were examined and the presence of IRU in the middle or distal phalanges was recorded. Osseous abnormalities were detected in 45.7% of feet, 18.8% of which had osseous and CL injury alone; the remainder had CL-related osseous injury and multiple injuries within the hoof capsule.
Endosteal reaction and entheseous new bone were the most common types of osseous pathology associated with the ligament origin, accounting for 8.3% (n = 26) and 7.3% (n = 23) of feet, respectively. The most frequent types of insertional osseous pathology were entheseous new bone (36.4%, n = 114) and focal increased signal intensity on short tau inversion recovery (STIR) images (11.5%, n = 36). Osseous cyst-like lesions occurred in only 13 feet (4.1%), of which only two were associated with the ligament origin. Osseous cyst-like lesions in the distal phalanx were all situated distal and palmar to the site of CL insertion. Diffuse increased signal intensity on STIR images was the most frequent type of osseous injury located within the distal phalanx, occurring in 7.3% (n = 23) of feet. Twenty-eight feet (8.9%) had diffuse increased signal intensity on STIR images and concurrent reduced signal intensity on T1-weighted images in either the middle or distal phalanges.

Some degree of mineralization of a palmar process ipsilateral to CL injury, characterized by reduced signal intensity in both T1W and T2*W GRE images, occurred in 17.3% of feet (n = 54). Osseous abnormalities of the DIP joint were identified in 12 feet (3.8%), characterized by increased signal intensity in fat-suppressed images in the subchondral bone and/or in the trabecular bone distal to the subchondral bone plate, with/without localized increased thickness of the subchondral bone. These lesions probably reflect instability of the joint or acute overload of the subchondral bone. Subluxation of the DIP joint was evident in only four feet, all with severe CL injury. A fragment associated with the ligament origin or insertion was the least common type of osseous pathology seen, occurring in only two feet.

Moderate or intense focal IRU was seen either at the site of insertion of the CL (n = 80, 15%) or at the insertion and extending into the ipsilateral palmar process (n = 20, 4%). Increased RU associated with osseous abnormality related to CL injury of the DIP joint was more frequently observed in the distal phalanx (n = 123, 97%) than the middle phalanx (n = 4, 3%), coexistent with the higher incidence of insertional osseous pathology. Focal intense IRU in one or more palmar process was observed in 14% of feet. There was a higher incidence of osseous abnormalities medially than laterally and at the ligament insertion than at the origin. There was a significant association between presence of IRU and osseous injury (P = 0.001), but normal RU did not preclude significant osseous pathology associated with CL injury.

Increased RU was often situated just palmar to the site of ligament insertion most obvious on solar scintigraphic images, in accordance with other studies. We previously reported a positive association between the presence of IRU at the site of insertion of a CL on the distal phalanx with severity of injury relative to histological grade of CL pathology. A small proportion (4%) of feet in the current study had IRU at the site of the ligament insertion extending into the ipsilateral palmar process, which was often associated with abnormal mineralization identified by MRI, characterized by decreased signal intensity in T1W and T2*W GRE images, with or without increased signal intensity in fat-suppressed images. Thus, abnormalities at the CL insertion may be associated with injury to adjacent structures. Focal IRU in a palmar process, seen in 14% of feet, has been previously documented in association with a variety of other lesions detected with the use of MRI, causing lameness or as an incidental finding in the non-lame limb of a bilaterally lame horse. In the previous study, mild to intense IRU was seen in the medial palmar process of 5.6% of 512 feet and 2.4% of lateral palmar processes. The higher frequency of IRU in a palmar process of the distal phalanx in the current study association with CL injury suggests that CL injury may increase the risk of trauma to the ipsilateral palmar process. Nuclear scintigraphic examination may therefore be useful for the detection of osseous pathology associated with CL injury, although a negative result does not preclude the presence of osseous injury.

There was a significant negative association between the presence of all CL-related osseous injury and duration of lameness (P = 0.036). It is well recognized that radiological abnormalities may predate the onset of pain and lameness associated with OA, and it is likely that the same is true for lesions detected with MRI. It is possible, however, that the presence of an osseous abnormality such as entheseous/endosteal reaction associated with the
ligament origin or insertion may afford some stabilization of the injured ligament, which may account for the negative association between the duration of lameness and presence of an osseous abnormality.

Relationship Between Ossification of the Ungular Cartilages and Injuries of the CLs of the DIP Joint

To determine the relationship between ossification of the ungular cartilages and CL injury, dorsopalmar (dorsoplantar) radiographs of one foot from each of 462 horses were examined and ossification of the cartilages of the foot graded with the use of a modification of a previously published scale. The presence or absence of CL injury was recorded. There was left-right symmetry of ossification between feet and significant association between grades of each foot, with lateral greater than or equal to medial cartilages. Possibly significant ossification (PSO) (grade ≥ 3 + separate center of ossification [SCO]) occurred in the maximally ossified cartilage in 59 (12.8%) feet. There was a significantly higher frequency of PSO in cobs and cross-breeds compared with all other breeds (P = 0.0002). There was a significantly higher frequency of PSO in cobs and cross-breeds compared with all other breeds without CL injury (P = 0.0179). There was a significant association between PSO of the maximally ossified cartilage and injury of the distal phalanx (P = 0.0391). There was no association between distal phalanx injury and marked asymmetry of the ossified cartilages of the foot.

The left-right symmetry and association between lateral and medial cartilages were similar to those reported previously. The higher proportion of PSO in cobs compared with other breeds was in accordance with a previous study and may be genetically determined. This could also be associated with the greater body weight to height ratios typical of this type of horse, or the greater component of vertical movement in the stride of these horses compared with horses with a more extended gait, creating a greater amount of vertical force per unit area, with less damping through soft tissues and the distal limb joints.

It has been hypothesized that the force of impact with the ground compresses the digital cushion, forcing the cartilages of the foot abaxially at their connection with the distal phalanx. It was also hypothesized that the force is transferred, and hence dissipated, through the venovenous anastomoses running through the cartilages. Venovenous anastomoses are more common at the base of the cartilage and most ossification starts here. The base of the cartilages of the foot had greater RU uptake than more proximally, indicating that it is a site of continued modeling throughout life, reflecting the focal concentration of force. Extensive ossification reduces the flexibility and capacity for energy dissipation of the cartilages, thus transferring a greater proportion of the force through other structures of the foot, possibly causing adaptive changes or injury in adjacent tissues. The predilection for fractures of ossified cartilages at their base may be explained by maximum stress or strain concentration at this point.

There is a close anatomical relationship between the ungular cartilages and the CLs of the DIP joint, which is likely to be important in the association between PSO and CL injury. The axial surface of an extensively ossified or fractured cartilage may abrade the abaxial surface of the CLs during flexion and extension of the DIP joint. The cartilages of the foot are connected to surrounding structures, such as the digital cushion, proximal, middle, and distal phalanges and navicular bone by small ligaments. Increased tension in these ligaments has been suggested to be partially responsible for the ossification process. The chondrocoronal ligament runs from the axial aspect of each cartilage to the middle phalanx, and the chondroungular ligament connects the distal aspect of the cartilage with the ipsilateral palmar process of the distal phalanx. These ligamentous connections may be responsible for energy transmission, which results in injury of the distal phalanx in association with extensive ossification. The chondroungular ligament in particular may be implicated because if ossification extends in a palmar (plantar) direction, the ligament may become completely enclosed within ossified tissue. In some horses, the dorsal aspect of the cartilage fuses with the ipsilateral CL, allowing direct transmission of force from the cartilage to the CL, potentially resulting in injury. It has previously been suggested that forces mediated by ligamentous attachments to the cartilages may be transmitted differently through a rigid osseous structure compared with an unossified cartilage, possibly resulting in increased stress, modeling, and risk for bone trauma or fracture at the base of the extensively ossified cartilage.

Response to Treatment of Horses With Injury of a CL of the DIP Joint

Magnetic resonance images from 313 feet of 289 horses with foot pain and a definitive diagnosis of collateral desmopathy of the DIP joint were retrospectively analyzed for presence of osseous abnormality associated with the ligament origin or insertion and the middle and distal phalanges. Horses were assigned to groups according to the combination of their injuries. Type of treatment (box rest and controlled walking exercise; intra-articular medication of the DIP joint; corrective trimming to restore appropriate foot balance and shoeing according to foot conformation and to facilitate breakover; extracorporeal shockwave therapy [ECSWT] or radial pressure wave therapy [RPWT]) was recorded and follow-up information was ob-
tained. Thirty-two horses with additional sources of lameness were excluded from analysis of outcome. Follow-up data were available for 182 horses, 55 of which had follow-up information for up to 2 years after presentation. Forty-four percent of horses ($n = 41$) with CL injury alone and 43.2% of horses with CL related osseous injury ($n = 55$) returned to their previous athletic function. Prognosis for a combination of injuries to multiple soft tissue and osseous structures within the hoof capsule was substantially worse, with only 14.6% of 71 horses returning to their original level of work. There was no effect of ECSWT or RPWT on outcome, but this may reflect the low power of the study. The presence of mild to moderate CL-related osseous injury did not appear to influence prognosis compared with CL injury alone; however, horses with OCLL in the distal phalanx at the insertion of CL had a poor outcome. Paradoxically, there was association between an excellent outcome at 12 months and the presence of enthesial new bone at the ligament insertion (odds ratio = 6.8, 95% confidence interval = 1.7–27.1, $P = 0.05$), suggestive that horses with this type of osseous abnormality were more likely to become sound.

If the results of horses with increased signal intensity in fat-suppressed images in the injured CL were considered ($n = 41$), 51% returned to full athletic function for a minimum of 6 months after return to full function. These results are comparable with a previous study in which 12 of 20 horses (60%) with collateral desmopathy returned to previous level of athletic function with a minimum follow-up of 9 months after injury. In the current study, four horses that were unresponsive to conservative management underwent bilateral palmar digital neurectomy and returned to their previous function, remaining sound for a minimum of 12 months and up to 4 years. On the basis of these small numbers, it appears that palmar digital neurectomy is a safe procedure to perform with primary CL injury. Our previous correlative MRI, scintigraphic, and histopathological study demonstrated that IRU at the insertion of an injured CL of the DIP joint was associated with a more severe histological score of the injured CL; however, there was no relationship between the presence of IRU and osseous abnormalities detected with the use of MRI. In the current study, there was no association between the presence of IRU at the insertion of a CL of the DIP joint and outcome at 6 or 12 months. The lack of significant association may reflect the low power of the study or that the presence of IRU does not relate to prognosis.

Follow-up data are now required for horses treated by the intrasional injection of mesenchymal progenitor (stem) cells and intra-articular medication with interleukin 1 receptor antagonist protein. Other Injuries Associated With Ossification of the Ungular Cartilages (Cartilages of the Foot)

Functional Anatomy

The ungular cartilages provide support to the palmar (plantar) aspect of the foot, dissipate forces of the foot’s impact with the ground, and are involved in venous return from the digit. Ossification of the ungular cartilages (sidebone) normally starts at the base of the cartilage and extends proximally or can originate from an SCO. Lateral ossification is frequently more extensive than medial, although heritability of ossification in Finnhorses is similar for both lateral and medial cartilages. Marked mediolateral asymmetry of ossification is unusual but has been linked with injuries, including fractures of the ossified cartilage, trauma to the base of the ossified cartilage, and injury of the distal phalanx. Ossification reduces the energy dissipating capacity of the cartilages of the foot.

Injuries of the Ungular Cartilages and Related Structures

Ossification of the ungular cartilages has long been recognized and historically was considered of clinical importance especially in draft breeds, but, in more recent years, the clinical significance of injuries of the ungular cartilages has largely gone unrecognized. However, there is a growing body of evidence to suggest that the ossified cartilages can sustain primary injury or be associated with other injuries in the foot, notably the distal phalanx and the CLs of the DIP joint.

Primary injuries of unossified cartilages with/without related ligaments (chondrocoronal and chondrocoraloid) of the foot are unusual but have been seen in a small number of horses and have probably been previously overlooked. Diagnosis was dependent on MRI. In three horses with low collapsed heels and a large body size relative to foot size and in one horse with markedly asymmetrical heel bulbs, the affected cartilage(s) were markedly thickened, had irregular contours (especially abaxially), were hypervascular, and had diffuse increased signal intensity in fat-suppressed images, which extended into the ipsilateral aspect of the distal phalanx.

One horse with grade 0 ossification of the medial ungular cartilage had reduced signal intensity at the base of the cartilage and throughout the medial palmar process of the distal phalanx in T1W and T2* GRE images and increased signal intensity in fat-suppressed images, consistent with mineralization and bone trauma. There was marked enlargement of the medial chondrocoronal ligament with increased signal intensity in fat-suppressed images.
Mineralization of Ossified Ungular Cartilages, Trauma at the Junction Between Separate Centers of Ossification, and Trauma or Fracture at the Base of an Ossified Ungular Cartilage

Injury of the junction between SCsO, fractures of an ossified cartilage, usually at the base, and more generalized trauma of an ossified cartilage have been described as primary causes of lameness seen in association with an ossification grade of $\geq 3$. These are relatively uncommon causes of lameness; trauma or fracture of an ossified cartilage was identified as primary injury in 24 of approximately 4500 horses (0.53%) undergoing lameness investigation over 9 years (2001–2009). The diagnosis was based on radiological and scintigraphic findings in 12 horses; the other 12 horses also underwent MRI. Fifteen of 32 horses (46.9%) with collateral desmopathy of the DIP joint in association with grade $\geq 3$ ossification of the cartilages of the foot also had evidence of trauma of one or both ossified cartilages, characterized by focal or diffuse regions of increased signal intensity in fat-suppressed images. Seven of 22 horses (31.8%) with other causes of foot pain determined using MRI in association with grade $\geq 3$ ossification of one or both cartilages of the foot also had evidence of trauma of an ossified cartilage of the foot. Horses with at least one ossified cartilage grade $\geq 4$ predominated.

A clear radiolucent line at the base of an ossified cartilage surrounded by osseous modeling was indicative of a fracture (Fig. 13A). Diagnosis was substantiated by nuclear scintigraphy demonstrating focal intense IRU correlating anatomically with the fracture site. It was not always possible to differentiate between a fracture or trauma to the junction between SCsO. Moreover, not all fractures were detected radiologically and MRI was required, although at the base of the ossified cartilage clear differentiation between trauma to the junction between SCsO and a fracture was again not always possible. Modeling at the base of an ossified cartilage combined with focal IRU, supported by increased signal intensity in fat-suppressed images, was defined as trauma to the base of an ossified cartilage. Osseous modeling around the junction between SCsO in the mid shaft of an ossified cartilage detected radiologically, associated with IRU reflected probable instability or trauma. Trauma was supported by MRI findings, including increased signal intensity in fat-suppressed images at the opposing ends of the SCsO. Other radiological findings included mediolateral thickening of an ossified cartilage, irregular cortical contours, heterogeneous radiopacity, or focal or diffuse areas of increased opacity especially distally, and ill-defined transverse radiolucent lines, especially at the base of an ossified cartilage. Careful evaluation of all radiographic projections was crucial because some fractures were only evident in one projection. Other MRI findings included focal or diffuse areas of increased signal intensity in the injured ossified cartilage in fat-suppressed images.

In horses examined with the use of MRI, there were focal or diffuse areas of decreased signal intensity in both T1W and T2W GRE images in the injured ossified cartilage, consistent with mineralization in 50% of limbs with either primary injury of an ossified cartilage (6/12) or collateral desmopathy of the DIP joint (16/32) in association with grade $\geq 3$ ossification of the cartilages of the foot. Whether such mineralization reflects a response to chronic trauma or is a physiological response is not known. It may further stiffen an ossified cartilage and alter force transmission through the ossified cartilage and

![Fig. 13. A, Dorsopalmar radiographic image of a foot. Medial is to the left. There is extensive ossification of the lateral ungular cartilage. There is a transverse incomplete fracture at the base of the ossified cartilage with periosteal callus axially (arrow) and generalized increased opacity across the ossified cartilage. Note the asymmetrical shape of the pastern and hoof capsule; there is more pastern and foot lateral to a line bisecting the proximal and middle phalanges, compared with medially. B, Transverse fast spin echo low-field magnetic resonance image of the same foot as Fig. 13A. Lateral is to the right. The lateral chondrocoronal ligament is enlarged and has increased signal intensity (arrow). Note also that the lateral ungular cartilage is thicker than medially.](image-url)
possibly increase the risk of injuries of the cartilages themselves or closely related osseous and soft tissue structures. The predilection for fractures of ossified cartilages at their base may be explained by maximum stress/strain concentration at this point. The junction between SCsO is a potential weak link.

**Trauma of the Distal Phalanx Associated With Ossification of the Ungular Cartilages**

Alterations in signal intensity in the ipsilateral aspect of the distal phalanx were identified in seven of 12 horses (58.3%) with a primary injury of an ossified ungular cartilage.\(^{66}\) This was characterized either by diffuse areas of hypointense signal in T1W and T2\(^*\)W GRE images consistent with mineralization or areas of hyperintense signal in fat-suppressed images, consistent with bone trauma. One horse had an incomplete fracture of the axial aspect of the distal phalanx. Thirteen of 32 horses (40.6%) with collateral desmopathy of the DIP joint in association with grade ≥3 ossification of one or both ungular cartilages also had evidence of abnormal mineralization or bone trauma of the distal phalanx. In both groups, the presence of active bone modeling was generally supported by IRU in the corresponding anatomical location. These osseous changes may be related to altered force distribution associated with ossification of the ungular cartilages.

**Injuries of Associated Ligaments**

Injuries of the chondrocoronal and/or chondrosesamoidean ligaments were characterized by increased size (Fig. 13B), loss of demarcation of margins and increased signal intensity in fat-suppressed images, or evidence of entheseous reaction at their origin on the ungular cartilages seen as increased signal intensity in fat-suppressed images or irregularities of the axial cortex.\(^{66}\) Endosteal and periosteal reactions were also seen at the insertion of the chondrocoronal ligaments on the middle phalanx. Such injuries may be seen with or without ossification of the ungular cartilages.

It is suggested that on the basis of our recent advances in knowledge, the identification of extensive ossification of the ungular cartilages either uniaxially or biaxially at a prepurchase examination should be documented and the potential clinical significance discussed with the purchaser. Although extensive ossification may be a normal variant in some breeds, it is not usual in most sports horse breeds and may be a risk factor for future lameness.

**Injuries of the DDFT**

**Functional Anatomy**

Within the digit, the DDFT induces axial compression of the articular surfaces of the PIP and DIP joints.\(^{70,71}\) It has an important role in stabilizing the DIP joint. The anatomical arrangement of the collateral sesamoidean ligaments (CSLs) facilitates compression of the articular surfaces of the navicular bone into those of the middle and distal phalanges.\(^{70}\) The DDFT has a dorsal fibrocartilaginous pad that supports pressure of the *tuberositas flexoria*, the transverse prominence on the proximopalmar aspect of the middle phalanx.\(^{71}\)

The relationship of the DDFT to the navicular bone varies with the phase of the stride. During the full weight-bearing stance phase of the stride, the DDFT is only in contact with the distal aspect of the bone, whereas in the propulsion phase the DDFT bends over the middle scutum (the fibrocartilaginous insertion of the straight sesamoidean ligament on the middle phalanx) and comes into full contact with the navicular bone. Tension in the DDFT is maximal, and active muscle contraction and the elasticity in the tendon and its accessory ligament result in extension of the DIP joint.\(^{71}\) At the beginning of the swing phase of the stride, the tension in the DDFT contributes passively to induce flexion of the interphalangeal joints. During extension of the DIP joint, which is maximum at the propulsion phase of the stride, pull on the DDFT creates a shear force between the DDFT and the distal sesamoidean impar ligament (DSIL).\(^{70}\)

**Injuries of the DDFT**

Several lesion types have been described, including core lesions, focal or diffuse dorsal border lesions and sagittal plane splits (Fig. 14).\(^{72-74}\) A comparative study of MR images of horses with and without DDFT injuries demonstrated that there was strong left forelimb and right forelimb symmetry in the cross-sectional area (CSA) of the DDFT, and within limbs there was strong medial lateral symmetry.\(^{75}\) There was a positive correlation between bodyweight and CSA. Cross-sectional area increased in association with core lesions of the DDFT but not in association with other types of lesions.

A second study compared horses with foot pain \((n = 34)\) and age-matched control horses \((n = 25)\).\(^{76}\) Lesions of the DDFT were graded in severity as absent, mild, moderate, or severe. In the majority of sound horses, lesions of the DDFT were absent (85%) or graded mild (15%), whereas in the lame group moderate (41%) and severe (35%) lesions predominated.

Lesions of the DDFT are common, occurring in 82.6% of limbs of 264 horses with foot-related lameness examined using MRI; however, some of these were isolated sagittal plane splits or minor dorsal irregularities of questionable clinical significance.\(^{77}\) Lesions occurred most commonly at the level of the CSL (59.4%) and the navicular bone (59.0%). At the level of the proximal phalanx core, lesions predominated (90.3%), whereas at the level of the CSL and navicular bone sagittal plane splits, dorsal abrasions and focal core lesions were most common.

Primary lesions of the DDFT are defined as lesions that are the principle cause of lameness and usually comprise core lesions proximal or distal to...
the navicular bone. Primary lesions of the DDFT often involve principally one lobe and extend a variable distance proximodistally, anywhere from the proximal phalanx to the tendon’s insertion on the distal phalanx. Occasionally lesions extended proximal to the metacarpophalangeal joint, but not all limbs were examined this far proximal. Acute inflammatory or necrotic lesions were seen on T1W and T2*W GRE images and fat-suppressed images, whereas more chronic lesions with fibroplasia may only be seen in T1W and T2*W GRE and FSE images. Lesions identified only in T1W images may be chronic or degenerative. Lesions confined to the insertion were often best identified in fat-suppressed images. Core lesions were often associated with swelling of the affected lobe, resulting in loss of the normal mediolateral symmetry. There was often associated distension of the DFTS and/or the navicular bursa, with or without soft tissue proliferation within the bursa. Severe lesions involving the dorsal aspect of the tendon may be associated with adhesion formation between the DDFT and either the CSL and/or the DSIL. Definitive diagnosis of adhesion formation can be challenging without fluid distension of the navicular bursa. Core lesions of the DDFT have also occurred in conjunction with other soft tissue injuries contributing to lameness. These other lesions were often on the ipsilateral side of the foot, suggesting that similar biomechanical forces contributed to injury.

Histopathology of core lesions has revealed no evidence of inflammatory reaction but extensive core necrosis in horses with lameness of less than 6 months’ duration whereas in horses with more chronic lameness, the core lesions were predominantly fibroplasia and/or fibrocartilaginous metaplasia. Core necrosis was characterized by vacuoles in the fascicles, which tended to coalesce with breakdown material in the vacuoles, and some “floating” chondrocytes and fibroblasts within the vacuoles. There was some evidence of revascularization toward necrotic core lesions, especially those extending into the pastern.

Other DDFT lesion types identified with the use of MRI include dorsal border irregularity and sagittal plane splits. Such lesions occurred with greatest frequency at the level of the CSL and the navicular bone. In some horses, different lesion types are seen at different levels of the tendon. Horses with

Fig. 14. A, Transverse T2* weighted (W) gradient echo (GRE) high-field magnetic resonance image at the level of the middle phalanx. Dorsal is to the top and lateral to the right. The lateral lobe of the deep digital flexor tendon (DDFT) has an irregular dorsal border with contiguous tissue of intermediate signal intensity protruding into the navicular bursa (arrow), consistent with granulation tissue. The navicular bursa is somewhat distended. B, Transverse T2* W GRE high-field magnetic resonance image at the level of the middle phalanx. Dorsal is to the top and lateral to the right. The medial lobe of the DDFT is enlarged and there is an oblique core lesion of intermediate signal intensity (arrow). This lesion, which extended proximally to the distal aspect of the proximal phalanx, also had increased signal intensity in fat-suppressed images. The navicular bursa is distended. There is tissue of intermediate signal intensity on the dorsal aspect of the DDFT axially traversing the bursa. C, Sagittal short tau inversion recovery high-field magnetic resonance image. Dorsal is to the left. There is diffuse increased signal intensity in the distal aspect of the deep digital flexor tendon, which is enlarged.
lesions of both the navicular bone and the DDFT often had multifocal lesions involving the medial and lateral lobes of the DDFT, especially from the level of the proximal aspect of the navicular bursa distally. Lesions of the navicular bone were often in the same sagittal plane as the DDFT lesions. Defects in the palmar compact bone of the navicular bone often had focal adhesions to the DDFT. Small focal adhesions may be more difficult to identify in low-field images because of the thicker slice thickness compared with high-field images. These DDFT lesions appear to be degenerative with vascular compromise, especially in the septae, and matrix changes, characterized by increased proteoglycan deposition and changes in tenocyte morphology, and fibrocartilaginous metaplasia. There was no evidence of any acute inflammatory response.

Clinical Features of Primary Injuries of the DDFT

A previous study demonstrated that horses that jump had a higher frequency of occurrence of primary DDFT injuries than horses from other disciplines. A more recent study indicated that elite show jumpers were particularly at risk. Horses 10 to 15 years of age had an increased risk of primary DDFT lesions than horses < six years old (odds ratio, 3.30). A theoretical model indicated that a 1° decrease in the angle of the solar surface of the distal phalanx would result in a 4% increase in force of the DDFT on the navicular bone at the end of stance. However, in a recent study of 300 horses with foot pain, there was no significant association between injury type and angles of the distal phalanx, although there was a trend for the angle of the dorsal aspect of the distal phalanx with the horizontal to be smaller in horses with injuries of the podo-trochlear apparatus or the DDFT compared with other groups. In a small radiological study comparing 20 horses with DDFT lesions and 20 control horses, the angle at which the DDFT passed over the palmar aspect of the navicular bone was more acute than in the control horses.

Primary lesions of the DDFT as the principal cause of lameness were seen in 80 horses between January 2001 and March 2005. These horses had closed core lesions or dorsal or less commonly palmar abrasions or long full-thickness splits; horses with short isolated parasagittal plane splits were not included. Horses with increased signal intensity in the palmar third of the navicular bone in fat-suppressed images were included, but horses with other forms of navicular pathology or other lesions potentially contributing to pain and lameness were excluded. Horses presented with either unilateral (n = 48) or bilateral (n = 30) forelimb lameness or unilateral hindlimb lameness (n = 2). Lameness often improved with rest but was exacerbated by work. Lameness varied considerably in degree (3 to 7 of 8; the most frequent lameness score was 4) but was usually worse on a circle on a hard surface. There were usually no localizing clinical signs, although some horses pointed at rest. Palmar digital analgesia rendered 25 horses (31%) sound; improved lameness >50% in a further 33 (41%), but produced no change in 22 (27%). Lameness was abolished in all horses by palmar nerve blocks at the base of the proximal sesamoid bones. Intra-articular analgesia of the DIP joint (5 mL mepivacaine; lameness assessed at 5 minutes after injection) improved or abolished lameness in 41 of 75 horses (55%) but produced little change in 34 (45%). Intrathecal analgesia of the navicular bursa resulted in improvement in lameness in 25 of 37 horses (68%). Intrathecal analgesia of the DFTS improved lameness in three of 12 horses (25%). Nineteen of 76 horses (25%) had IRU in the region of the DDFT in lateral pool phase scintigraphic im-

Fig. 15. A, Transverse section of a deep digital flexor tendon showing early core necrosis. Some fascicles undergoing degeneration contain spindle-shaped and rounded cells, resembling fibroblasts and tenochondrocytes, which are believed to have migrated into the fascicle from the surrounding interstitium. At the bottom of the image centrally there is a focal area of collagen dissolution. Haematoxylin and eosin. Magnification × 100. B, Transverse section of a deep digital flexor tendon showing core necrosis. There is a central area with loss of normal collagen structure, basophilia and increased cellularity, comprising numerous fibroblasts and differentiating chondrocytes. The striated appearance of the surrounding tendon tissue is a processing artefact, the result of separation of collagen fibres. Haematoxylin and eosin. Magnification × 100. (Reproduced from Equine Vet J 2009;41:25–33, with permission).
ages. Twenty-four horses (32%) had IRU in bone phase images in the region of insertion of the DDFT on the distal phalanx. Horses with lameness of <3 months’ duration were more likely to have IRU than horses with a longer duration of lameness. Only 10 of 75 (13.3%) horses had detectable ultrasonographic abnormalities of the DDFT, although many lesions did extend into the pastern when evaluated with the use of MRI.

Treatment and Outcome of DDFT Lesions

Treatment varied according to the site and severity of the lesion(s). Focal lesions restricted to the navicular bursa were surgically debrided. Most other lesions have been treated conservatively by rest and controlled exercise, combined with trimming and shoeing according to the individual’s foot conformation, with or without shock wave therapy.

Follow-up information for 76 horses examined before September 2004 was available. Three horses were humanely destroyed for unrelated causes, and two horses were lost to follow-up. Twenty-three of 71 horses (32%) returned to full athletic function for a minimum of 6 months and a maximum of 3 years; eight horses (11%) were sound and in light work. Forty horses (56%) had persistent or recurrent lameness.

In a study of 92 horses that underwent endoscopic evaluation of the navicular bursa, 37 of 89 horses (42%) returned to full function for an unspecified period, after an unspecified period of rest. The results for treatment with biological preparations require critical assessment.

A New Form of Navicular Disease

Since the introduction of MRI, our understanding of navicular disease has greatly increased. It is now known that navicular bone pathology in isolation is comparatively rare and frequently occurs in association with injuries of the DDFT, the CSL, and/or the DSIL. It is clear that there are a variety of forms of navicular bone pathology, such as thickening of the palmar compact bone, or degenerative lesions of the palmar compact bone, including deep erosions involving both the fibrocartilage and subchondral bone. There are lesions restricted to the spongiosa characterized by diffuse increased signal intensity in fat-suppressed images in the spongiosa; linear increased signal intensity between the attachments of the CSL and DSIL; or OCLls in the distal half of the bone. In addition, there are abnormalities restricted to the distal border including marked increase in number and size of synovial invaginations, enchondal new bone, and distal border fragments. Potentially reversible lesions consistent with acute trauma of the navicular bone may also occur.

A recent correlative MRI and postmortem study demonstrated that diffuse increased signal intensity in the spongiosa of the navicular bone in fat-suppressed images was associated with alterations of the marrow fat which was either focal or diffuse, with loss of clear cytoplasmic boundaries and increased vascularization of the interstitium, with frequent small capillaries (Fig. 16). There was fibroplasia and enlarged intertrabecular bone spaces. There was accompanying bone loss with thinning of trabeculae and irregularly spiculated borders. Osteoclasts were seen occasionally. In some bones there was multifocal accumulation of pale to acidophilic material in the interstitium of the marrow fat.

It has previously been suggested that degenerative change of the spongiosa is generally only seen dorsal to extensive fibrocartilage damage. However, in the Dyson et al study, abnormalities of the spongiosa could be seen in isolation. In a previous study of horses with chronic palmar foot pain (although one inclusion criterion was lameness of >2 months’ duration, all horses had been lame for considerably longer), mild or moderate focal or generalized increased signal intensity in fat-suppressed images was associated with trabecular thinning and widened intertrabecular spaces. High signal intensity in fat-suppressed images, associated with decreased signal intensity in T1W gradient echo images and mixed signal intensity in T2W GRE images, was associated with generalized osteonecrosis and fibrosis, with irregular trabeculae, adjacent adipose tissue edema, and prominent capillary infiltration.

A recent postmortem study of feet with advanced radiological abnormalities of the navicular bone demonstrated that increased signal intensity in fat-suppressed images correlated with areas of degenerate adipose tissues, with hemorrhage or replacement by fibrocollagenous material, or fluid-filled cystic spaces. Marrow space fibrosis was also previously reported in horses with advanced navicular disease.

In the Dyson et al study, horses with more recent onset lameness without any radiological abnormalities, we identified fat atrophy, with loss of clear definition of lipocyte cytoplasmic borders, accompanied by an interstitial capillary proliferation, perivascular or interstitial edema, fibrosis, enlarged spongiosa bone spaces, and thinned bone trabeculae, showing loss of bone with irregularly spiculated edges of moth-eaten appearance (Fig. 16). These changes are similar to those seen in emaciated horses with generalized serous atrophy of bone marrow fat in the medulla of the distal limb bones, although in those horses there was more widespread distribution of edema and accumulations of macrophages. Macrophages were not seen in the current study.

Our recent study was not a longitudinal study, so we do not know how changes in the spongiosa may progress. Increased signal intensity in fat-suppressed images parallel and dorsal to the palmar cortex seen in association with primary DDFT lesions may resolve over time, or occasionally more...
severe navicular pathology develops. However, diffuse increased signal intensity in fat-suppressed images in the spongiosa of the navicular bone associated with primary navicular pathology has often persisted unchanged at follow-up examinations in association with persistent lameness, although in horses with an acute-onset trauma of the navicular bone, both lameness and high signal intensity may resolve. In other osseous anatomical locations, increased signal intensity in fat-suppressed MR images may persist, despite resolution of pain and lameness, whereas in other horses pain also continues. It is possible that the difference between these clinical groups relates to what histological features the high signal intensity on fat-suppressed images represents and, therefore, whether these are likely to be reversible changes. The potential clinical importance of increased signal intensity in fat-suppressed MR images may persist, despite resolution of pain and lameness, whereas in other horses pain also continues.

Previous studies with the use of histomorphometry, tetracycline labeling of bone, and scintigraphic studies have indicated that there is evidence of increased bone turnover in association with some forms of navicular disease, even in the absence of radiological abnormalities of the bone. Increased RU predominantly reflects increased osteoblastic activity but is not synonymous with either pain or lameness and may reflect a functional adaptation to foot conformation and the biomechanical forces on the navicular bone or preclinical disease. Comparison between scintigraphy and MRI has demonstrated that many horses with focal moderate or intense IRU have abnormalities of the navicular bone detectable using MRI. However, scintigraphy can also produce false-negative results, indicating that pathological abnormalities of the navicular bone are not always associated with increased osteoblastic activity. Radiopharmaceutical uptake was normal in the navicular bone of seven of 11 limbs in which the principle abnormality on MR images was diffuse increased signal intensity in fat-suppressed images and hypointense signal in T1W GRE images, and there was mild IRU in the remaining four bones. In contrast, in three horses with...
acute-onset, severe, unilateral lameness associated with increased signal intensity in the spongiosa of the navicular bone in fat-suppressed images, which was believed to be traumatically induced, there was intense IRU. Lameness in these horses resolved with rest and time. If increased signal intensity in the spongiosa was the principle pathological change in horses with foot pain, without IRU, it was speculated that treatment with the bisphosphonate, tiludronate, may be of benefit, particularly in light of osteoclasts identified in the current study, but negligible improvement was seen in 12 of 12 horses.

Injuries of the Distal Sesamoidean Impar Ligament

The DSIL is an integral part of the podotrochlear apparatus, and we have demonstrated a significant association between the severity of grade of IRU in the navicular bone of lame horses, reflecting abnormal bone modeling, and combined lesions of the CSL and DSIL detected with the use of MRI. The DSIL consists of bundles of longitudinally oriented collagen fibers, interspersed by synovial invaginations from the DIP joint and the navicular bursa, and penetrating blood vessels. As a result, a normal DSIL has a heterogeneous appearance on MR images, and differentiation between anatomical variants and pathological changes is not always straightforward. Nonetheless, in a comparison of lame horses and age-matched control horses, high-field MR abnormalities were consistently more severe in horses with foot pain, characterized by osseous fragments or focal mineralization near the origin, irregularity of fiber pattern, swelling of the ligament, and adhesions to the DDFT. However, comparison of MRI findings with histopathology in the same group of horses revealed only fair agreement; thus, there is a significant risk of false-positive results.

To further characterize lesions of the DSIL and to determine relationships between the MRI appearance of the navicular bone and distal phalanx, 50 limbs from 28 horses were examined with the use of high-field MRI and histopathology. Magnetic resonance abnormalities of the DSIL, its origin on the navicular bone and its insertion on the distal phalanx were graded. Sections of the axial third of the DSIL were examined histologically and graded according to fiber orientation, integrity of fibroblasts, collagen architecture, and vascularity.

There were significant correlations between the presence of an OCLL in the distal one-third of the navicular bone, or a distal border fragment, or increased signal intensity in fat-suppressed images at the insertion of the DSIL on the distal phalanx and the histological grade of the body of the DSIL (Fig. 17). There were significant associations between an OCLL in the distal one-third of the navicular bone and the presence of either a distal border fragment, entheseous new bone at the insertion of the DSIL and dorsal (B) spoiled gradient echo and transverse short tau inversion recovery (C) high-field magnetic resonance images illustrating features that showed a positive correlation with histological abnormalities of the distal sesamoidean impar ligament (DSIL). (A) An osseous cyst-like lesion (OCLL) in the distal third of a navicular bone. (B) Distal border fragments at the medial and lateral angles of the bone (arrows), with concave defects in the parent bone and associated osseous reaction. Increased signal intensity in the distal phalanx at the site of insertion of the DSIL (arrows).
DSIL, swelling of the DSIL, and increased signal intensity in the DSIL in fat-suppressed images. There was a significant association between distal elongation of the palmar border of the navicular bone and the presence of one or more distal border fragments. There were also significant associations between swelling of the body of the DSIL and irregularity of its palmar border or increased signal intensity in fat-suppressed images in the DSIL.

Bone resorption at the insertion of the DSIL and/or DDFT on the distal phalanx has also been seen radiologically and/or with the use of MRI in association with lesions of either or both the DSIL and DDFT. Such lesions have been characterized histologically by bone necrosis.

The combination of swelling of the DSIL, irregularity of its palmar border and increased signal intensity in fat-suppressed images is more likely to reflect genuine injury than any one of these features alone. The results indicated that alteration in size, number, or symmetry of the synovial invaginations in the DSIL is not a reliable feature of injury.

Lesions of the DSIL are rarely seen in isolation, usually occurring in conjunction with other injuries of the podotrochlear apparatus. However, we recently documented a horse in which a distal border fragment of the navicular bone, an OCLL in the distal third of the navicular bone and focal distal sesamoidean impar desmitis were identified as the most likely causes of pain and lameness. No other lesions were identified on MR images. The OCLL was characterized histologically by enlarged bone lacunae containing proliferative fibrovascular tissue.

Distal Border Fragments and Navicular Disease

There is considerable controversy concerning the potential clinical significance of distal border fragments of the navicular bone. In a radiological study comparing 55 sound horses and 377 lame horses, we observed fragments in 3.7% and 8.7% of sound and lame horses, respectively. In lame horses, distal border fragments were present in 24.1% of horses with a diagnosis of primary navicular pathology and in 12.9% of horses with navicular pathology and other associated lesions. There was an association between fragments and the overall navicular bone grade ($P = 0.0013$), radiolucent areas at the angles of the distal border of the navicular bone ($P < 0.001$), and the number and size of the synovial invaginations along the distal border of the navicular bone ($P < 0.001$). It was concluded that fragments may be part of navicular disease. Further evidence was provided for this by a high-field MRI study of 427 horses. Fragments were classified as small, medium, or large on the basis of...
their MRI appearance and were graded 1 to 5 on the basis of changes in the adjacent distal border of the navicular bone. Large fragments were usually grade 4 or 5, whereas small fragments were usually grade ≤3. There was a significant association between the presence of a fragment and the total navicular bone grade (P < 0.001), OCLLs (P < 0.001), increased number and size of the synovial invaginations of the distal border (P < 0.0067), increased signal intensity on fat-suppressed images in the distal half of the navicular bone (P < 0.001), and size of distal border entheseophytes (P < 0.0067). There was an association between grade of the DSIL and grade 4 or 5 navicular bone fragments (P < 0.0086). The majority of fragments were associated with proximal extension of abnormal signal intensity in the adjacent navicular bone.

We also investigated the correlation between the presence of distal border fragments detected using high-field MRI and their radiological detection in 427 horses. Medium and large fragments were most likely to be detected radiologically especially if grade 4 or 5, but up to 43% of large fragments were missed. There were significant associations between the presence of a fragment on radiography and the total MRI grade of the navicular bone (P < 0.0001); the presence of a radiolucency at the junction between the distal horizontal and sloping borders of the navicular bone and both a fragment observed on MR images (P < 0.0001) and OCLLs on MR images (P < 0.0001). There was also an association between an increased number and size of the synovial invaginations along the distal border on radiographs and the presence of both a distal border fragment on MR images (P < 0.0001) and OCLLs on MR images (P < 0.0001).

On histology, partially detached bone islands were separated from the distal border by areas of fibroplasia or fibrocartilaginous metaplasia and were associated with focal osteonecrosis. Thus, increased signal intensity on STIR images in the distal border of the navicular bone associated with some fragments may reflect bone necrosis. There may be transverse fissures at the origin of the DSIL (Fig. 18). It has been suggested that movement of a fragment relative to the navicular bone may be a potential cause of pain. In addition, lesions of the DSIL observed in association with high-grade fragments could be painful because of the rich sensory innervation of the DSIL, particularly at its insertion.

5. Conclusions

Accurate lameness diagnosis remains challenging. As knowledge advances, more questions arise. Currently our diagnostic capabilities are superior to our ability to treat lameness successfully. Progress will be made by careful clinical observations, combined with accurate interpretation of diagnostic imaging and evidence-based studies documenting the responses to treatment in sufficiently large numbers of horses.

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References and Footnotes


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