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How to Use Manipulative Tests to Diagnose and Manage Equine Foot Pain

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A thorough examination and assessment of the equine foot forms an essential part of any lameness examination. Manipulative tests can give the examiner incredible insight into the pain the horse is feeling. This provides the examiner with the critical information necessary for the next diagnostic step, not only diagnostic but therapeutic information for relief of pain. Author’s address: 16445 70th Street, NE Elk River, MN 55330; e-mail: turner@anokaequine.com. © 2014 AAEP.

1. Introduction

The equine foot is the most common site for lameness to develop. It is therefore of utmost importance to perform a thorough examination of the foot in order to identify problems or predisposing factors that may lead to lameness. The recent use of magnetic resonance imaging (MRI) in equine lameness diagnostics has markedly improved our understanding of foot-related lameness. Unfortunately, MRI examinations frequently reveal more than one area of pathology. It is then imperative to rely on the clinical examination to determine a hierarchy of importance of the MRI identified lesions.

There are numerous causes of foot pain in the horse. These causes can be categorized as (1) conditions of the hoof wall and horn producing tissues, (2) conditions of the third phalanx, and (3) conditions of the podotrochlear region. Hoof problems would include: hoof wall defects, such as cracks that involve the sensitive tissue; laminitis, laminar tearing (local, due to hoof imbalance), separation or inflammation of the sensitive laminae from the insensitive laminae; abscess formation; contusions of the hoof causing bruising or corn formation; neoplasia, and pododermatitis (thrush or canker). Third phalanx problems include: fractures of the coffin bone (types I–VII), deep digital flexor insertional tenopathy, pedal osteitis (generalized or localized inflammation of the bone), desmopathy of the collateral ligaments, cyst-like lesion formation, and remodeling disease. Conditions of the podotrochlear region have been reported to include distal interphalangeal synovitis/capsulitis, deep digital flexor tendinitis, desmitis of the impar (distal navicular ligament) or collateral sesamoidean ligaments, navicular osteitis or osteopathy, vascular disease of the navicular arteries, and navicular fractures. The common denominator of all of these conditions is that they are characterized by pain that can be localized to the foot.

The examination requires comprehensive evaluation of the external hoof, evaluation for deep pain, evaluation of “hoof balance,” and evaluation of radiographs or other imaging modalities. The evaluation of the horse’s foot, like all examinations, requires a thorough medical, performance, and shoeing history. The purpose of this paper is to describe the value of manipulative tests for the diagnosis of
foot problems and to describe how the results of these tests can be used to help manage the foot pain.

2. Manipulative Tests

Diagnostic tests that should be performed are hoof tester examination, distal limb flexion, hoof extension wedge test, palmar hoof wedge test, and lateral medial wall wedge tests. Positive response to any of these tests is important but a negative response is equivocal and does not rule out any problem.

Hoof tester examination should be performed systematically; how you perform the exam is unimportant but get used to a routine. I like to begin at the heel on my left side and work around the hoof in a clockwise fashion. Begin with the bar, move to the heel, to quarter and then toe, and then back toward the heel on my right. Space the tester’s progress at approximately one-inch intervals. Be sure to include each exit point of the shoeing nails. Next, place the testers in each of the collateral sulci and across the hoof to the opposite hoof wall (I like to progressively move the hoof tester along the hoof wall caudal to cranial to check for alterations in the pain response; then place the testers in the central sulcus to the hoof wall at the toe and then across the heels). Remember that the closer the ends of the hoof testers are, the more accurate the exam is in localizing pain. A positive response should be repeatable and, in the frog region, the pain response should be uniform over those areas and must be evaluated in relation to examination of the remaining foot. That is, a positive response in the heels and quarters of the sole would also be expected to cause a positive response across the distal sesamoidean region in the same area of the foot. Pain is noted as a distinct withdrawal of the foot at the time pressure is applied.

A distal limb flexion test may exacerbate lameness if any of the three distal joints of the leg are affected by synovitis or osteoarthritis. A positive response could also be expected by any condition that causes induration of the tissues of the distal limb. The distal limb flexion test is performed by flexing the distal limb, holding the limb in that position, and trotting the horse away after 30 seconds. In the author’s opinion, the time is not critical but the examiner should always be consistent so as to develop a feel for the effect of the test. A positive test is noted if the manipulation causes lameness or lameness is exacerbated.

The hoof extension test is performed by elevating the toe with a block, holding up the opposite limb, and trotting the horse away after 60 seconds. The block the author uses is an old hoof knife wrapped in tape as protection from the blade. The palmar hoof wedge test is performed in a similar fashion except that the block is placed under the palmar two-thirds of the frog and forces the horse to stand on that foot. The opposite limb is held up for 60 seconds and the horse is trotted off. The test can be further modified so that the wedge can be placed under either the medial or lateral wall to determine if the pressure or hoof imbalance caused by the wedge exacerbates the lameness. As before, the opposite limb is held up for 60 seconds before the horse is trotted in hand. A positive test is once again noted if the manipulation causes lameness or lameness is exacerbated.

3. Diagnostic Significance

The significance of the hoof tester examination is obvious; it is a simple assessment of pain, with the painful area between the tips of the testers. Abscesses or fractures usually show the most reaction when using hoof testers. This is important so that further diagnostics, specifically radiography, can be centered on the exact point of pain. This improves the likelihood of finding a lesion. Less painful injuries would show less pain. Traditionally, pain over the frog upon hoof tester examination was indicative of navicular bone pain; however, one study showed that hoof testers only had a 50% predictive value for navicular pain. The author believes hoof testers are very accurate for assessment of pain in the sole or hoof wall but over the frog hoof tester pain only represents nonspecific deep pain in the foot.

Flexion tests have been shown to exacerbate 90% of foot lameness. However, there has not been shown to be any specificity. One lameness diagnostician suggests that if the limb is flexed under the torso pain is more likely from the fetlock; whereas if the limb is flexed by pulling it forward, this is more likely to exacerbate coffin joint lameness. This has not been confirmed but the author always flexes the limb by pulling the leg forward. If the test is positive the author will follow this test with medial lateral wedge tests.

The medial to lateral wedge will apply pressure to the wall under the wedge and stretch the capsule and collateral ligaments of the coffin joint opposite the wedge. The test appears to be more specific for collateral ligaments. The author reviewed their last 50 cases where the wedge test was positive; over 90% were positive with the wedge placed laterally, and 33 of the 50 showed a distal interphalangeal collateral ligament lesion upon ultrasonographic examination.

The frog wedge test has an 85% positive predictive value for navicular pain. This makes it the best single test for that condition. Whenever this test is positive, the author recommends a navicular bursectogram as part of the imaging assessment. The toe wedge test is the most commonly recommended test after flexion and hoof tester examination. However, the test has only a 50% predictive value for navicular pain and is only positive in about 50% of the cases. Despite those poor predictive values, the author uses a positive response as an indicator of deep flexor pain and performs sonography of the deep flexor in the pastern and foot. However, only a small percentage have had a deep flexor lesion.
4. Treatment Significance

Hoof tester examination pinpoints the region where, in the case of an abscess, decompression needs to be performed. Furthermore, the information can indicate the areas of the foot that need to be protected.

Flexion tests indicate to the author potential joint injection as part of the therapeutic options. Which joint depends on the response to diagnostic analgesics and radiography. An exception is a positive medial/lateral wedge test or sonographic finding of distal interphalangeal (DIP) collateral desmopathy. The author believes that the risk of making the injury worse is too great if the DIP joint is injected.

A positive frog wedge test is an indicator for treatment of the navicular bursa. The author uses this single test as a positive indicator for the diagnosis and treatment of navicular pain. A positive toe wedge test, in the author’s opinion, is an indicator to relieve pressure on the navicular bursa. An important point is that a positive frog wedge indicates that any frog pressure applied to the foot may cause more pain. This is important to know when deciding therapeutic options for shoeing. Positive lateral/medial wedge tests indicate DIP collateral ligament or joint capsule stress. These are cases that need rest if there is a lesion in the collateral ligament. In the absence of collateral lesions, the author recommends shoeing the horse with an asymmetric shoe with the wide branch under the opposite wall of the positive test. This prevents that wall from sinking into the ground and helps prevent the stress from being applied to the ligament during normal movement.

5. Conclusions

Manipulative tests of the foot provide unique information to the examiner that is both beneficial in determining further diagnostics but also provides information important in developing treatment strategies for the lameness.

Acknowledgments

Conflict of Interest

The Author declares no conflicts of interest.

References

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A club foot results from a flexural deformity of the distal interphalangeal joint that is characterized by a shortening of the deep digital flexor tendon musculotendinous unit. Flexural deformities are a problem not only in foals but are also responsible for the club foot conformation seen in adult horses. Treatment is most successful when the cause is investigated and therapy is initiated as early as possible and when the biomechanical properties of the foot are thoroughly understood. Flexural deformities in foals to mature horses are addressed through appropriate farriery, often combined with surgery. Author's address: Northern Virginia Equine, PO Box 746, Marshall, VA 20116; e-mail: sogrady@look.net. © 2014 AAEP.

1. Introduction

A club foot can be defined as an upright conformation of the foot associated with a flexural deformity of the distal interphalangeal joint (DIPJ). Grossly, the dorsal hoof wall angle is upright or steep accompanied by a broken forward foot-pastern axis. A flexural deformity of the DIPJ can be defined as a shortening of the musculotendinous unit of the deep digital flexor tendon (DDFT) that results in hyper flexion of the joint. The mechanism of this shortening of the musculotendinous unit is not well understood, and the initiating cause is often undetermined but may be related to lameness, nutrition, or genetic predisposition. Flexural deformities are not only observed in foals but are also responsible for the club foot conformation seen in mature horses. The deformity may be congenital or acquired and in many instances may have a genetic basis. Treatment is most successful when the cause is investigated, therapy is initiated as early as possible, and when the biomechanical properties of the foot are thoroughly understood. Furthermore, knowledge of the anatomical changes that occur in a club foot and biomechanical principles will enhance interaction with the farrier whose input will be necessary for a successful outcome. Flexural deformities in foals to mature horses are addressed through appropriate farriery, which is often combined with surgery. Flexural deformities have been traditionally referred to as “contracted tendons”; however, the primary defect appears to be a shortening of the musculotendinous unit rather than a shortening of just the tendon structure, thus making “flexural deformity” the preferred descriptive term. Shortening of the musculotendinous unit produces a structure of insufficient length to allow normal alignment of the distal phalanx (P3) relative to the middle phalanx, resulting in variable clinical signs ranging from an upright hoof angle to a club foot. As it seems many of the club feet seen in mature horses result from inappropriate management in the first year of life, this paper will discuss the management of club feet (flexural deformities) from birth to the adult horse. As a true club foot is synonymous with a flexural deformity, the terms will be used interchangeably throughout this paper.
2. Anatomy Review

In the antebrachium, the muscle bellies of the DDFT lie directly on the caudal aspect of the radius and are covered by the muscle bellies of the superficial digital flexor tendon (SDFT) and the flexors of the carpus. The deep digital flexor muscle consists of 3 muscle bellies (the humeral head, the inconsistent radial head, and ulnar head), which form a common tendon proximal to the carpus. This tendon, along with the SDFT, passes through the carpal canal and continues down the palmar aspect of the third metacarpal bone. Below the fetlock, at the level of the middle phalanx, the DDFT passes between the medial and lateral branches of the SDFT, continues distally, and inserts on the flexor surface of the distal phalanx (P3). A strong tendinous band known as the accessory ligament of the DDFT (AL-DDFT) originates from the deep palmer carpal ligament and fuses with the DDFT at the middle of the metacarpus (Fig. 1). The design and function of the anatomical structures is such that any prolonged shortening of the musculotendinous unit affects the position of the DIPJ. This palmar surface of the distal phalanx is pulled palmarly by this shortened musculotendinous unit, placing the DIPJ in a flexed position. The alignment of the bone within the hoof capsule remains constant while the hoof capsule is pulled with the distal phalanx. The flexed position of the DIPJ combined with the altered load on the foot leads to a rapid distortion of the hoof capsule and thus the club foot conformation. It can also be noted from the anatomy that transecting the AL-DDFT lengthens the musculotendinous unit either functionally or by allowing relaxation of the proximal muscle belly associated with the DDFT.

3. Classification of Club Feet

Traditionally, club feet or flexural deformities have been classified as type 1 where the hoof-ground angle is 90° or less and type 2 where the hoof-ground angle is greater than 90°. A recent method of classifying club feet using a grading system (grade 1–4) has been proposed. It would appear beneficial to classify the severity of the flexural deformity to devise an appropriate treatment plan and monitor the response to a given therapy. A grading system would also enhance record keeping as well as improve communication between the veterinarian, farrier, and owner with regard to treatment strategies and follow-up. A grade 1 club foot has a hoof angle 3° to 5° greater than the contralateral foot and a characteristic fullness present at the coronet. The hoof-pastern axis generally remains aligned rather than being broken forward. A grade 2 club foot has a hoof angle 5° to 8° greater than the contralateral foot, the angle of the hoof-pastern axis is steep and slightly broken forward, growth rings are wider at the heel than at the toe, and the heel may not touch the ground when excess hoof wall is trimmed from the heel. A grade 3 club foot has a broken-forward hoof-pastern axis, often a concavity in the dorsal aspect of the hoof wall, and the growth rings at the heels are twice as wide as those at the toe. A grade 4 club foot has a hoof angle of 80° or greater, a marked concavity in the dorsal aspect of the hoof wall, a severe broken-forward hoof-pastern axis, and the coronary band from the toe to the heel has lost all slope and is horizontal with the ground (Fig. 2). For simplicity, the author uses a grading system based on the severity or degree of flexion noted in the DIPJ on a well-positioned weight bearing lateral radiographic projection to classify flexural deformities. Any marked flexural deformity should be considered significant and treated accordingly.

4. Club Feet in the Young Horse

Club feet or flexural deformities in foals can be divided into congenital or acquired deformities. As such, congenital deformities are noted at birth, and acquired deformities generally occur from 2 to 8 months of age as the foal grows and develops.

Congenital Flexure Deformities

Congenital flexural deformities are present at birth, may involve a combination of joints (e.g., carpus, metacarpophalangeal, and distal interphalangeal joints), and are characterized by abnormal flexion of these joints and the inability of these joints to ex-
LAMENESS EXAMINATION AND THERAPY

Fig. 2. Grade 4 club foot. Note the broken forward hoof-pastern axis, fullness of the coronet, the disparity between hoof wall growth at the toe and the heel, the concavity in the dorsal hoof wall, and the poor hoof wall consistency at the ground surface of the capsule.

tend. Proposed etiologies of congenital flexural deformities include malpositioning of the fetus in utero, nutritional mismanagement of the mare during gestation, teratogens in various forages ingested by the mare, maternal exposure to influenza virus, or the deformities could be genetic in origin.2,6,8,10 The affected foal tends to walk on the toe of the hoof capsule, is unable to place the heel on the ground, and assumes a so-called “ballerina” stance. Treatment of foals with a congenital flexural deformity varies with the severity of the deformity. A mild to moderate flexural deformity in which the foal can readily stand, nurse, and ambulate is generally self-limiting and resolves without treatment. Brief intervals of exercise once or twice daily in a small paddock or on firm footing for the first few days of life may be all that is necessary for the deformity to resolve. If the condition is severe or has not improved by the third day post-foaling, every-other-day intravenous administration of oxytetracycline (2–3 g q 24 hrs) is frequently beneficial.2,10,11 A variety of bandaging techniques, often combined with splints, can be used to fatigue the muscle portion of the musculotendinous unit. Physical therapy to “stretch” the involved area may hasten recovery. Foals with bilateral congenital flexural deformities usually don’t have just 1 isolated structure or joint that is responsible for the deformity, therefore, in the author’s opinion, the use of a toe extension is not indicated. In the author’s experience, a toe extension will often impede movement and cause the foal to stumble when it attempts to ambulate.

Acquired Flexural Deformities

Acquired flexural deformities generally develop when the foal is between 2 and 8 months old and generally involves the DIPJ initially. It is commonly a unilateral condition but occasionally affects both limbs. The etiology of this deformity is unknown, but speculated causes include genetic predisposition, improper nutrition (i.e., overfeeding, excessive carbohydrate [energy] intake, unbalanced minerals in the diet), and excessive exercise.2,10 A recent study looking at grazing patterns in a small number of foals showed that foals with long legs and short necks had a tendency to graze with the same limb protracted.12 Fifty percent of the foals in this study developed uneven feet with a higher heel on the protracted limb leading researchers to feel there may be a possible correlation between conformational traits and an acquired flexural deformity. It is the author’s opinion that a large contributing factor to this syndrome is contraction of the muscular portion of the musculotendinous unit caused by a pain response, the source of which could be discomfort anywhere along the limb, physeal dysplasia, or trauma from foals exercising on hard ground. Discomfort may follow aggressive hoof trimming where excessive sole is removed, thus rendering the immature structures within the hoof capsule void of protection. The foal then becomes unwilling to bear full weight and is susceptible to trauma and bruising. Any discomfort or pain in the foot or lower portion of the limb coupled with reduced weight-bearing on the affected limb appears to initiate a flexor withdrawal reflex; this causes the flexor muscles proximal to the tendon to contract, leading to a shortened musculotendinous unit and an altered position of the DIPJ. This shortening of the musculotendinous unit shifts weight-bearing to the dorsal section of the foot causing a decrease in sole depth, bruising of the sole, reduced growth of the dorsal aspect of the hoof wall, and excessive hoof wall growth at the heel. As the flexural deformity may be secondary to pain in these cases, it is essential that a possible source of pain should be carefully evaluated and located by physical examination and, if necessary, by regional analgesia and diagnostic imaging. A genetic component must also be considered for acquired flexure deformities, as some mares consistently produce foals that develop a flexural deformity in the same limb of the dam or grand dam in which a similar deformity is present.10 However, at present, there is no conclusive scientific evidence to substantiate a genetic basis.

5. Mild Acquired Flexural Deformities (Initial Stage of a Club Foot)

Clinical Signs

The initial clinical sign of a club foot may only be abnormal wear of the hoof at the toe, which is often discovered by the farrier during routine hoof care. Closer or subsequent investigation may reveal that the dorsal hoof wall angle is increased and that after the heels of the hoof capsule have been trimmed to a normal length, the heels may no longer contact the

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A prominent coronary band may be present at this stage. Most foals affected to this degree may already have a mildly broken forward hoof-pastern axis. Increased palpable digital pulse, heat in the affected foot, and signs of pain when a small hoof tester is applied to the solar aspect of the toe dorsal to the frog are not uncommon clinical findings. Hoof tester pain is generally the result of trauma or excessive weight-bearing on the toe.

Treatment
Conservative treatment such as restricting exercise to reduce further trauma is paramount. Correcting the nutritional status of the foal (i.e., weaning the foal to avoid possible excessive nutrition from the mare and/or decreasing carbohydrates), administering an anti-inflammatory agent (NSAID) to relieve pain, administering oxytetracycline to facilitate muscle relaxation, and carefully trimming the hoof are, in the author's opinion, a good starting point. The NSAIDs should be administered short-term and judiciously in foals due to the potential side effects, such as gastroduodenal irritation and nephrotoxicity. For analgesia, the author will administer firocoxib (0.1 mg/kg bwt q 24 hr) or flunixin meglumine (1.1 mg/kg bwt q 24 hr) combined with a gastric protectant. Hoof trimming is directed toward improving the hoof angle by lightly trimming the heels from the middle of the foot palmarly until the hoof wall at the heels and the frog are on the same plane. The bars can be thinned or removed to possibly improve heel expansion, and the heels adjacent to the sulci should be angled to 45° to promote spreading. Breakover is moved palmarly by creating a mild bevel with a rasp, which begins just dorsal to the apex of the frog and extends to the perimeter of the dorsal aspect of the hoof wall (Fig. 3A and 3B). If improvement is noted, this trimming regimen is optimally performed at 2 week intervals. If the toe is constantly being bruised or undergoing abscessation, a hoof composite\textsuperscript{a,b} can be applied to the dorsal aspect of the sole and the distal dorsal aspect of the hoof wall to form a toe “cap” to provide protection. The acrylic composite-impregnated fiberglass or urethane composite used to form the toe cap covers the solar surface to the apex of the frog, protecting that area from further damage. A bevel toward the toe can be created in the composite with a rasp or Dremel\textsuperscript{®} tool to facilitate breakover. If there is adequate integrity of the dorsal section of the hoof wall, the author believes that application of a toe extension to be unwarranted and actually contraindicated. Farriers have traditionally applied toe extensions to create a lever arm using a shoe or a composite, but they only exacerbate wall separation and delay breakover. Furthermore, extensions may contribute to lameness due to excessive stresses on the DDFT when the foal puts full weight on its foot. The above treatment can be temporary, appears to work best when initiated at the first sign of foot deformity before a marked flexural deformity is noted and, when possible, following elimination of any possible inciting causes. The farriery should always be combined with restricted exercise. If the affected foot continues to improve or does not regress, conservative treatment is continued. If a mild flexural deformity progresses in severity to the stage where a marked radiographic flexural deformity is noted, the foal becomes a surgical candidate.

6. Severe Acquired Flexural Deformities (Club Foot)

Clinical Signs
A mild acquired flexural deformity may progress in severity despite conservative treatment, or a severe acquired flexural deformity may be acute in onset. A severe acquired flexural deformity is characterized by a foot with a hoof angle greater than 80°, a prominent fullness at the coronary band, and a broken forward pastern-axis, disparity in the length of the

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**Fig. 3.** A mild flexural deformity before trimming (A) and after trimming (B). Note the change in breakover created under the dorsal hoof in B.
heel relative to the toe of the hoof, and heels that fail to contact the ground (Fig. 4). If the flexural deformity is allowed to persist, the foot eventually assumes a boxy, tubular shape due to the overgrowth of the heels to accommodate the lack of ground contact; heel length will approach the length of the toe. Increased stress on the toe will eventually cause a concavity along the dorsal surface of the hoof wall. Stress exerted on the sole-wall junction in the toe area will cause it to widen, allowing separations to occur.

The diagnosis is straightforward and based on the characteristic foot and limb conformation described above. Radiographs should be used to confirm the diagnosis and assess changes in the joint. The author will administer mild sedation (half the recommended dose of xylazine [0.33–0.44 mg/kg, IV] combined with butorphanol [0.022–0.066 mg/kg] IV) and place each of the foal’s feet on separate wooden blocks of equal height, which allows normal loading of both forefeet. Lateral-to-medial weight bearing images of both forefeet should be obtained. The degree of flexion of the DIPJ, the angle of the dorsal hoof wall, and abnormalities at the margin of the distal phalanx should be assessed (Fig. 5).

Treatment

When a marked flexural deformity is present and confirmed by radiographic examination of the feet, conservative treatment and hoof trimming alone are generally unsuccessful in resolving the problem. Elevating the heels has been advocated to reduce tension in the DDFT and to promote weight-bearing on the palmar section of the hoof. However, although elevating the heels improves the hoof-pastern axis and makes the foal more comfortable initially, the author has not been able to subsequently lower the heel or remove the wedge and establish a normal hoof angle with the heel on the ground. Once a marked flexural deformity of the DIPJ and distortion of the hoof capsule is apparent or progressing, the author recommends transection of the AL-DDFT combined with the appropriate farriery.

7. Desmotomy of the AL-DDFT

The author has been consistently successful in treating foals with severe flexural deformities with desmotomy of the AL-DDFT combined with the appropriate farriery. A toe extension is not used at the time of surgery, but an acrylic composite is applied to the solar aspect of the toe to create a reverse wedge. The wedge affords protection for the toe region and appears to redistribute the load to the palmar aspect of the foot, thus mildly increasing the stresses on the DDFT, and restores the concavity to the sole. The farriery is generally performed prior to the surgery either before or while the foal is

Fig. 4. Severe flexural deformity. Note the prominent coronet, the steep angle of the dorsal hoof wall, the load being placed on the toe, and the heels of the hoof capsule off the ground.

Fig. 5. Radiographic view of the right front (RF) foot shows a broken forward hoof-pastern axis when compared to the left front (LF) foot which shows normal alignment of the digit.
anaesthetized to prevent manipulating the limb and handling the surgical site following the procedure. The heels are lowered from the point of the frog palmarly, until the sole adjoining the hoof wall (sole plane) at the heels becomes solid. Any concavity in the dorsal aspect of the hoof wall is removed with a rasp. The ground surface of the foot dorsal to the frog and the perimeter of the dorsal hoof wall are prepared for a composite using a rasp or Dremel® tool. Deep hoof wall separations in the sole-wall junction at the toe are explored and filled with clay, if necessary, to prevent infection beneath the composite. Foals undergoing this procedure are usually between 3 and 5 months old, and so, because of their size and weight, reinforcing the composite with fiberglass is necessary. A small section of fiberglass is separated into strands and mixed with the composite. The composite is applied to the solar surface of the foot beginning at the apex of the frog and extending to the perimeter of the hoof wall where a thin lip is formed. The aluminum is pushed down so that the composite material extrudes through the holes, and the aluminum plate is then covered with additional composite. This additional reinforcement allows the older foals to be walked daily or turned out in a small paddock without the composite wearing out. The foal is placed under general anesthesia, and the surgery is performed in a routine manner as described elsewhere.8,10,14–17

8. Aftercare
The surgical aftercare is at the discretion of the attending clinician. Controlled exercise in the form of daily walking or turn-out in a small paddock with firm footing such as a round pen is essential. There is the potential for pain with the initiation of exercise, requiring close monitoring of the foal, and exercise should be increased sequentially. The foal is trimmed at roughly 2 week intervals, based on the amount of hoof growth at the heels with the objective of establishing normal hoof capsule conformation. The composite wedge is removed 1 month after the surgery. At subsequent trimmings, the heels are lowered as necessary from the middle of the foot palmarly, and hoof wall at the toe is trimmed from the dorsal aspect of the hoof wall until the desired conformation is attained. No sole dorsal to the frog is removed. When the desired conformation is reached, the foot is trimmed in a routine manner on a monthly basis. It is important to emphasize that when the hoof capsule returns to an acceptable conformation, only that portion of the sole that is shedding should be removed. This avoids causing discomfort in the dorsal solar area that can result in the horse redeveloping, to some degree, the original deformity.

9. Flexural Deformities in the Mature Horse

Club Feet
There is minimal information in the veterinary literature regarding the management of a mature horse with a club foot. An upright conformation of the foot associated with a flexural deformity of the DIPJ is defined as a club foot2,10,18–21 (Fig. 7). A flexural deformity is generally diagnosed and treated while the horse is immature but often a mild flexural deformity is ignored or the foal is treated inappropriately. When the horse enters training, the existing flexural deformity may become exacerbated by the type and amount of exercise, inadequate farrier care, such as inappropriate or infrequent trimming and shoeing, or some type of underlying disease. When a club foot conformation is acquired in the adult horse, it is almost always secondary to an underlying cause or disease, such as an injury that results in a non-weight bearing lameness, excessive trimming of the toe resulting in solar pain, chronic low-grade laminitis, or chronic heel pain. Furthermore, flexural deformities have been reported as a cause of decreased athletic performance and chronic, low-grade lameness in the mature horse.18–21 The altered biomechanics of the foot result in an increased load (i.e., weight-bearing) being placed on the dorsal section of the foot leading

Fig. 6. Illustration of the lateral side of the foot shows the placement of a reverse wedge after the foot has been trimmed. Illustration of the ground surface of the foot shows the composite wedge reinforced with an aluminum plate.
to decreased sole growth, sole bruising, a shortened stride on the affected limb, and various degrees of lameness and poor performance. The majority of horses with a club foot maintain soundness and ability to perform often with minimal therapeutic farriery, yet the club foot conformation and the altered load on the foot may be responsible for poor performance or lameness.

The Hoof Capsule Distortion

To apply the appropriate farriery, understanding the proposed mechanism leading to the club foot conformation is helpful. When a flexural deformity is present, the musculotendinous unit is shortened, the degree of which is dependent on the amount of flexion in the DIPJ. This causes a disparity of hoof wall growth, with more growth at the heel than at the toe to compensate for the decreased length of the musculotendinous structures. The frog generally recedes below the hoof wall due to the excessive hoof wall growth at the heels so that the energy of impact is assumed entirely by the hoof wall, bypassing the soft tissue structures and transferring the load directly onto the bones of digit through the laminar interface. The flexural deformity, combined with the excess hoof wall growth at the heels, places the DIPJ in flexion and the distal phalanx in an abnormal alignment relative to the digit; this promotes toe-first landing and, therefore, excessive load on the dorsal section of the joint and hoof capsule. Hoof abnormalities associated with a club foot conformation are thin flat soles, poor hoof wall consistency, especially at the toe, toe cracks, hoof wall separation, and “white line disease.”22 Injuries associated with a high hoof angle are thought to include inflammation of the DIPJ due to abnormal loading of the joint, sole bruising, and increased strain on the suspensory ligaments of the navicular bone.19,23

Radiography

Good quality radiographs, consisting of lateral to medial and a weight bearing (horizontal 0°) dorso-palmar views, are necessary for the clinician and farrier to evaluate the condition and apply the appropriate farriery for the club foot. Good soft-tissue detail allows distortion of the hoof capsule to be accessed.9 A lateral to medial radiograph reveals the weight-bearing properties of the foot, allows assessment of the hoof capsule, the position of the distal phalanx within the hoof capsule, solar depth, length of the heels, the osseous integrity of the perimeter of the distal phalanx, and the severity of the flexural deformity of the DIPJ. The degree of flexion indicates the amount of shortening of the musculotendinous unit. The radiographs are used to diagnose any pathology present, determine treatment options, and can be used as a template for farriery (Fig. 8).

10. Therapeutic Farriery

Therapeutic farriery forms the mainstay of treatment for club feet. Farriery should be based on principles rather than a particular method, and the principles remain the same regardless of the severity of the flexural deformity.18,19,22,24 The principles are to achieve normal alignment between the first, middle, and distal phalanges and thus normal orientation and loading of the distal phalanx relative to the ground. Trimming and shoeing is aimed at removing weight-bearing from the toe and dorsal aspect of the distal phalanx and reestablishing weight-bearing to the entire solar surface of the distal phalanx and the corresponding hoof capsule.
Historically, farriers have been taught to trim (lower) the heels to correct the distorted hoof capsule and promote weight-bearing in the heel area, but this type of trimming comes with a price. As the severity of the flexural deformity increases, so too does the shortening of the musculotendinous unit; therefore, lowering the heels directly increases the tension within the musculotendinous unit, and these stresses may lead to irresolvable tearing of the dorsal lamellae, widening of the sole-wall junction similar to that seen in the chronic laminitic hoof, and increased pain. The increased forces placed on the DDFT from this type of trimming also promote hoof capsule distortion and abnormal loading. Furthermore, if there is pathology present in the soft tissue structures of the palmar foot, decreasing the height of the heels is likely to place more strain in this section of the foot.

11. Farriery

Distinguishing between a foot with steep hoof angle and a true club foot is important. High hoof angles without phalangeal misalignment or with mild phalangeal misalignment can generally be managed by adhering to good farriery guidelines for trimming such as using the hoof-pastern axis, the center of rotation, and trimming the heels of the hoof capsule to include the frog. It may be necessary with a high hoof angle or mild phalangeal alignment to gradually trim the heels in a tapered fashion from the apex of the frog to the heels. This increases the ground surface of the foot and attempts to reestablish weight-bearing on the entire solar surface of the foot. Breakover is moved palmarly at the same time to compensate for any increased tension in the DDFT created by lowering the heels. This can be accomplished by rolling, rockering, or grinding breakover into the toe of the shoe. If improvement is noted, the horse should be trimmed/shod at 4 week intervals.

Farriery to correct a high hoof angle accompanied by a flexural deformity (club foot) becomes more of a challenge. Again, the object of farriery is to load the heels, compensate for the shortening of the DDFT, and improve the hoof-pastern axis. To accomplish these objectives, farriery is directed at trimming the heels of the hoof capsule, but the amount of heel to remove can be difficult to determine. In mild to moderate club feet, an estimate of how much heel to remove can be made by placing the thick end of a 2° or 3° pad under the toe of the foot and allowing the horse to stand on it (Fig. 9). If the horse does not resent the tension this places on the DDFT, this test allows the farrier to safely trim the hoof wall at the heels in a tapered fashion starting in a palmar direction from the widest part of the foot using the thickness of the degree pad as a guide. The toe is shortened by trimming the outer surface of the dorsal hoof wall with a rasp. The trimmed foot is fitted with a shoe that has the breakover forged or ground into it starting just dorsal to the apex of the frog and tapering toward the toe to further decrease the stresses on the DDFT. There are also commercial shoes available that have a rockered toe that provide appropriate breakover.

With the more advanced cases of club feet, the heels should still be lowered to load the heels and unload the toe, but the addition of heel elevation following the trim is necessary to compensate for the shortening of the musculotendinous unit. The concept of lowering the heels with the trim then wedging the palmar aspect of the hoof back up is often not understood. When the heels are trimmed back to the widest point of the frog, the load bearing surface area of the foot increases, and this is necessary for normal function of the hoof. However, the musculotendinous unit must be accommodated and maintained without excessive tension and pain. This is accomplished by decreasing the breakover and by adding elevation to the palmar aspect of the hoof. The degree of wedge that is applied often mimics the amount of heel removed, but in many cases may be less due to mechanical contributions made by rockering or rolling the toe of the shoe. The amount of heel elevation needed if necessary can be demonstrated following the trim by placing the trimmed foot on the ground 6 to 8 inches palmar to the contralateral limb. A space will generally appear between the heels of the foot and the ground (Fig. 10). The author uses either a wedge shoe or places a degree pad or a bar wedge between the heels of the foot and the shoe to compensate for the shortening of the muscle-tendon unit (Fig. 11). This method allows the heels to be weight-bearing but at the same time decreases the stresses on the musculotendinous unit. Creating breakover in the shoe to further relieve stress in the DDFT, as described above, is essential. It is important to note that when the heels are elevated with a wedge shoe, the normal
ground reaction forces and load bearing structures are altered. To redistribute the load, it is beneficial to apply a “pour-in” pad or impression material to the sole of the hoof between the branches of the shoe to reestablish load sharing of the weight bearing structures of the hoof (Fig. 12). Without increasing the surface area over which the ground reaction force is distributed, the heels may become overloaded over time and possibly result in quarter cracks, contracted heels, and subsolar bruising of the heel region. Following the farriery, it is necessary to see the horse have a flat strike pattern rather than a toe first landing. If the horse does not land flat, heel elevation should be considered.

In severe cases of club feet, the hoof wall consistency may be compromised such that alternative methods of application (other than nailing) may need to be integrated. The most common method of “gluing” a shoe on is termed “direct gluing,” which attaches a shoe directly to the weight bearing surface of the hoof wall and sole via some type of adhesive. Aluminum rather than steel shoes are typically used for this purpose because aluminum is very porous and the adhesives adhere with more affinity. The same principles for shoe fit are used with glue-on shoes vs. shoes that are nailed. Recently, polyurethane shoes have reached the marketplace and can be applied via the direct gluing technique (Fig. 13). These shoes are mentioned because they are flexible, allow the heels to expand, yet still carry the advantage of being attached with an acrylic adhesive (photo courtesy of Curtis Burns).
adhesive. Although glue-on shoes will not take the place of traditional farriery, they provide an alternative when necessary to prevent the compromised club foot from continuing to lose sole depth and develop further hoof capsule distortion by not having a shoe applied. It is always the goal, however, to transfer the horse into the most normal and simple shoeing/trimming protocol when possible.

12. Farriery Combined With Surgery

In selected cases, horses with a severe flexural deformity or horses that have not responded to appropriate farriery and remain lame may benefit from a desmotomy of the AL-DDFT.\textsuperscript{2,10,18,19,21,23,25} This release procedure, along with therapeutic farriery, allows realignment of the distal phalanx within the remainder of the digit, loads the entire surface of the hoof capsule, and readily allows the accompanying distortion of the hoof capsule to be improved. It is the author’s opinion that if this surgery is being contemplated, it should be performed early in the horse’s athletic career, before there is a significant hoof capsule distortion and before pathological changes involving the DIPJ and or the margin of the distal phalanx become evident on radiographs.

The surgery is generally performed under general anesthesia but in the mature horse, it can be performed standing using sedation and local or regional analgesia if necessary. If surgery is performed with the horse standing, the heel should be elevated by taping a 12° wedge to the foot to decrease tension in the AL-DDFT/DDFT complex allowing the ligament to be easily identified, separated, and transected away from the cutaneous incision. The client should be forewarned that the surgery involves an extended recovery period, and a blemish or fibrous thickening at the surgery site is inevitable due to the mature nature of the tissue. Caution is advised when performing the farriery that accompanies the surgery because the soft tissue structures within the hoof capsule and the digit have adapted/accommodated for the distortion of the hoof capsule. The author will trim the palmar section of the foot moderately using the information obtained from the radiograph for guidance and then apply two or three 2° wedge pads using either a shoe or a cuff (Fig. 14). After surgery, the horse is walked daily, and a degree pad is removed every 7 to 10 days depending on the comfort of the horse. After 3 weeks, the horse is allowed turnout in a small paddock such as a round pen for an additional 3 weeks and then turnout in a larger area for 3 to 6 months before exercise is resumed. The appropriate farriery must be maintained post surgery or the foot will have a tendency to revert to the original foot conformation. The cosmetic appearance of the limb is maximized by keeping the limb bandaged for the first 6 weeks. In a limited number of cases that the author has managed or consulted on, the benefits returning the horse to soundness have far outweighed the rehabilitation process being labor intensive. The author has not realized any benefit in applying a toe extension to the shoe in the adult horse following surgery. Many mature horses with a club foot frequently have damage to the dorsal lamellae and a stretched white line similar to that found in horses with laminitis, and therefore, toe extensions may markedly exacerbate detrimental mechanical forces on the lamellae and dorsal hoof wall.

13. Conclusion

The club foot is an important cause of equine lameness, decreased athletic performance, and a challenge to the veterinarian and farrier. The clinician must recognize and understand the altered mechanics that are placed on the osseous structures within the hoof, the DIPJ, and on the hoof capsule that accompany a flexural deformity involving the DIPJ. This understanding allows the clinician to apply the appropriate treatment and appreciate subsequent improvement. Additionally, it is essential to look beyond the deformed foot to identify and remove, if possible, any underlying cause(s). As with most disorders, early recognition and intervention signifi-
LAMENESS EXAMINATION AND THERAPY

Significant increase the chances for a successful outcome. This is especially true when dealing with the young horse. A thorough physical examination, high quality radiographic imaging, understanding biomechanical principles, familiarity with composite materials, and surgical competence are all essential to properly treat horses presented with a wide range of club foot abnormalities. Owners and trainers must be informed regarding the severity of any individual horse’s flexural deformity, the treatment options, the expected outcome, and the aftercare. Knowledge, skill, and interaction between the veterinarian and farrier are necessary for a successful outcome when treating a horse with a club foot, regardless of whether treatment is limited to farriery or combined with surgery.

Acknowledgments
Conflict of Interest

The Author declares no conflicts of interest.

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Characterization of Equine Hoof Lamellar Tissue Microanatomy With Fluorescent Markers

Hannah L. Galantino-Homer, VMD, PhD, DACT*; Robert K. Clark, PhD; and Renata L. Linardi, DVM, PhD

Equine histological studies are impaired by the lack of reagents validated for use on horse tissues. This paper describes the use of fluorescently-conjugated lectins and antibodies for the identification of tissue and cellular microanatomy in equine hoof lamellar tissue. Structures that can be specifically labelled include connective tissue extracellular matrix, epidermal basal cells, and subcellular components of epidermal cells such as cell membranes, cytoskeletal intermediate filaments, and cytolinkers that join the cytoskeleton to adhesion complexes. Authors’ addresses: Department of Clinical Studies, New Bolton Center, 362 West Street Rd., Kennett Square, PA 19348 (Galantino-Homer, Linardi); and STEM and Health Division, Cumberland County College, Vineland, NJ 08360 (Clark); e-mail: hghomer@vet.upenn.edu. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction

Our long-term goal is to improve the understanding, treatment, and prevention of equine laminitis. Laminitis is a common and debilitating disease characterized by tissue damage, inflammation, and dysplasia of the epidermal and dermal lamellae that can result in mechanical failure of the suspensory apparatus of the distal phalanx causing chronic pain and lameness, frequently necessitating euthanasia. Equine laminitis results in the generation of abnormal hoof capsule growth and formation of the lamellar wedge, a cornified but weak wedge of epidermal tissue produced by hyperplastic, metaplastic/dysplastic keratinocytes lying between the third phalanx and the hoof wall. Relative few studies have investigated the histological localization of the altered protein/glycoprotein expression during laminitis pathogenesis. These studies are essential to an improved understanding of laminitis pathogenesis since laminitis is, ultimately, a disease of tissue structural failure and altered differentiation. Indirect immunofluorescence microscopy is a useful tool for these studies due to its high sensitivity and specificity, ability to precisely determine antigen colocalization, applicability to confocal microscopy, and amenability to quantification.

We have demonstrated that the metaplastic epidermal lamellae in horses with chronic laminitis have significantly decreased expression of the nuclear transcription factor, p63, a marker of epidermal stem cell proliferative capacity, consistent with the altered growth, differentiation, and regenerative capacity of this tissue. It has also recently been demonstrated that epidermal basal cell proteoglycan depletion in concert with increased metalloproteinase (ADAMTS-4) activity occurs early in the development of the lesion. Wang and co-workers have shown that suppression of the canonical Wnt signaling pathway in lamellar tissue from horses

NOTES
with laminitis is associated with changes in lamellar epidermal expression of cell-cell and cell-matrix adhesion proteins, which is also consistent with altered cell and tissue differentiation.9 We have characterized the keratin isoform composition of normal hoof lamellae by quantitative proteomic analysis, including the identification of 2 novel isoforms that comprise more than half of the keratin content,10 but the expression of these keratin isoforms in laminitic horses and in relation to basal cell keratin markers has not been investigated. For all of these studies, indirect immunofluorescence utilizing commercial antibodies validated for use on equine tissues have provided an effective means of identifying protein localization. However, without appropriate fluorescence counterstains that identify specific tissue and cellular structures, the relative microanatomical localization of proteins of interest cannot be precisely documented.11,12 Even nuclear counterstains fail to provide any information on the structure of matrix-rich connective tissues.11,12 Suitable counterstains include double-label indirect immunofluorescence with cell type-specific, connective tissue and subcellular-localizing antibodies. In addition, plant lectins, which specifically bind to saccharide moieties within tissues (13 and references therein), may have utility as counterstains. The object of this study was to identify and characterize antibody- and lectin-based fluorescent markers of lamellar microanatomy.

2. Materials and Methods

Blocks of hoof tissue from the mid-dorsal stratum internum and including the hoof epidermal and dermal lamellae were dissected from humanely-euthanized nonlaminitic and chronic laminitic horses according to a protocol approved by the Institutional Animal Care and Use Committee, as described.5 Tissues were fixed in 4% paraformaldehyde for 24 hours and then dehydrated in 10% and 30% sucrose, respectively, for 24 hours each. Tissue blocks were coated with embedding mediuma and snap-frozen in liquid nitrogen-chilled 2-methylbutane. Tissue blocks were stored at −80°C prior to cryostat sectioning at 6 microns. Sections were air-dried and stored at −20°C until needed. Indirect immunofluorescence histology was performed on cryosections as described elsewhere.5,10 Tissues and cells were incubated with primary antibodies overnight at 4°C followed by fluorescent dyeb-conjugated secondary antibodies incubation at room temperature for 1 h.

The following primary antibodies were used at the following dilutions: mouse monoclonal anti-K14, clone LL002® (1:100) and rabbit anti-desmoplakin I and II antiserum® (1:400). The following secondary antibodies were used: fluorophore 488-conjugated goat anti-mouse IgG® [green] (1:500) and fluorophore 488-conjugated goat anti-rabbit IgG® [green] (1:500). Nucleic acids were stained with 4’,6-diamidino-2-phenylindole, dilactate® (DAPI, 0.5 μg/ml) for a 5 min incubation at room temperature. Sections were mounted using mounting medium® for light microscopy with epifluorescence illumination and confocal microscopy® using software for confocal imaging®.

3. Results

As shown in Figs. 1 and 2, keratin-14 (K14) is restricted to the basal epidermal cells of the secondary epidermal lamellae (SELS) in nonlaminitic horses, as detected by indirect immunofluorescence with the mouse monoclonal anti-K14 antibody. K14 localization is abnormal in laminitic tissue and extends into suprabasal layers.

As shown in Fig. 2, fluorescently-conjugated WGA stains the plasma membrane regions of the SEL basal and suprabasal cells and connective tissue in the primary and secondary dermal lamellae from nonlaminitic horses. Double labeling using K14 indirect immunofluorescence and fluorescently-conjugated WGA allows the precise demarcation of the membrane and cytoskeletal components of epidermal basal cells (Fig. 2B). Fluorescently-conjugated UEA stains epidermal, but not dermal, lamellar cells with the highest intensity apparent in cytoplasmic and plasma membrane regions of suprabasal cells adjacent to the keratinized axis (data not shown).

As shown in Fig. 3, the localization of WGA to the epidermal cell membrane allows the precise localization of the cytolinker protein, desmoplakin, to the epidermal cell membrane, but not along the basal side of the cells adjacent to the extracellular basement membrane zone in the primary and secondary epidermal lamellae from nonlaminitic horses. Colocalized WGA (red) and desmoplakin (green) along epidermal cell membranes appear yellow to yellow-green in merged images (Fig. 3B). Figure 4 illustrates the use of fluorescently-conjugated WGA combined with K14 immunofluorescence and DAPI staining to demarcate cell nuclei, extracellular matrix connective tissue, epidermal lamellar cell membranes, and epidermal basal cell cytoskeletal intermediate filaments.

4. Discussion

The high prevalence and frequently poor outcomes associated with laminitis make it a high priority for equine practitioners and veterinary medical researchers alike.14 The varied etiologic pathways of...
this condition combine with the complex tissue architecture to make definitive answers regarding even the most basic questions elusive. Immunofluorescence techniques are frequently employed in efforts both to identify cells that may play important roles in the development of laminitis and to identify molecules that may be impacting those cells. Better localization of each of these target types is dependent on our ability to recognize the non-immunoreactive tissues immediately adjacent to the fluorescent signal. This precise identification is particularly difficult when good technique and reagents render the best results, specifically when there is a strong immunofluorescence signal with little to no background fluorescence. By identifying fluorescent markers for specific foci within the lamellae, we increase the power of our immunofluorescence technique to do exactly what we intend it to do—enable us to identify the precise location of our target antigen.

While this goal has been of interest to our laboratory for some time, we did not originally set out to identify such markers. We fortuitously saw the utility of fluorescent lectin markers as counterstains

Fig. 1. Localization of K14, a basal epidermal cell marker, in nonlaminitic and laminitic mid-dorsal lamellae. (A) and (B) show comparable, mid-primary epidermal lamellar regions from a nonlaminitic (A) and laminitic (B) horse, with the primary epidermal lamellar keratinized axis and adjacent primary dermal lamellae indicated by an asterisk (*) and (PDL), respectively. (C) is from the axial region (primary epidermal lamellar tip) of a laminitic horse. (A) K14 (red) is localized exclusively to the basal epidermal cell layer (arrowhead) of the secondary epidermal lamellae (SELs) in nonlaminitic horses. In horses with chronic laminitis (B, C), K14 positive cells extend into the suprabasal layers (arrows) in areas of lamellar dysplasia and hyperplasia. Scale bar = 50 μm, images captured from epifluorescence microscopy with a 20× objective as described in the Materials and Methods section. This experiment was performed at least 5 times using lamellar tissue from 5 different nonlaminitic horses and 5 different laminitic horses. Representative images are shown.

Fig. 2. Localization of K14 combined with WGA counterstaining on equine hoof lamellae. Red (A), green (C), and merged (B) images of a lamellar tissue region from a nonlaminitic horse that includes portions of a primary dermal lamella (PDL), secondary dermal lamellae, as indicated by the asterisk (*), keratinized axis of a primary epidermal lamella (KA), and secondary epidermal lamellae. Red fluorescence demonstrates localization of WGA binding to extracellular matrix fibers in the primary and secondary dermal lamellae and to cell membranes in secondary and primary epidermal lamellae (A, B). Green fluorescence demonstrates immunolocalization of K14 to the cytoplasm of basal cells of the secondary epidermal lamellae (B, C). Note that red WGA fluorescence augments the precise localization of the green K14-positive cells. Scale bar = 25 μm, images captured by confocal microscopy with a 63× objective as described in the Materials and Methods section. This experiment was performed at least 6 times using lamellar tissue from 6 different nonlaminitic horses. Representative images are shown.
when employing this technique in our investigation of the breakdown of proteoglycans at the onset of laminitis. The cytoplasm of the basal epidermal cells of the SEL have been shown to contain aggregan, a member of the chondroitin sulfate proteoglycan family.\(^8\) This large molecule carries numerous covalently-linked chondroitin sulfate and oligosaccharide side chains.\(^{15}\) Given its role in the extracellular matrix of cartilage, it may be expected that, through osmotic attraction of water, it contributes a cushioning effect within the hoof.\(^8\) In fact, it may be degraded quite early in a number of disease processes including laminitis.\(^7,8,16\)

It is against this backdrop that we sought to examine carbohydrate content of hoof lamellae epidermal basal cells through the use of lectins. Lectins have been used in the characterization of carbohydrate expression in epidermal cells and adnexae for decades and across many species including the dog,\(^{13}\) horse,\(^{17,18}\) and human.\(^{19,20}\) The complex, stratified cytoarchitecture of these tissues is generated by pathways of differentiation that lead through distinct cell phenotypes.\(^{21}\) The above referenced studies demonstrate that these phenotypes can be distinguished based on their lectin binding pattern.

Our studies quickly yielded interesting findings other than the answers we sought. The affinity of WGA for fibers in the extracellular matrix and membranes of epidermal cells render it a rapid, economical, and simple counterstain to immunofluorescence studies on equine hoof lamellae. We then questioned which other reagents might be similarly useful. The affinity of UEA for suprabasal cells, especially those at the intersection between SELs

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**Fig. 3.** Localization of desmoplakin combined with WGA counterstaining on equine hoof lamellae. Red fluorescence demonstrates localization of WGA binding to extracellular matrix fibers in the dermal corium (●) and secondary dermal lamellae (*) and to cell membranes in the primary epidermal lamella (PEL) and secondary epidermal lamellae (A, B) from a nonlaminitic horse. Green fluorescence demonstrates immunolocalization of desmoplakin to the cell membrane of basal (arrowhead) and suprabasal (arrow) cells of the secondary epidermal lamellae but not to the basal membrane of basal cells (B, C). Colocalization of red WGA fluorescence with green desmoplakin fluorescence appears yellow-green. Scale bar = 25 μm, images captured by confocal microscopy with a 63× objective as described in the Materials and Methods section. This experiment was performed at least 6 times using lamellar tissue from 6 different nonlaminitic horses. Representative images are shown.

**Fig. 4.** Triple-fluorescence staining to identify multiple tissue components of equine hoof lamellae. Red fluorescence demonstrates localization of WGA binding to extracellular matrix fibers in the primary dermal lamellae (PDL) and secondary dermal lamellae (*) and to cell membranes in secondary and primary epidermal lamellae (keratinized axis [KA] indicated). Green fluorescence demonstrates immunolocalization of K14 to the cytoplasm of basal cells of the secondary epidermal lamellae (arrowhead). Blue fluorescence demonstrates localization of DAPI staining to cell nuclei. Image captured by epifluorescence microscopy with a 20× objective as described in the Materials and Methods section. This experiment was performed at least 5 times using lamellar tissue from 5 different nonlaminitic horses. A representative image is shown.
and the KA, make it potentially useful as a marker of differentiation in the epidermal keratinocytes of the hoof lamellae. Moreover, altered UEA reactivity might be consistent with tissue metaplasia and the resultant widening of the keratinized axis of primary epidermal lamellae during lamellar wedge formation. On the other hand, a marker that identifies the less differentiated epidermal cell population would be equally useful. Antibodies against the intermediate filament protein K14 specifically label this cell type. The abnormal localization of K14 to suprabasal cells in the lamellar wedge is consistent with the presence of dysplastic epidermal cells in this tissue. Altered epidermal differentiation might contribute to laminitis disease pathogenesis.

Since the cytolinker desmoplakin interconnects intermediate filaments with desmosomal adhesion complexes, antibodies immunoreactive with this protein are useful in the localization of the plasma membranes of epidermal cells interlinking with other epidermal cells. The restriction of desmoplakin staining to the non-basal SEL cell membranes is consistent with its absence from hemidesmosomes linking these cells to the basement membrane. We are currently using combinations of these lectins and immunoreagents to investigate K14 and desmoplakin localization during laminitis pathogenesis.

5. Conclusion
Fluorescently-conjugated lectins and antibodies are useful for the identification of tissue, cellular, and subcellular microanatomy in equine hoof lamellar tissue. Structures that can be specifically labelled include connective tissue extracellular matrix, epidermal basal cells, and subcellular components of epidermal cells such as cell membranes, cytoskeletal intermediate filaments, and cytolinkers that join the cytoskeleton to adhesion complexes. Ongoing studies in our laboratory are using these markers to investigate laminitis histopathology and molecular pathogenesis.

Acknowledgments
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Conflict of Interest
The Authors declare no conflicts of interest. The manuscript was not reviewed by any financial sponsors prior to submission.
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Neg 50, Richard Allen/Thermo Scientific, Kalamazoo, MI 49008.

Alexa Fluor®, Life Technologies, Grand Island, NY 14072.

Anti-Cytokeratin 14 antibody (LL002) (ab7800) and Human Desmoplakin I + II antibody (ab71690), Abcam, Cambridge, MA 02139.

DAPI Nucleic Acid Stain (D3571), Life Technologies, Grand Island, NY 14072.

VectaShield™, Vector Laboratories, Burlingame, CA 94010.

Model DM5000B, Leica, Bannockburn, IL 60015.

Model Leica DMI6000 B with Leitz TCS SP5 confocal system; Leitz Microsystems CMS GmbH, Mannheim, DE.

LAS (Leica Application Suite) Advance Fluorescence System software for confocal imaging, Leica, Bannockburn, IL 60015.

Exi Blue, QImaging, Surrey, BC, V3S 6K3, Canada.

Image-Pro Plus, v.7.0, MediaCybernetics, Rockville, MD 20850.

Lectin Kit Rhodamine (RLK-2200), Vector Laboratories, Burlingame, CA 94010.
Safety of Tiludronate Disodium in the Management of Lameness Associated With Equine Navicular Syndrome

Valentine S. Williams, DVM, MS, DACVS

Intravenous infusion of tiludronate disodium used as directed is safe for the control of lameness associated with navicular syndrome. Author’s address: Ceva Animal Health, LLC, 8735 Rosehill Rd., Suite 300, Lenexa, KS 66215; e-mail: valentine.williams@ceva.com. © 2014 AAEP.

1. Introduction
The safety studies characterized the drug’s safety profile.

2. Materials and Methods
Safety was assessed in a target animal safety (TAS) study at 1× (1 mg/kg), 3×, and 5×, a renal safety study (1× and 3×), a hyperkalemic periodic paralysis (HYPP) carrier study (1×), an infusion rate study (1×), and two field studies (1×). Infusion times varied from 30 to 120 minutes.

3. Results
The majority of horses were treated with no adverse effects. Forty-four percent (44%) of horses infused over 90 minutes exhibited signs consistent with colic. The majority of horses resolved with hand-walking only or use of non-NSAID treatment. All cases resolved uneventfully.

In the TAS study, there were mild decreases in ionized calcium and mild elevations in blood urea nitrogen (BUN) and/or creatinine. In the renal study, horses had normal renal function and no evidence of toxicity on histopathology. Horses that developed azotemia in the field studies resolved with fluid therapy.

In the HYPP study, treatment did not induce HYPP episodes in carrier horses. All cases resolved without medical intervention.

4. Discussion
Tiludronate sodium (1 mg/kg IV infusion) offers a safe treatment for the management of navicular syndrome.

Acknowledgments

Conflict of Interest
This study was funded by Ceva Santé Animale. The author is employed by Ceva Animal Health, LLC, the U.S. subsidiary of the sponsor.

Footnote
aTildren, Ceva Sante Animale, 33500 Libourne, France.
Tiludronate Disodium in the Management of Lameness Associated With Equine Navicular Syndrome

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Intravenous tiludronate disodium significantly improved lameness associated with navicular syndrome. Authors’ addresses: Ceva Animal Health, LLC, 8735 Rosehill Rd., Suite 300, Lenexa, KS 66215 (Williams); and Virginia Equine Imaging, 2716 Landmark School Road, The Plains, VA 20198 (Allen); e-mail: valentine.williams@ceva.com. *Corresponding author; †Presenting author. © 2014 AAEP.

1. Introduction
Navicular syndrome is a common cause of lameness and disability in performance horses. The objective of this study was to examine the effectiveness of the bisphosphonate tiludronate disodiuma plus corrective shoeing on lameness associated with navicular syndrome in a blinded randomized placebo controlled clinical trial.

2. Materials and Methods
Cases were selected based on lameness examination (AAEP lameness score of 2 or 3), palmar digital nerve blocks, radiographic findings, and magnetic resonance imaging findings (navicular bone edema with minimal soft tissue lesions). Suitable cases were randomly assigned to a single treatment with tiludronate disodium (1.0 mg/kg) or control (250 mg mannitol) in 1 liter of 0.9% saline administered by intravenous infusion over 30 or 60 minutes. All horses were subjected to corrective shoeing. Effectiveness was defined as an improvement of ≥1 grade lameness score in the lamest limb with no worsening of lameness in the opposite limb at 2 months post treatment.

3. Results
Located at 12 sites, 181 cases (119 treated group and 62 control group) complied with all requirements for inclusion in data analysis. Tiludronate disodium was significantly more effective compared to the control group in reducing lameness score (64% vs 48%, respectively).

4. Discussion
Tiludronate disodium in conjunction with corrective shoeing effectively reduced lameness associated with navicular syndrome.

Acknowledgments
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Conflict of Interest
This study was funded by Ceva Santé Animale. Dr. Williams is employed by Ceva Animal Health, LLC, the U.S. subsidiary of the sponsor.

Footnote
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Comparison of Subjective and Objective Methods to Identify Mild Forelimb Lameness in Horses

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1. Introduction
The goal of this study was to compare subjective and objective lameness detection methods to identify the presence of mild forelimb lameness using an established model of osteoarthritis.

2. Materials and Methods
A unilateral carpal osteochondral fragment (OCF) was created in 16 horses. Force platforms, inertial-sensors, and subjective evaluation were used to detect forelimb lameness at 4 time points. Agreement was measured for identification of the limb with an OCF using each individual method and for identification of the same limb identified as lame between methods. Pearson correlations were calculated between all output parameters.

3. Results
Fifteen days post OCF agreement was 87% for subjective evaluation, 63% for force platforms, and 50% for inertial-sensors identifying the OCF limb. Average agreement over all time points for identifying the same forelimb as lame was 53% between subjective evaluation and inertial-sensors, 45% between inertial-sensors and force platforms, and 33% between subjective evaluation and force platforms.

4. Discussion
The OCF limb was more reliably identified by subjective evaluation at day 15. It should be noted that the evaluator was aware of study day and design introducing potential bias. The agreement between the subjective evaluation and inertial-sensors may be due to the increased number of strides evaluated by each method as compared to the force platforms.

Acknowledgments
Conflict of Interest
The Authors declare no conflicts of interest.
Clinical Hindlimb Lameness is Frequently Associated With Significant Compensatory Forelimb Lameness

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Compensatory ipsilateral forelimb lameness commonly occurs with hindlimb lameness and must be ruled out as a cause of forelimb lameness before blocking. Observing the effect of hindlimb blocking on forelimb movement can assist in determining the response. Authors’ address: Weipers Centre Equine Hospital, University of Glasgow, Bearsden Road, Glasgow G61 1QH, U.K. e-mail: sylvia.maliye@hotmail.com. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction
Compensatory forelimb lameness associated with hindlimb lameness has not been fully characterized in a significant number of clinical cases. Therefore, we aimed to describe and quantify the compensatory effects of naturally occurring hindlimb lameness.

2. Materials and Methods
Data from lameness investigations using an inertial sensor-based system with positive response to hindlimb diagnostic anesthesia were reviewed. Horses were grouped into (1) clinical hindlimb lameness only (HL, n = 16, 60%), (2) HL and ipsilateral forelimb lameness (IFL, n = 9, 33%), or (3) HL and contralateral forelimb lameness (CFL, n = 2, 7%). The change in measures of head (HDMax, HDMin, HMA) and pelvic (PDMax, PDMin, PDA) movement following diagnostic anesthesia was determined. The data were analyzed to determine the effect of abolishing hindlimb lameness using a paired t-test or signed rank test as appropriate. Statistical significance was set at $P < 0.05$.

3. Results
In the HL and IFL groups, the abolition of HL significantly decreased ipsilateral forelimb ($-38\%, -41\%$) and increased contralateral forelimb movement asymmetry ($+36\%, +131\%$). A significant decrease in the difference in minimum head height (HDMin) occurred in both groups ($-69\%, -71\%$); a significant decrease in maximum head height (HDMax) occurred in the IFL group only ($-69\%$).

4. Discussion
We identified a common and significant effect of hindlimb lameness on forelimb and head movement in horses with and without clinical forelimb lameness.

Acknowledgments
Conflict of Interest
The Authors declare no conflicts of interest.
Review of Mistakes That Can Be Made When Interpreting the Results of Diagnostic Analgesia During a Lameness Examination

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Results of diagnostic analgesia can be misinterpreted during lameness examinations. Many diagnostic tests have been known to produce both false positive and false negative results. Authors’ addresses: Department of Large Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, TN 37996 (Jim Schumacher); Department of Clinical Sciences, College of Veterinary Medicine, Auburn University, AL 36849 (John Schumacher); Equine Clinic, National Veterinary School of Lyon, Marcy L’Etoile, France (Schramme); and Department of Clinical Sciences, College of Veterinary Medicine, Texas A&M University, College Station, TX 77845 (Moyer); email: schumjo@auburn.edu. *Corresponding author; †Presenting author. © 2014 AAEP.

1. Introduction

Many diagnostic tests used during the clinical examination of a lame horse are known to produce both false positive and false negative results (e.g., application of the hoof tester, the distal interphalangeal extension test, flexion tests, and pressure applied to the proximal aspect of the suspensory ligament). Diagnostic intrasynovial and regional analgesia is, therefore, an important component of lameness examination if the site of pain causing lameness remains uncertain after the horse undergoes a thorough clinical examination. The results of diagnostic analgesia are usually subjectively evaluated, sometimes with difficulty, and errors in the subjective evaluation lead to errors in diagnosis and, therefore, in prognosis and treatment. To accurately evaluate the results of diagnostic analgesia, the clinician should have a thorough knowledge of the nuances of interpretation. Although interpreting the results of diagnostic analgesia is usually straightforward, the clinician should be aware of the many ways in which diagnostic analgesia can be misinterpreted.

The results of diagnostic analgesia can be misinterpreted if:

- The horse is inconsistently or insufficiently lame.
- Lameness improves or resolves as a result of exercise (i.e., warms out of lameness) rather than from diagnostic analgesia.
- The incorrect limb was chosen for evaluation.
- The clinician is biased by the expected or desired result of an analgesic technique.
LAMENESS EXAMINATION AND THERAPY

- The horse’s gait has been altered by the use of sedatives.
- Testing of regional desensitization after diagnostic analgesia is inaccurate.
- Administration of anesthetic solution is inaccurate.
- Local anesthetic solution has migrated proximally following perineural injection.
- Anesthetic solution leaks or diffuses from a synovial structure to desensitize an adjacent nerve.
- Time of gait assessment after an analgesic technique is inappropriate.
- The clinician does not understand what structures are desensitized by the diagnostic block.
- Disease of the subchondral bone contributes to joint pain.
- The horse has aberrant nerves.
- Long-term pain causes the horse to develop an abnormal protective gait that is unchanged by diagnostic analgesia.
- An abnormal gait is caused by mechanical restrictions rather than pain.
- Lameness is caused by severe pain that cannot be significantly ameliorated by diagnostic analgesic techniques.

2. Effect of Subtle or Inconsistent Lameness on Interpretation

For subjective evaluation of a horse’s gait, the horse should be consistently and sufficiently visibly lame before diagnostic analgesia is performed, so that any improvement in gait can be detected and attributed to the diagnostic analgesic injection. The clinician should be aware that lameness of some horses improves or resolves during exercise and, so, for these horses, a false positive response to diagnostic analgesia can occur if the horse has not been sufficiently exercised to attain a consistent state of lameness.

Often, the lameness may only be improved, rather than completely abolished, after administering diagnostic analgesia. Many clinicians consider a positive response to regional analgesia to be 70% or more improvement in gait and a positive response to intrasynovial analgesia to be 50% or more (authors’ opinion). If the lameness is subtle, a 50% or even a 70% improvement in gait may be difficult to appreciate. Keegan et al. demonstrated, in a study comparing objective and subjective evaluations of mildly lame horses, that agreement among experienced equine clinicians on subjectively determined lameness scores is poor.

Methods by which a subtle lameness can be made more apparent include exercising the horse before the examination (e.g., by riding or lunging), changing the ground underfoot (e.g., from soft to hard ground or vice versa) and evaluating the horse’s gait while the horse trots in a circle. Pain in the digit can usually be exacerbated by exercising the horse on a hard surface and pain more proximal in the limb can sometimes be exacerbated by exercising the horse on a soft surface. In some cases, a lameness is subtle because the horse is bilaterally lame. When a nerve or joint block causes a temporary resolution of pain in one limb, lameness in the other limb may become obvious. When lameness is too subtle for consistent subjective evaluation, thereby prohibiting the use of diagnostic analgesia, the use of an automated lameness detection device, such as one that uses body-mounted, wireless, inertial sensors, may still enable measurement of mild but consistent gait asymmetry to allow the clinician to proceed with the use of diagnostic analgesia.

3. Evaluating the Incorrect Limb

The results of diagnostic analgesia can be misinterpreted if the incorrect limb is chosen for evaluation. Lameness of a single limb can cause alterations in gait symmetry that make the horse appear lame on other limbs, causing the wrong limb to be chosen for evaluation. Uneven loading in a fore limb can cause compensatory uneven loading in the hind limbs and vice versa. Keegan cited the “law of sides” to aid in deciding which limb is the truly lame limb. According to this “law of sides,” when a horse appears to be lame in the ipsilateral fore and hind limbs, the site of pain causing lameness is likely to be located in the hind limb. The ipsilateral fore limb appears lame because of a compensatory change in loading in the fore limbs. When a horse appears to be lame in a fore limb and the hind limb of the contralateral side, the site of pain causing lameness is most often in the fore limb. The hind limb appears lame because of a compensatory change in loading or push-off in the hind limbs. The second part of the “law of sides” is less frequently applicable because only marked fore limb lameness tends to cause a visibly noticeable compensatory decrease in impact loading in the ipsilateral hind limb and a compensatory increase in push-off in the contralateral (i.e., diagonal) hind limb. A mild lameness in a hind limb, however, may cause a change in gait that mimics a substantial lameness of a fore limb, whereas lameness of a fore limb is less likely to cause a visibly noticeable change in gait of a hind limb.

4. Clinician Bias Towards the Expected or Desired Result of an Analgesic Technique

One clinical trial showed that clinicians are biased by knowing that a diagnostic block has been performed on a lame horse. This bias may reveal itself in a positive or a negative way and a clinician may be inclined to see an improvement in a horse’s lameness where there is none or fail to see the improvement. The former situation may occur when external circumstances, such as intractable behavior by the horse, time of day, and owner pressure, complicate continuation of the lameness examination with diagnostic analgesic techniques and either situation may arise when a clinician has a strong expectation of the site of pain causing the lameness
based on the findings of the clinical examination. The use of an automated lameness detection device, such as one that uses body-mounted wireless inertial sensors, removes subjective bias from assessment of the horse’s gait after diagnostic blocks have been performed.

5. Gait Alteration by Sedation or Tranquilization

Applying a lip twitch or lip chain to the horse usually provides sufficient restraint to allow diagnostic analgesia to be safely administered, but some horses must be sedated or tranquilized to administer diagnostic analgesia, either because the owner resents having a twitch or lip chain applied to his or her horse or because diagnostic analgesia cannot be safely administered even after a twitch has been applied. The degree to which sedation may interfere with assessment of gait may depend on the severity of lameness and the skill of the clinician performing the examination. The effect of sedation or tranquilization on gait should be observed before the effect of diagnostic analgesia on gait is evaluated. A change in gait of a sedated horse can be erroneously attributed to the effects of diagnostic analgesia rather than to the effects of sedation if the gait was not evaluated after the horse was sedated but before diagnostic analgesia was administered.

Xylazine (0.2 mg/kg, IV), detomidine (10 μg/kg, IV), or acepromazine (0.01–0.02 mg/kg) administered in the course of a lameness examination usually does not substantially interfere with assessment of gait10–13 and lameness may even become more apparent after a horse is tranquilized.14 A tranquilizer, such as acepromazine, has no analgesic effect, whereas a sedative, such as xylazine or detomidine, provides some analgesia.15 Although a sedative may provide more restraint for administering diagnostic analgesia than does a tranquilizer, we believe that a tranquilizer is less likely to adversely affect a lameness examination.

If the clinician is concerned that the effects of sedation may confound the lameness evaluation, the horse can be examined after the effects of sedation dissipate, provided that analgesia imparted by the local anesthetic solution persists longer than the effects of the sedative. Some clinicians have advocated using bupivacaine hydrochloride when administering diagnostic analgesia to intractable horses after sedating the horse with xylazine so that regional analgesia remains in effect when sedation has completely dissipated.16 Bupivacaine, however, appears to be chondrotoxic and its use in diseased joints may be harmful.17 Waiting until the effects of the sedative dissipate may confuse the interpretation of the results of regional analgesia because the local anesthetic solution may diffuse with time from the site of injection, desensitizing unintended structures.3,18–22

After diagnostic analgesia is administered, the effects of an α2-agonist, such as xylazine or detomidine, can be diminished by administering an adrenergic blocking agent such as yohimbine or tolazoline. Yohimbine, however, is not approved for use in the horse and administration of either drug to reverse the sedative effects of an α2 agonist has been associated with adverse reactions in horses, including death.23

6. Inaccurate Testing of Regional Desensitization After Diagnostic Analgesia

The efficacy of a regional nerve block can be tested by observing the horse’s response to stimulation of skin in the area meant to be desensitized by the block. Testing efficacy by testing for lack of skin sensation is not infallible, however, because a lack of reaction to stimulation verifies only that the skin has been desensitized. When lameness is still present, distinguishing between effective and ineffective desensitization of the section of the limb supplied by the innervation targeted by the block is not always easy.

Pain sensation, skin sensation, and deep sensation are supplied by different afferent fibers, each of which may require a different amount of time to become fully anesthetized by the local anesthetic solution, depending on the degree of myelination of individual fibers.24,25 A local anesthetic injection can, therefore, remove pain without totally desensitizing the skin. For the same reason, there is frequently a time-lapse between removal of superficial sensation (e.g., skin sensation at the coronary band) and blockade of sensitivity to deep pressure (e.g., sensitivity to application of hoof testers or sensitivity to a flexion test) by the local anesthetic solution.25 The clinician cannot be certain, therefore, that after administering regional analgesia, deeper structures have been fully desensitized, even though skin sensation has been abolished.

Local anesthetic solution deposited around a nerve diffuses from the periphery of the nerve towards the center of the nerve.25 Sensation to the most distal portion of the limb is supplied by the core fibers of the main nerve trunk and, therefore, desensitization proceeds from the site of deposition of local anesthetic solution distally.25 After administering a tibial and peroneal (fibular) nerve block, for example, the time required for analgesia of the distal hock joints should be less than the time required for loss of skin sensation at the bulbs of the heel.

After perineural injection of a local anesthetic solution, sensations disappear in the following order: pain, cold, warmth, light touch, joint proprioception, and deep pressure.25 Re-examining a horse too soon after a nerve block, before all sensations have been abolished, may lead to erroneous interpretation of results of the nerve block. A clinician may, after accurately administering a nerve block that would effectively eliminate pain causing lameness, re-administer the block without first trotting the horse if skin sensation is still present. By not trotting the horse, the clinician fails to realize that the first administered block was effective in ameliorat-
ing lameness. As a consequence of increased volume of local anesthetic solution and increased time before the horse’s gait is evaluated, the response to diagnostic analgesia may no longer be specific.

If skin sensation is no longer present after a diagnostic nerve block (e.g., a peroneal digital nerve block) and the horse, when trotted, still appears to be lame, the clinician may come to the inaccurate conclusion that the cause of lameness is not located in the area that should have been desensitized by the nerve block because sensitivity to deep pressure in the foot has been not yet been abolished. Consequently, when the horse’s lameness is significantly improved by a more proximally administered nerve block in the diagnostic sequence, the clinician may conclude erroneously that the cause of lameness is located between the apparently ineffective block (e.g., a palmar digital nerve block) and the apparently effective block (e.g., an abaxial sesamoid nerve block) rather than in the area that should have been desensitized by the first nerve block (e.g., a palmar digital nerve block) because sensitivity to deep pain in that area has been abolished after a longer period of time.

Judging the effectiveness of a regional nerve block performed on the proximal aspect of the limb (i.e., anesthesia of the median, ulnar, deep peroneal (fibular), or tibial nerve) is often difficult. After a regional nerve block, skin can be tested for loss of sensation at a specific site on the limb for each nerve, but this method of testing may yield erroneous information for several reasons: (1) the horse may be stoic and show little reaction to noxious stimulation of skin, (2) the region of skin desensitized may vary somewhat among horses, and (3) some horses react progressively more violently to the slightest provocation, making a positive reaction to skin testing difficult to interpret. In particular, the efficacy of a tibial nerve block cannot be reliably tested, especially if the distal portion of the limb has been desensitized by a low 4-point or 6-point nerve block; loss of skin sensation after a peroneal nerve block is extremely variable.

A perineural injection of local anesthetic solution in the distal portion of the limb aims to result in deposition of injectate within the neurovascular bundle, thereby establishing direct contact between the local anesthetic solution and the nerve. Perineural injection to anesthetize a nerve may result in deposition of the local anesthetic solution superficial to the perineural fascia, thereby preventing the local anesthetic solution from rapidly contacting the nerve. As a consequence, therefore, the time between injection and diffusion of the local anesthetic solution through the perineural fascia to contact and anesthetize the nerve is likely to be prolonged. The time required for diffusion of local anesthetic through the perineural fascia remains unknown.

Directly instilling local anesthetic solution into a joint, bursa, or tendon sheath leaves little or no doubt that intrasynovial structures are desensitized and is an advantage of intrasynovial analgesia over regional analgesia. The clinician cannot always be certain, however, that the local anesthetic solution was instilled within the intended synovial structure. The most accurate technique to assure accurate synoviocentesis is the use of diagnostic imaging. Radiography can be used to ascertain accurate positioning of the needle (e.g., for navicular bursectesis) or accurate intrasynovial instillation of a mixture of local anesthetic solution and contrast medium (Fig. 1). Ultrasonography can be used to monitor needle placement and instillation of local anesthetic solution into the synovial space in real-time.

Successful synoviocentesis can be assumed when synovial fluid is observed exiting the needle, but often synovial fluid cannot be aspirated even though the needle is correctly inserted within a synovial structure. The volume of synovial fluid in some synovial structures, such as the distal joints of the tarsus and the navicular bursa, may be so small that fluid cannot be aspirated. Or the clinician may fail to aspirate fluid if the opening of the needle lies against synovial villi or cartilage.

In some cases, the degree of resistance to pressure on the plunger of the syringe during injection may be the only means of determining the likelihood of having entered a synovial structure. If the needle was correctly placed, resistance to injection is slight, but for some synovial structures, such as the scapulohumeral joint or the synovial compartments of the stifl, lack of resistance to pressure on the plunger of the syringe is not a reliable indication that the joint has been entered. The ability to aspirate a portion of the injectate is a good indication of accurate synoviocentesis for some synovial structures such as the coxofemoral, scapulohumeral, and cubital joints, and the bicipital bursa. When a periaricular injection of local anesthetic solution may affect motor nerves (e.g., a periaricular injection of the cubital, scapulohumeral, or coxofemoral joints), the authors inject isotonic saline solution, before instilling the
local anesthetic, to allow aspiration to verify placement of the needle within the synovial structure, rather than risk peri-articular injection of local anesthetic solution.

Another cause of misinterpretation of the results of regional analgesia due to inaccurate administration is inadvertent instillation of the local anesthetic solution into an adjacent blood vessel or synovial structure. This complication occurs most commonly when performing regional analgesia in the distal portion of the limb. For example, when anesthetizing the palmar nerves at the level of the distal aspect of the second and fourth metacarpal bones, as part of the low 4-point nerve block, the digital flexor tendon sheath is often inadvertently penetrated because, at this site, there is no connective tissue interposed between the neurovascular bundle and the wall of the tendon sheath. The metacarpophalangeal joint is also occasionally inadvertently and unknowingly entered when blocking the palmar metacarpal nerves as part of the low 4-point nerve block. Techniques to anesthetize the proximal aspect of the palmar metacarpal nerves are associated with a risk of inadvertent injection of either the carpometacarpal joint or the carpal sheath, potentially leading to a false negative or false positive result. Local anesthetic solution administered subtarsally to anesthetize the plantar metatarsal nerves can, on occasion, be deposited into the tarsometatarsal joint, the tarsal sheath, or a blood or lymphatic vessel to produce a false negative or a false positive result. Besides possibly producing false negative or positive results, inadvertent administration of local anesthetic solution into a synovial structure while performing regional analgesia may result in synovial infection of that structure because skin preparation for perineural injection is generally less thorough than is skin preparation for synoviocentesis. Administering the local anesthetic solution as the needle is withdrawn decreases the likelihood of depositing the entire amount of solution in an unintended structure and results in deposition of the solution in more than one tissue plane, which increases the likelihood of the solution contacting the nerve.

When using the lateral approach to ameliorate pain in the distal interphalangeal joint, the clinician should be aware that the navicular bursa or digital flexor tendon sheath can be entered inadvertently. When inexperienced veterinary students attempted to inject the proximal interphalangeal joint of horses, 64% of injections were inadvertently placed into the digital flexor tendon sheath using the palmaroproximal approach (Fig. 2). When assessing the effects of anesthesia of nerves in the distal portion of the limb, the clinician should be aware that, after injection, anesthetic solution might migrate proximally along the neurovascular bundle to anesthetize more proximal structures, thus confusing the results of the examination. For example, anesthetizing palmar digital nerves at the level of the cartilages of the foot may desensitize the pastern and fetlock joints of some horses, at least partially, and anesthetizing the palmar digital nerves at the level of the proximal sesamoid bones (i.e., an abaxial sesamoid nerve block) may completely desensitize the fetlock joint. Perineural analgesia of the deep branch of the lateral plantar nerve at the subtarsal level may desensitize the distal tarsal joints and other structures of the tarsus. To help avoid unintended anesthesia of more proximal nerve branches when anesthetizing nerves in the distal portion of the limb, the needle should be directed distally during insertion. The degree of proximal extension of local anesthetic solution is likely related to the volume of local anesthetic solution administered. A minimal amount of local anesthetic solution should be administered to anesthetize a nerve when performing diagnostic analgesia of the distal portion of the limb to minimize the flow of local anesthetic solution proximally along the
neurovascular bundle (Fig. 3). A very small volume of local anesthetic solution can be used when a nerve can be subcutaneously palpated because the solution can be placed more accurately. To the authors’ knowledge, there are no studies to suggest the smallest volume of local anesthetic solution necessary to anesthetize a particular nerve. A volume of as little as 1 mL has been recommended for anesthesia of the palmar digital nerve administered at the level of the proximal sesamoid bone (i.e., an abaxial sesamoid nerve block).1

The likelihood of desensitizing more proximal structures increases as the time after injection increases. Nagy et al.18 showed that substantial proximal migration of positive radiocontrast solution occurred within 10 min after perineural injection of the palmar nerves, yet they pointed out that a period of 10 min is often required for desensitization of the foot during clinical examination.

9. Leakage or Diffusion of Local Anesthetic Solution From a Synovial Cavity Can Desensitize Adjacent Nerves Leading to Misinterpretation

The results of intrasynovial analgesia can be misinterpreted if anesthetic solution leaks or diffuses from a synovial structure to desensitize a nerve. The most widely recognized example of this situation is the desensitization of nearly the entire foot after local anesthetic solution is injected into the distal interphalangeal joint.46 Intrasynovial analgesia of the distal interphalangeal joint desensitizes the toe region of the sole47 and the podotrochlear apparatus.48,49 The coronary band may also be desensitized after intrasynovial analgesia of the distal interphalangeal joint. Diffusion of local anesthetic solution from the distal interphalangeal joint to the palmar digital nerves, which lie in close proximity to the palmar pouches of the distal interphalangeal joint, is likely responsible for desensitization of these structures. The palmar digital nerves also lie in close proximity to the navicular bursa and, so, intrasynovial analgesia of the navicular bursa also desensitizes the toe region of the sole.50

Another example is the potential desensitization of suspensory branch injuries and proximal lesions.
of the distal sesamoidean ligaments by intra-articular analgesia of the metacarpophalangeal or metatarsophalangeal joint. Recent imaging studies using magnetic resonance imaging have identified these ligamentous injuries as the cause of lameness in horses with significant improvement or elimination of lameness after intra-articular analgesia.

Leakage of local anesthetic solution at the injection site or its diffusion from the digital flexor tendon sheath may result in desensitization of the skin at the heel region of the foot by anesthetizing the palmar digital nerves of some horses receiving intrasynovial analgesia of the digital flexor tendon sheath and may, perhaps, result in desensitization of the distal portion of the limb. The effect of leakage of local anesthetic solution from the digital flexor tendon sheath on the palmar digital nerves varies, however, according to the site of synoviocele. One study found that intrasynovial analgesia of the digital flexor tendon sheath using the palmar axial sesamoidean approach did not ameliorate lameness caused by pain from the sole, navicular bursa, or distal interphalangeal joint, indicating that the palmar digital nerves were not anesthetized by direct analgesia of the digital flexor tendon sheath performed at this site. Nevertheless, to avoid misinterpreting the results, skin sensitivity at the heel bulbs should always be tested after intrasynovial analgesia of the digital flexor tendon sheath to determine if leakage of local anesthetic solution at the injection site has caused inadvertent anesthesia of one or both palmar digital nerves.

Intrasynovial injection of a large volume of anesthetic solution or repeated punctures of a synovial membrane should logically result in leakage of local anesthetic solution. Even when a small volume of local anesthetic solution is injected into the scapulohumeral joint after numerous attempts at centesis, leakage of that solution through the multiple punctures of that joint can temporarily paralyze the sural spinae and infraspinatus muscles by anesthetizing the suprascapular nerve or part of the brachial plexus. When a large volume of local anesthetic solution (i.e., \( \geq 10 \text{ mL} \)) is injected into the tarsometatarsal joint, the joint capsule may rupture, causing leakage of local anesthetic solution, which may desensitize the insertions of the tibialis cranialis and peroneus tertius muscles and the tarsal sheath and anesthetize the medial and lateral dorsal metatarsal nerves or plantar metatarsal nerves. Presumably, overdistention of other synovial structures with local anesthetic solution could also cause leakage of solution and inadvertent analgesia of peripheral nerves in close vicinity to the site of injection or synovial rupture.

10. Time of Assessment After an Analgesic Technique is Inappropriate

The results of diagnostic analgesia can also be misinterpreted if the horse's gait is assessed before the onset of relief of pain. Relief of pain and resolution of lameness after local anesthetic solution is administered near a nerve in the distal portion of the limb usually occurs within 5 min, but anesthetia of larger nerves in the proximal portion of the limb may take 20 to 40 min. In a study concerning the extent of proximal migration after perineural injection of positive radiocontrast solution along the palmar nerves at the base of the proximal sesamoid bones, 11% of injections appeared to be outside the neurovascular bundle. The amount of time required for local anesthetic solution to anesthetize a nerve when the anesthetic solution is deposited outside the perineural fascia is not known, but presumably, more time would be required for anesthesia of that nerve.

Some clinicians prefer to confine a horse after a nerve block because they fear that walking the horse may increase diffusion of local anesthetic solution causing desensitization of more proximal structures, thereby complicating interpretation of the nerve block. Walking, however, does not seem to influence the extent of proximal or distal migration of local anesthetic solution after perineural injection.

The results of intrasynovial analgesia can be misinterpreted if the expectation for timing of onset of analgesia is incorrect. Lameness has been observed to resolve within 5 min after injecting mepivacaine into a painful intercarpal joint and, therefore, administering local anesthetic solution into a synovial structure of the distal portion of the limb also probably results in synovial analgesia within 5 min. Onset of analgesia is delayed when local anesthetic solution is administered into a synovial structure larger than those of the distal portion of the limb. For instance, analgesia of the coxofemoral joint may not occur for 30 min after local anesthetic solution is administered into this joint (authors’ observation).

11. The Clinician Does not Understand What Structures are Desensitized by the Diagnostic Block

The misinterpretation of regional analgesia can arise if clinicians do not realize that a diagnostic block may desensitize more than the target region or that the diagnostic block may not completely desensitize the target region. For example, the results of a low palmar nerve block, as part of a low 4-point nerve block, can be misinterpreted if one palmar nerve is anesthetized proximal to the ramus communicans and the other is anesthetized distal to the ramus communicans, because sensory fibers travel in both directions in the ramus communicans to connect the medial and lateral palmar nerves. When administering a low palmar nerve block, both palmar nerves should be anesthetized distal or proximal to the ramus communicans to avoid leaving nondesensitized sensory nerve fibers traveling through this neural connection. Alternatively, local anesthetic solution could also be deposited adjacent to the ramus communicans when anesthetizing the palmar nerves. Because the ramus communi-
cans of the hind limb is difficult to palpate (and does not exist for many horses), ascertaining if the plantar nerves are anesthetized proximal or distal to this communication may not be possible (Fig. 4; authors’ observation).

An advantage of intrasynovial analgesia over regional analgesia is that intrasynovial analgesia can be administered at any level on the limb, if pain in a particular synovial structure is suspected by clinical findings, because intrasynovial analgesia does not preclude the subsequent use of regional analgesia. There are several situations, however, where a positive response to intrasynovial analgesia does not exclusively verify intrasynovial pain as the cause of lameness. For instance, analgesia of the distal interphalangeal joint anesthetizes the palmar digital nerves as they course close to the joint capsule and, therefore, a positive response to analgesia of the distal interphalangeal joint could implicate any structure within the foot as the site of pain causing lameness.46 In some cases, local anesthetic solution injected into the tarsometatarsal joint results in anesthesia of the plantar metatarsal nerves, which lie in close proximity to the plantar outpouchings of this joint (Fig. 5A). Resolution of lameness after a tarsometatarsal joint block may, therefore, be erroneously interpreted as resolving pain within that joint even though pain from the proximal portion of the suspensory ligament is the cause of lameness.46 In some cases, local anesthetic solution injected into the tarsometatarsal joint results in anesthesia of the plantar metatarsal nerves, which lie in close proximity to the plantar outpouchings of this joint (Fig. 5A). In some cases, local anesthetic solution injected into the tarsometatarsal joint results in anesthesia of the plantar metatarsal nerves, which lie in close proximity to the plantar outpouchings of this joint (Fig. 5A). Resolution of lameness after a tarsometatarsal joint block may, therefore, be erroneously interpreted as resolving pain within that joint even though pain from the proximal portion of the suspensory ligament is the cause of lameness.46 In some cases, local anesthetic solution injected into the tarsometatarsal joint results in anesthesia of the plantar metatarsal nerves, which lie in close proximity to the plantar outpouchings of this joint (Fig. 5A). 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solution to these nerves from the carpometacarpal joint may have the same effect in desensitizing the origin of the suspensory ligament.63

Because analgesia of the intercarpal joint or the tarsometatarsal joint may desensitize the proximal portion of the suspensory ligament and improve lameness caused by pain in the proximal aspect of the suspensory ligament and because subcarpal or subtarsal nerve blocks can, on occasion, improve lameness caused by disease of the distal carpal or distal tarsal joints, performing “cross blocking” to determine more accurately which structure is painful may be prudent. For example, if lameness is ameliorated with a subtarsal nerve block, analgesia of the tarsometatarsal joint could be performed after the effects of the subtarsal block have dissipated. If both analgesic techniques ameliorate lameness, the block with the highest percentage of improvement most likely identifies the most important site of pain.51

12. Disease of Subchondral Bone Contributes to Joint Pain

A negative response to intra-articular analgesia does not always exclude joint pain as a cause of lameness. Intrasynovial analgesia may not resolve lameness if disease of the subchondral bone contributes to joint pain because the subchondral bone is innervated by branches of nerves that enter the bone through its nutrient foramen (Fig. 6). Subchondral bone pain of the metatarsophalangeal or metacarpophalangeal joint is a common cause of lameness in young racehorses that is commonly inadequately ameliorated by intra-articular analgesia of these joints.66 Intra-articular analgesia of the antebrachiocarpal joint of horses lame because of a chip fracture of the distal portion of the antebrachium may fail to abolish lameness when the cartilage remains intact, preventing local anesthetic solution from penetrating subchondral bone.67

13. Aberrant Innervation

Aberrant distribution of the appendicular nervous system is claimed to be uncommon25 but Kainer68 described four variations in innervation of the digit

Fig. 6. A, When pain originates in the subchondral bone, local anesthesia of the nerve distal to the level at which the nerve enters the nutrient foramen to supply the subchondral bone (B) does not attenuate lameness, while local anesthesia of the nerve proximal to the nutrient foramen (A) does. C, Direct analgesia of the joint may not substantially attenuate lameness.

Fig. 7. Topography of branches of the principal nerves of the foot varies considerably.69 Common variant branches of major nerves (arrows) as cited by Kainer68 are shown in this illustration.
of the equine forelimb (Fig. 7). He found that 30% of horses possess an intermediate branch originating from the dorsal digital nerve, that 30 to 50% have a continuation of the medial palmar metacarpal nerve to the coronary corium and, that occasionally, an extra palmar branch of the medial palmar nerve, originating in the middle third of the metacarpal region, courses distally into the digital cushion. He also found that, occasionally, an extra branch from the lateral palmar nerve, originating in the proximal metacarpal region, turns dorsally and extends to the coronary band. In a study of the nerves of the equine digit, Sackö6 concluded that the topography of the branches of the principal nerves of the foot varies considerably. Aberrant nerve branches may be responsible for ineffective desensitization of the foot after a palmar digital nerve block, leading to misinterpretation of the block, especially when the skin at the coronary band is no longer sensitive. When aberrant nerve routes are suspected, ring blocks may be a useful adjunct to perineural analgesia.25

14. Development of a Protective Gait

We believe that chronic pain causing lameness (months to years) may cause the horse to acquire a protective stance and/or gait as an accommodation to lessen the pain creating the lameness. Some horses, therefore, may still retain an abnormal gait even after diagnostic analgesia has completely relieved the pain causing the lameness. This abnormal gait may persist for a long time after the horse has been adequately treated for the condition causing lameness.

15. Mechanical Lameness

When an abnormal gait is caused by restrictive tissue rather than by pain, diagnostic analgesia does not identify the site of the lesion. Examples of mechanical lameness include fibrotic myopathy usually involving adhesions between the semitendinosus, semimembranosus, or biceps femoris muscles, but other muscles as well.70 Momentary upward fixation of the patella and fibrosis of various ligaments and tendons may also cause abnormal gait unassociated with pain.

16. Evaluating Horses With Severe Limb Pain

In the experience of the authors, extremely painful disease of the foot (e.g., fracture, laminitis, or sepsis) often cannot be substantially ameliorated by regional analgesic techniques, especially when they are performed close to the lesion causing pain. Analgesic techniques performed more proximally on the limb seem to ameliorate severe foot pain better than palmar/plantar digital or abaxial sesamoid nerve blocks. The site of pain causing lameness might be misconstrued if the influence of severe pain on the results of diagnostic analgesia are not considered (authors’ experience).

Acknowledgments

Conflict of Interest

The Authors declare no conflicts of interest.

References and Footnote


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*Schumacher, unpublished data, 2012.*
Long-Term Outcome of Standing Medial Patellar Ligament Splitting to Manage Horses Exhibiting Delayed Patellar Release: Sixty-Four Horses

Sarah J. James, DVM, DABVP*; Timothy G. Eastman, DVM, MPVM, DACVS; and Justin D. McCormick, MS, DVM

1. Introduction
This study describes a standing technique for medial patellar ligament splitting and reports long-term (average 4.5 years) efficacy in horses exhibiting delayed patellar release.

2. Materials and Methods
The medical records of 64 horses that had a standing medial patellar ligament splitting surgery performed to treat delayed patellar release were retrospectively analyzed. Horses were sedated in standing stocks. A number 15 scalpel blade was used to percutaneously split the medial patellar ligament from just proximal to its insertion on the tibial tuberosity to its attachment on the parapatellar fibrocartilage, with the goal of inducing a localized desmitis and subsequent thickening of the ligament. Aftercare consisted of oral antibiotics, 14 days stall rest with hand walking, light exercise for 14 days, and full work at 4 weeks. Follow-up information was obtained through telephone calls to owners and/or clinical evaluation by a veterinarian.

3. Results
Eighty-nine percent of horses benefitted from the procedure, with complete resolution in 58% of horses and improvement in 31% of horses. Seventy-three percent of horses were able to perform at the desired level of performance following the procedure. Sixty-three percent of horses showed signs of improvement or resolution within 30 to 60 days. Two horses had complications following the procedure: one had an incisional infection and one had a medial patellar ligament rupture.

4. Discussion
This study shows that standing medial patellar ligament splitting is a successful long-term sur-
LAMENESS EXAMINATION AND THERAPY

gical option for treatment of delayed patellar release. The procedure has few complications and allows a rapid return to desired performance.

Acknowledgments

Conflict of Interest

The Authors declare no conflicts of interest.
How to Perform Centesis of the Bicipital Bursa Using a Trans-Tendinous Approach

Robert Cole, DVM; John Schumacher, DVM*; Ray Wilhite, MS, PhD; and Joseph Newton, DVM, PhD

1. Introduction
Lameness caused by disease associated with the bicipital bursa in horses is uncommon,1,2 but centesis of this synovial structure is often performed when attempts at localizing pain to other structures in the limb using regional and intra-articular analgesia have failed to ameliorate lameness or when clinical signs of bicipital bursitis are obvious. Signs of bicipital bursitis include signs of pain when the affected limb is extended, reluctance to advance the limb, and signs of pain when pressure is applied to the tendon of the biceps brachii muscle.1,3,4

Centesis of the bicipital bursa can be performed by inserting a spinal needle near the deltoid tuberosity and advancing the needle proximomedially toward the bursa.2,5–8 For this technique, a 9 cm spinal needle is usually used. The bursa can also be accessed by inserting a 3.8 cm hypodermic needle medial to, or slightly distal to, the palpable edge of the cranial prominence of the greater tubercle of the humerus, in a plane parallel to the bearing surface at about a 45° angle to the longitudinal axis of the horse and advancing the needle until it strikes the cartilage of the intertubercular groove.6,9–11 The intent of these methods is to access the bursa while avoiding placing the needle through the tendon of the biceps brachii muscle. In a previous study,11 we found that clinicians who had no previous experience performing centesis of the bicipital bursa are unlikely to be successful in centesis of the bursa using either approach.

Because other synovial structures are accessed by advancing a needle through a tendon or ligament without apparent deleterious effects to those structures, we reasoned that penetration of the tendon of the biceps brachii muscle approximately 2 cm medial to the cranial prominence of the lateral tuberosity of the humerus would provide easy, consistently accurate access to the bicipital bursa.

2. Materials and Methods
To attempt centesis of the bicipital bursa using a trans-tendinous approach on an unsedated horse, three operators are likely necessary: one to restrain the horse, another to elevate the limb, and one to perform the centesis. The horse is restrained with sedation or by applying a lip twitch. Desensitizing skin at the site of injection is optional. The limb is pulled forward to extend the scapulohumeral joint
and flex the cubital joint. The radius is held horizontally (or slightly lower) to the bearing surface. This maneuver likely lifts the tendon from bone to create a space for the needle tip to enter.

An 18-gauge 9 cm spinal needle is used for centesis of the bicipital bursa. The needle is inserted at a site approximately 2 cm medial to the cranial part of the greater tubercle of the humerus (Fig. 2) until the needle tip strikes cartilage. The tip of the needle enters into the lateral portion of the intertubercular groove if the needle is inserted at an angle approximately parallel to the longitudinal axis of the horse (Fig. 3). Resistance is felt as the needle is advanced through tendon, and then, as the bursa is entered, a sudden lack of resistance can be appreciated. Unless the bursa is inflamed, synovial fluid does not fill the needle hub; synovial fluid, however, can usually be aspirated (Fig. 3C). Solution is easily injected; difficulty injecting solution indicates the needle was inserted inaccurately. The ability to aspirate injected solution is another indication that centesis was accurate. Local anesthetic solution injected inadvertently outside the bursa may temporarily paralyze the extensor muscles of the forearm (Fig. 4).

Accurate injection can be confirmed radiographically if positive iodinated (ionic or nonionic) contrast medium is added to the solution injected, or by using ultrasonographic visualization. Examination of a mediolateral radiographic projection obtained with the horse standing and its limb extended with the radius horizontal to ground provides good visualization of the bursa outlined by contrast medium. Centesis can be considered successful if a cylinder-shaped radiopacity created by the contrast medium is seen cranial to the cranial prominence of the greater tubercle of the humerus and pooled in two sac-like structures several centimeters ventral to the cranial prominence of the greater tubercle (Fig. 5A); the injection is likely outside the bursa if the medium outlines the contour of the cranial border of the tendon (Fig. 5B).

To determine the extent of damage to the tendon caused by a trans-tendinous approach to the bicipital bursa, we inserted an 18-gauge spinal needle through the lateral head of each tendon of the *biceps brachii* muscle of two horses immediately after they were euthanized. The tendons were removed immediately from the cadavers and examined grossly for damage caused by the needle; two of these tendons were also examined histologically.

3. Results

A radiocontrast study that examined the accuracy of a trans-tendinous approach to the bicipital bursa indicated that the approach is highly accurate. In that study, no attempt was made to aspirate synovial fluid. Rather, before radiographic assessment of accuracy of synoviocentesis, accuracy of synoviocentesis was based on appreciation of the texture of the tissues as the needle was advanced, sudden loss of resistance as the needle entered the bursa, and ease of injection. Since that study, we have performed centesis of the bicipital bursa in each shoulder of 12 horses free of evidence of bicipital bursitis. We found, in every instance, that synovial fluid could be aspirated using the trans-tendinous approach.

Minimal damage was found in the tendon of the *biceps brachii* muscles of two horses that underwent a trans-tendinous approach to each bicipital bursa immediately after euthanasia. Evidence of needle-induced damage, other than plaques of hemorrhage on the cranial surface of each tendon, could not be found. Neither the site of needle entry on the cranial surface nor needle exit on the caudal surface could be found during gross examination of three tendons; the probable site at which the needle exited was found on one tendon. A needle tract could not be found in the two tendons examined histologically.

4. Discussion

A similar trans-tendinous approach to the bicipital bursa was suggested by Wyn-Jones who advised...
that the bursa be accessed by inserting a 6.35 to 9 cm (2½ to 3½ in.) needle at the point of the shoulder and directing it to the intertuberal groove. He advised, however, that the shoulder be flexed to tense the bursa to ease penetration. This method may be suitable for aspirating fluid from a fluid-distended bursa, but the authors have found that even with the scapulohumeral joint in a neutral position, injecting fluid into the bursa of horses without disease of the biceps brachii tendon or tendon sheath to be difficult. The authors believe that manipulation of the limb to extend the scapulohumeral joint and flex the cuboidal joint lifts the bicipital tendon from the humerus to create a space for the needle to penetrate.

We selected a 9 cm (3½-inch) spinal needle to perform centesis of the bicipital bursa not only for its length but also because, with its short bevel, its orifice is more likely to reside entirely within the synovial space than is a standard hypodermic needle with its longer bevel. Additionally, spinal needles are particularly flexible and more likely to bend than break and, thus, safer to use if there is a possibility the horse may move the limb. Using a flexible needle is especially important when the difference in the range of movement between skin and deeper tissues is large. We have directed 20-gauge spinal needles through the dense tendon of the biceps brachii muscle, but needles of this gauge tend to bend as they are advanced. It is the authors’ experience that a 3.8 cm (1½-inch) hypodermic needle is too short to penetrate the bicipital tendon where the tendon is the thickest; the authors be-

Fig. 3. A, If the needle is inserted at an angle approximately parallel with the longitudinal axis of the horse, B, its tip will enter into the lateral portion of the intertubercular groove. C, Synovial fluid can usually be aspirated to verify accuracy of synoviocentesis.

Fig. 4. A, Centesis can be considered successful if a cylinder-shaped image created by the radiopaque contrast medium is seen cranial to the cranial prominence of the greater tubercle of the humerus and radiopaque contrast medium is seen pooled in two sac-like structures several centimeters ventral to the cranial prominence of the greater tubercle (30 mL of radiocontrast medium was injected into this bursa); B, if the medium follows the contour of the tendon on its cranial border, the injection is likely outside the bursa.
lieve, however, the bicipital bursa can sometimes be entered with a needle of this length if the needle is directed through a thin portion of the tendon, toward the periphery of the groove.12

Substantial damage to the tendon of the *biceps brachii* muscle seems unlikely, provided that movement of the limb is minimal during centesis. The authors are not aware of reports of damage to a tendon associated with other procedures of synovio-centesis in which the needle is directed through a tendon, such as during centesis of the navicular bursa13 or during centesis of the digital flexor tendon sheath (palmar/plantar axial sesmoidean approach).14

Clinicians should be aware that the bicipital bursae may communicate with the scapulohumeral joint. Direct communication between these two synovial structures has been noted by Sisson,15 Dyson,6 and Cole et al.12 but to the authors’ knowledge, the frequency of this communication, direct or indirect, has not been examined.

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**Conflict of Interest**

The Authors declare no conflicts of interest.

**References**