NC STATE UNIVERSITY College of Veterinary Medicine

Equine Sports Medicine and Rehabilitation Symposium

October 14-15, 2017

NC State College of Veterinary Medicine
1060 William Moore Drive
Raleigh, NC 27607

Saturday, October 14th

Time	Event
8:00 a.m. - 8:30 a.m.	Registration & Breakfast
8:30 a.m. - 12:30 p.m.	Dr. Tracy Turner Topics: *1 - Sport Horse Lameness* *2 - Gizmos & Gadgets, Witchcraft or Wizardry?* *3 - Practical Equine Rehabilitation for the Equine Practitioner*
12:30 p.m. - 1:30 p.m.	Box lunch pickup and transport to Edelweiss Farm: 7012 Wildlife Trail in Raleigh, North Carolina
1:30 p.m. - 2:45 p.m.	Dr. Kirsten Tillotson - *Demonstration and Discussion of Equine Rehabilitation Equipment*
3:00 p.m. - 4:30 p.m.	Dr. Richard Mansmann, Dr. Robert Meyer, and Kurt VomOrde *Examination, radiology and shoeing to maximize equine rehabilitation*
4:30 p.m. - 5:30 p.m.	Transport back to NCSU CVM
5:30 p.m. - 7:00 p.m	Wine & Cheese Reception

Sunday, October 15th

Time	Event
8:00 a.m. - 8:30 a.m.	Registration & Breakfast
8:30 a.m. - 12:30 p.m.	Dr. Andrew van Eps Topics: *1 - Anatomy and pathophysiology of laminitis* *2 - Treatment and prevention of laminitis* *3 - Icing related to rehabilitation*
12:30 p.m. - 1:30 p.m.	Lunch
1:30 p.m. - 2:15 p.m.	Dr. Tracy Turner and Dr. Andrew van Eps *Case Discussions*
2:15 pm - 2:30 pm	Break
2:30 pm - 2:55 pm	Dave Richards - Equicast and EVA Clogs
2:55 pm - 3:30 pm	Dr. Cynthia Mackenzie - Luitpold and BetaVet
3:40 pm - 4:05 pm	Dr. Tillotson - SoftRide and ice boot use
4:05 pm - 4:30 pm	Dr. Schnabel - The future of sports medicine and rehabilitation at NC Stat

Sport Horse Lameness

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Take Home Message: Sport Horse lameness is no different than any other lameness with the exception that they are probably more subtle. The sensitivity of the rider, driver or trainer notices issues much sooner. In fact, these issues may be as simple as perceived loss of speed or other performance factor. The examination is critical and must be systematic and thorough.

Introduction: Lameness is an indication of a structural or functional disorder in one or more limbs that is manifested in progression or in the standing position.[1] Lameness can be classified in several different ways. A **supporting-leg lameness** is seen when the horse is supporting weight on the foot or when the horse lands on the leg. **Swinging-leg lameness** is seen when limb is in motion. **Mixed lameness** is seen when the leg is moving and when it is supporting weight. A **complementary lameness** is seen when pain in a limb causes uneven distribution of weight on another limb or limbs, which can produce lameness or the perception of lameness in a previously sound limb. Lameness can also be graded to help define the severity of the problem. However, lameness of the sport horse often manifests itself not as a noticeable gait anomaly but as a decrease in performance or change in attitude toward performance. Because of this the diagnosis of these problems can be very difficult. The purpose of this presentation is to discuss the systematic steps that the author has found useful in the evaluation of these horses.

Any evaluation begins with a good history. We will discuss the pertinent information and how and why it is helpful. The second stage is physical examination. Discussion will center on process and key points to evaluate. The third component of evaluating the sore performance horse is imaging. Usually in these cases it is difficult to pin point an area to evaluate with imaging. Our discussion will center on determining which imaging modality is most appropriate. We will discuss anatomic imaging versus physiologic imaging and the value of each.

History: An anamnesis (an accurate history) should be obtained in every case.[1] Information obtained from a careful history not only will help determine where lameness is (especially important for subtle lameness) but also may provide you with valuable therapeutic or prognostic information. Unfortunately, you will also find that most horsemen speak a foreign language. Questions that I have found to be useful to ask are: How long has the horse been lame? Does the owner know what caused the lameness? Does the horse warm out of the lameness? Does the horse stumble? What other signs does the horse show? When was the horse shod? For racing horses, what times has the horse ran? How well has the horse run since the onset of the problem

(i.e., is the horse making money)? Finally, what treatments have been done and were they helpful? In my experience, the veterinarian needs to get the owner to talk freely, once that happens the important details will flow; otherwise, the owner/trainer/rider may remain guarded.

There are breed variations of lameness. For the Thoroughbred, what is the horse used for? Racing, hunting. For racing horses, does the horse move toward or away from the rail? This breed is most commonly afflicted with carpitis, carpal fractures, injury to metacarpophalangeal joint, tendon and suspensory ligament injury, and sesamoid injury (racing). Hunters/jumpers look for forelimb feet problems, tarsitis, or back and quadriceps problems. Standardbreds are typically used for racing, so you want to know, is the horse worse in the straights or turns? Does the horse move toward or away from the rail? Is the horse on one line? Is the horse on one shaft? Has there been a recent change in shoeing (Borium, trailers, etc.)? These horses are most commonly afflicted with tarsal and stifle problems. Quarter Horses may be used for any number of things such as racing, gaming, or pleasure. Does the horse point a foot? Is the horse reluctant to take a lead? Has the horse lost suppleness? Has there been recent changes in the horse's attitude toward work? The gaming or working horse is most commonly afflicted with "ringbone", fractures of the phalanges, "bone spavin" and "navicular disease". Ponies and draft breeds one should look for signs of founder (laminitis) and question the owner accordingly; i.e., access to feed and feed changes, etc. Ask if the horse works in a team. Most importantly, I have learned to develop an understanding of what the owner expects. What do they think is wrong? Because no matter what else happens during the examination the practitioner must **show** them what is wrong.

Visual Examination: The next step is to perform a visual examination. It is most important to be systematic, do the exam the same way every time.[1] One will not only be quicker but less likely to miss something. The author is a firm believer in the adage “you will miss more things by not looking than not knowing.” The visual examination begins the second you see the horse. Look for clues to the location of the problem. Look for obvious swellings or enlargements. Observe the horse's posture or how it stands. Constantly pointing or flexing one leg is a good clue that leg is painful. Look for conformational defects that may predispose to lameness. Asymmetry of anatomical parts is often a clue to many problems. Look at hoof size, pectoral or shoulder muscles, or the gluteal muscles.

Following the initial observation one needs to determine if the horse is lame and where. Watch the horse move, coming toward you, going away, and from the side. It is easiest to detect lameness at the trot but you should always observe the horse at a walk. There are some breeds that you may find it difficult to get them to trot. The Tennessee Walking Horse, Standardbred, Paso Fino, and Icelandic have a natural pace and tend to look like they "waddle" when they move. Typical forelimb lameness is characterized by the "head nod". It is caused by the horse moving its head up and down to shift its center of gravity (weight) toward its hindquarters. The head rises when the lame leg hits the ground. Conversely, the head lowers when sound leg hits the ground. These movements are most easily seen when the horse is coming toward you. A typical

hind limb lameness is characterized by the "hip hike". This is caused by the horse shifting its pelvis to accommodate leg movement and weight bearing pain. It is most easily observed with the horse moving away from you. The observer must concentrate on the movement of the tuber coxae. This movement is symmetrical in the normal horse. Lameness is noted when one side moves more than the other. The side that moves the most is the lame side. Reluctance of the horse to flex the joints of the hind leg causes the horse to tilt the affected side of its pelvis up as the lame leg is moved forward. As the horse begins to place weight on the limb, the horse tilts the affected side down to avoid putting full weight on that leg. Observations of the horse from the side is necessary to fully evaluate limb function. The normal horse should land on its hoof either flat or heel first. Landing on the toe usually indicates pain in the caudal hoof. The length of stride should be evaluated and is symmetrical in the normal horse. Shortening usually indicates pain but may also represent a quick step to relieve the other leg, so do not “jump to conclusions.” The arc of the stride should also be symmetrical. A decrease in arc of foot flight usually indicates a reluctance to flex joints in that leg. Some lameness problems can be completely characterized by the changes in stride (these include stringhalt, fibrotic myopathy, upward fixation of the patella).

There are aids to help visualize a lameness such as working the horse on a hard surface such as pavement is probably the best one. Working the horse on a lounge like (i.e., small circles) is also very helpful especially for forelimb problems. Some horse need to be seen under saddle or being driven. Cinematography can be used to see the horse in faster gaits and to be able to slow the motion.

Palpation: Palpation is a crucial part of every examination. It is the systematic evaluation of the limb by touch (i.e., feel for structural changes and pain).[1] The examination needs to look at all portions of the limb but should be gauged so the most time is spent in examining the most common areas of injury (i.e., in the front limb concentrate on structures from the carpus distally and in the hind limb concentrate on the hock and stifle). To begin, the author checks the horse’s teeth and mouth. This is done by palpation by inserting the hand and arm into the mouth. By placing the hand in the space between the tongue and teeth, the tongue can be pushed to the opposite side and the teeth can be digitally palpated to the last molar, this must be done quickly and certainly does not replace a speculum examination but is a quick way to determine if the mouth or teeth may be an issue.

Examination of the foot involves evaluating hoof wall texture; the coronary band should be elastic and fit over the hoof like a cuff.[2] The alar cartilages should be easily palpable and flexible. Examination of the pastern should involve checking for increased digital pulse. The tendons should be assessed as to their relative size, which normally should be about 2/3 the width of the bones. The long and short pastern bones form the pastern joint, there usually is a slightly raised area on the dorsal surface of the pastern that is palpable and is roughly the location of the pastern joint. Palpating proximal to distal the pastern begins to narrow then flares out, this also roughly corresponds to the pastern joint. Cases of ringbone or exostosis usually have either obvious enlargements or distinct enlargements of the normal structures (always check symmetry when in

doubt). Examination of the fetlock starts by looking for distension of the fetlock which is most easily detected by palpation of the volar pouches (between the suspensory ligament and MCIII/MTIII).[1] The presence of a firm, movable nodule underneath the skin and joint capsule of the dorsal fetlock is indicative of a villonodular lesion. A pain response elicited by firm palpation over the sesamoids may indicate a fractured sesamoid or sesamoiditis. Pain elicited over the most proximal sesamoid, where the suspensory ligament inserts is often seen in cases of suspensory desmitis. Examination of the metacarpus/metatarsus regions requires that the bone, tendons and suspensory ligament is all that should be palpated. Careful palpation between MCIII and MCII or MCIV should reveal a shallow groove. Disappearance of this groove is indicative of a "splint". The suspensory ligament and flexor tendons should feel taut, be of uniform thickness, and easily palpated in the normal horse. Squeezing the suspensory ligament will cause a pain response in any horse and should not be mistaken for an indicator of injury. To check the suspensory, one should firmly press the ligament against the caudal aspect of MC/MT III. Any thickenings or areas where the tendons cannot be separated from each other indicate disease. Palpation of the distal tendon sheath often indicates a slight amount of effusion, this is normal. A common mistake is to think the "puffiness" caused by the digital veins is effusion in the tendon sheath. Only 2 of the 3 joints of the carpus are easily palpable, the antebrachiocarpal (formerly radio carpal) and the middle carpal (formerly intercarpal). Distension of these joints can be determined by pressing over the medial or lateral aspect of the joint and feel fluid move to the opposite side. When these joints are flexed the dorsal margins of each of the bones is easily palpated. The muscles and tendons of the antebrachium (forearm) are readily palpable and identifiable. The main thing to check is for swelling. Distension of the elbow is very difficult to determine. The main item to check is the olecranon. Little can be felt over the humerus, except an effort needs to be made to check the surrounding musculature for pain. Palpate the right and left scapula simultaneously to check for atrophy, swelling, etc. Gentle pressure over the bicipital bursa will usually result in a pain response. If the area is truly affected the response is very pronounced. Shoulder distension is difficult to determine. Pressure over the brachiocephelicus muscle followed by a pain response by the horse often indicates that is the lame leg. The reason being the horse begins to use that muscle differently causing soreness.

The examination of the distal rear limb is the same as the forelimb. Examination of the tarsus reveals that only the tibiotarsal joint is easily palpable.[1] The trochlea of the talus should be easily palpable. The saphenous vein on the dorsomedial aspect of the joint can be mistaken for joint distension. Firm palpation over the head of MTII (insertion of the cunean tendon) followed by a pain response by the horse often indicates inflammation of the distal tarsal joints (Churchill's test). The gaskin (tibia region) is simple to examine, palpate for swelling. Stifle distension can be determined by palpating the patellar ligaments. In the normal horse these ligaments are readily palpable but in the presence of stifle distension they become more difficult to feel. In most cases the patella is easily palpable and portions of the trochlea are palpable. While examining the hip region apply firm pressure over the greater trochanter, if this is followed by a pain response it is circumstantial evidence for trochanteric bursitis or gluteal tendon problems. Palpation over the semimembranosus/semitendinosus which reveals either a

hard or tense mass in the muscle is supportive evidence for fibrotic myopathy. Palpation over the quadriceps should reveal a firm tone. Muscle that feels “flabby” and lacks tone can predispose the horse to "loose patella syndrome". Examination should include a rectal examination to assess pelvic symmetry and muscular tone. A walking rectal is helpful to check for crepitus in the pelvis or sacroiliac region. A common mistake when examining the horse's back is to squeeze the horse's back. This will invariably cause a pain response. The proper way to test is to place firm pressure across the musculature. A pain response produced by this test is reliable. Palpate the spinous processes for pain. Then palpate the tuber sacrale for pain or asymmetry.

Manipulation: Manipulative tests or flexion tests are important adjunct to a lameness exam and can often give an important (lead to establish the cause(s) of lameness.[1-3] In many cases the pain caused by manipulation is so severe that the horse will retract the limb. In more subtle cases, the only satisfactory way of evaluating the effect of manipulation is to trot the horse immediately and note any exacerbation of a problem. Distal limb flexion is the same for both forelimbs and hind limbs. It is performed by picking the leg up, keeping the upper leg as straight as possible, grasping the toe of the hoof, and flexing the coffin, pastern and fetlock. Keep firm pressure on the flexion for 30-60 seconds. Attempts by the horse to retract the limb, to get away, or distinct behavior change indicates pain caused by the test. Exacerbation of the lameness by the test is also positive indicator of pain. The fetlock can be flexed alone by grasping around the pastern instead of the toe of the hoof. Normal range of motion for the fetlock is about 90 degrees. Range of motion for the coffin and pastern joints is difficult to assess. Carpal flexion is performed by picking a leg up and completely flexing the knee. Keep firm pressure on flexion for 1 to 2 minutes. Normal range of motion is nearly 180 degrees. One should be able to touch the palmar fetlock to the palmar forearm without difficulty and without causing the horse pain. Elbow flexion is performed by holding the leg at the level of the forearm and flexing the leg to its fullest. Keep flexed for 1 to 2 minutes. Shoulder flexion/extension/abduction is difficult if not impossible to manipulate without manipulating the elbow. Flexion of the shoulder is achieved by grasping the leg at the forearm and pulling the leg back. You should pull hard enough to allow the front hoof to touch the gaskin. Needless to say it is difficult to hold this portion for any length of time. Extension of the shoulder is achieved by holding the leg at the pastern and pulling the leg up as far as possible. Normally, a horse will tolerate this quite well. A positive response usually causes the horse to walk backwards in an attempt to get away from you. Abduction of the shoulder is achieved by holding the leg in flexion, grasping the horse's knee, and rotating it away from the horse. A positive response is seen either by the horse moving away from you (he is trying to get away from pain) or the horse may even try to go down. This is an excellent test for OCD of the shoulder. The spavin test (tarsal flexion) is performed by picking the leg up and completely flexing the hock for 1 to 2 minutes. These manipulations also causes flexion of the distal limb and stifle. A positive response is seen as an exacerbation of lameness. Be careful, this test is definitely not pathognomonic for hock problems. However, hock problems tend to have a very positive response to this test. Several stifle manipulations can be performed. The patellar ligament test is performed by placing firm pressure on the base of the patella and pushing. Normally, slight movement should be encountered.

Crepitus, excessive movement, or "locking" of the stifle are abnormal responses. The "drawer" test is done by placing hands around the horse's tibia and placing your knee behind the point of the horse's hock. Firmly pull back on the tibia. Crepitus or excessive movement is abnormal. Be careful, you could get seriously hurt performing this test. The collateral ligament test is performed by placing hands around the horse's tibia and placing your foot outside the horse's hoof. Firmly pull the tibia toward you. Crepitus is abnormal. Be careful, a lot of horses do not appreciate this test either. Stifle rotation starts by abducting the horse's distal hind leg and grasping the hoof or pastern with one hand. With your other hand grasp the calcaneus and rotate it outward. Normally, you will get very little rotation. Excessive rotation has been associated with ligamentous injuries. Hip manipulation is difficult but by abducting the leg as far as the horse will tolerate can give you some idea of soreness in the hip.

Foot Examination: Foot Examination is extremely important because it is the most common site of lameness.[2] It cannot be performed well without the foot being properly prepared (sole pared and shoes may need to be removed). Hoof testers are used to squeeze between the hoof wall and sole to check for areas of pain. Be systematic, generally I check the sole first and move left to right. I then check the "navicular area": medial sulcus to lateral wall, lateral sulcus to medial wall, central sulcus to toe, and heel to heel. Percussion is performed by tapping the hoof wall with a hammer. If an area of pain is encountered the horse will flinch. It is also an excellent idea to tap on the nail heads if a horse has shoes, this test is to see if any of the nails are causing pain. Wedge tests are performed by standing the horse on a small block of wood.[3] 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 to protect from the blade. The palmar hoof wedge test is performed in a similar fashion except the block is placed under the palmar two-thirds of the frog and forcing 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.

Saddle Fit: Saddle position should be assessed with the horse standing squarely.[4] The rider should be allowed to position the saddle and pads as he or she would normally on the horse. Many riders place the saddle too far forward, which interferes with movement of the scapula, and may tip the balance of weight distribution in the saddle caudally. The pads may not extend the full length of the saddle, resulting in an area of focal pressure under the rider’s seat. After the saddle has been positioned, the examiner should grab the pommel of the saddle and give it a sharp tug down and back. If the saddle moves and "locks in" further back, the saddle was not positioned correctly. On the other hand, if the saddle does not move, it is positioned correctly.

The saddle should next be placed on the horse's back without pads or girth to visually inspect the balance of the saddle, withers clearance at the pommel, evenness of the panel contact along the epaxial muscles, and width of the channel or gullet between the panels at the rear of the saddle.[4] With the saddle correctly positioned in relation to the shoulder, the lowest part of the seat is identified. This point can easily be found by placing a cylindrical object such as a pencil crossway on the seat and the pencil will roll to the lowest point. This point should be centered between the pommel and cantle. Otherwise, balance and weight distribution of the rider is problematic, because it is shifted toward the lower side. Next, placing fingers between the pommel and saddle checks, the clearance between the pommel and the withers should be 2.5 - 3 fingers wide without being cramped. The angle of the points of the saddletree should be checked next. Ideally, the "points" of the saddle (the projections at the front and sides of the saddle tree) should be far enough behind the back edge of the scapula so that the saddle does not interfere with the range of motion of the scapula. The points also determine the ideal width of the saddle, because the angle they form must also conform to the shape of the withers. The angle is determined either by visualizing the points of the saddle and back together or, more objectively, by molding a malleable ruler over the horse's back at the most forward spot where the saddle should sit. This curved angle can then be compared with the gullet of the saddle off the horse. The angles of the back and points should be parallel to within 10°. It is important to note that many horses are asymmetric through the withers and may have different angles or shapes between right and left sides of the withers. This asymmetry can also be appreciated by observing the horse's withers and shoulders when standing a safe distance behind the horse, usually on a stool or mounting block. All of these fit parameters should be assessed again after the girth is fastened and tightened.

With the saddle correctly positioned and the horse standing squarely, a hand should be placed on the center of the seat and light downwards pressure applied.[4] The other hand is used to run the length of the angle of the points and then under the panels of the saddle in a front to back manner. The examiner should feel for points of pressure or gaps in pressure or contact (bridging). Then, the hand on the seat should move forward and backward to check for "rocking" of the saddle. Pressure points, bridging, and rocking are all indicators of saddle fit problems. The examiner should now step back and look at the position of the saddle on the horse's back. The length of the saddle should fit between the withers and last rib. If it extends farther back, the saddle fits poorly. As the examiner walks around the horse and observes the saddle from the back, the gullet clearance can be assessed. The gullet should clear the spine and associated ligaments along the entire length of the saddle. Pressure on the seat should not change this parameter. Another potential source of discomfort is if the gullet width is too narrow and the saddle contacts the spine region, particularly in large horses or those with heavy riders.

Regional analgesia: Local nerve blocks or intra-articular blocks are extremely important.[5] The blocks provide indisputable evidence of the general location of lameness. But they are not 100% due to migration of local anesthetic or insufficient

desensitization due to aberrant nerves; regardless, the results should be assessed in light of the physical examination findings.

Imaging: There are several different methods of imaging the horse. Imaging is of utmost importance because it will provide pathologic and physiologic information necessary to treat the specific condition. Imaging can be divided into anatomic and physiologic imaging methods. Anatomic imaging modalities include radiology, ultrasonography, computer-aided tomography**,** and magnetic resonance imaging. Physiologic imaging modalities include scintigraphy and thermography.

Radiologic techniques are the most commonly used to evaluate the horse for lameness.[6] Utilizing radiography requires multiple projections to evaluate any area. Radiography provides the most information about bone but with digital radiography, one does get a gross evaluation of soft tissues. Essentially the practitioner must attempt to draw conclusions about a three dimensional object utilizing 2 dimensional pictures. Occasionally it becomes necessary to utilize radiographic techniques that provide more information. Contrast radiography is one such technique that provides information amount the articular cartilage and surfaces. It is of particular value in determining whether subchondral cysts communicate with the joint or in delineating a subcutaneous tract. Generally, 5 to 10 ml of contrast injected into the joint is adequate but this depends on the size of the joint or synovial structure. Pathologic diagnoses are usually made by radiography in conjunction with clinical examination. The future of radiography lies in digital radiography. There are 2 types, computed radiography (CR) and digital radiography (DR). CR utilizes a special plate that is read by the computer. The advantage of CR is that there will be fewer retakes, less radiation is necessary, and post processing of the film can eliminate contrast problems. DR utilizes a special plate but the radiation is directly read from the cassette to the computer where an image is produced. It has the same advantages as CR plus it has the advantage of being more rapid.

Ultrasonographic examination can be used to assess any soft tissue in the horse's body. The deeper the tissue that needs to be evaluated the lower wavelength probe needs to be used. The tissues are examined for changes in echogenicity and fiber alignment. Changes in echogenicity and fiber alignment correspond to changes in the tissue.[7] Ultrasonography is most useful in the evaluation of tendons and ligaments but it also can be used to evaluate muscle and cartilage.

Magnetic resonance imaging and computer-aided tomography are both interesting and high detail anatomic imaging tools.[8,9] They provide the best information regarding pathology, but due to their expense, their use is limited to referral centers and universities. But it is the author's opinion that the sport horse practitioner must be adept at reading and interpreting these modalities because they have the most insight into the patient and physical findings and therefore, can make a more informed interpretation of the results of imaging.

Physiologic imaging techniques would be those techniques that provide the evaluator with an image that reflects physiologic processes. Unlike anatomical imaging that reflects structure, these images give insight into metabolism or circulation. Thermography and scintigraphy provide the examiner with the opportunity to examine the entire horse. When combined with a thorough clinical examination, these methods are extremely useful in identifying injuries that may have otherwise gone undetected.

Thermography is the pictorial representation of the surface temperature of an object. It is a non-invasive technique that measures emitted heat. A medical thermogram represents the surface temperatures of skin making thermography useful for the detection of inflammation.[10] This ability to non-invasively assess inflammatory change, makes thermography an ideal imaging tool to aid in the diagnosis of certain lameness conditions in the horse. The circulatory pattern and the relative blood flow dictate the thermal pattern which is the basis for thermographic interpretation. The normal thermal pattern of any area can be predicted on the basis of its vascularity and surface contour. Skin overlying muscle is also subject to temperature increase during muscle activity. Injured or diseased tissues will invariably have an altered circulation. One of the cardinal signs of inflammation is heat which is due to increased circulation. Thermographically, the "hot spot" associated with the localized inflammation will generally be seen in the skin directly overlying the injury. However, diseased tissues may in fact have a reduced blood supply either due to swelling, thrombosis of vessels, or infarction of tissues. With such lesions the area of decreased heat is usually surrounded by increased thermal emissions, probably due to shunting of blood.

Scintigraphy utilizes polyphosphonate radiopharmaceuticals administered by intravenous injection and followed by measurement of the distribution of the pharmaceutical by a gamma camera.[11] Concentrations of the pharmaceutical can be detected, as the polyphosphonates bind rapidly to exposed hydroxyapatite crystal. This is generally in areas where bone is actively remodelling. This is the basis of the bone scan but prior to this the distribution of the drug goes through two other phases. It is these phases that can be useful to evaluate soft tissue changes.

There are three phases, the vascular phase, the soft tissue phase, and the bone phase.[11] The vascular phase or blood pool phase begins immediately after injection of the pharmaceutical. This phase is dependent on local variations in vascular supply. The most common clinical application for vascular phase scintigraphy is determination of patency of blood vessels. The second phase or soft tissue phase scintigraphy is performed while most of the pharmaceutical is in the extracellular fluid (ECF). This usually begins 1-2 minutes post pharmaceutical injection and lasts until significant uptake of the polyphosphonate by bone, usually 1-2 hours. The distribution of the radiopharmaceutical during this phase is due to local blood flow, capillary density, capillary permeability and regional ECF volume. Because inflammation causes an increase in blood flow, capillary permeability and ECF volume, inflamed tissues accumulate high levels of radiopharmaceutical. This is the basic principle behind evaluation of soft tissue injuries by scintigraphy. The bone phase is the most useful in that the uptake of the radiopharmaceutical always increases around areas of increased

remodeling or vascularity. Since injured bone is undergoing more rapid remodeling, this is the basis for using bone phase to detect injuries. Scintigraphy has been most useful for the detection of lesions in bone and ligaments. Scintigraphy has been particularly useful in the identification of enthesopathy (damage to the insertions of tendons and ligaments on bone).

The purpose of any lameness examination is to be able narrow the problem to a regional diagnosis. Once a regional diagnosis has been made it is possible to assess the area utilizing some type of anatomical imaging modality. Assessment of those anatomical changes serves as the basis for any pathologic diagnosis that may be made, as well as, being important in determining prognosis. For these purposes radiography and ultrasonography are complimentary. Radiography provides information regarding the boney tissues. Radiographs reflect change that has happened. Ultrasonography provides information about boney contour but more importantly provides insight to the soft tissues that connect bone or provide support. Sonography can give much better insight into the activity of a lesion. That is, is the lesion active or not, do the soft tissues changes reflect an ongoing process or is it a chronic process. In addition, sonography can provide information about joint capsule, collateral ligaments, the consistency of joint fluid, and provide insight into the articular cartilage.

However, the pursuit of a regional diagnosis can be difficult. There are 3 instances where this can be frustrating. One, when diagnostic analgesia has failed to eliminate the lameness; two, when the lameness is too subtle to avail itself to diagnostic analgesic techniques, and three, when the patient is not amenable to handling or injection. In these cases, other methods must be used to evaluate the patient. This is where physiologic imaging modalities can be so useful. By providing insight into physiologic changes in the tissues, this can lead the examiner to evaluate those areas utilizing anatomic imaging methods.

Another area in lameness evaluation where imaging can be useful is in preventing injury. This requires the early detection of the physiologic change of injury. Although, the frequent use of an anatomical imaging modality can discover change in one region, physiologic imaging allows the assessment of the entire animal on a routine basis.

The utilization of imaging modalities in the diagnosis and treatment of equine lameness is absolutely necessary. This is the only reliable method to assess the type and severity of the injury. In addition, the routine use of any method can provide insight into the stresses and strains of the athlete.

References

1. Baxter GM, Stashak TS: History, Visual Exam, Palpation and Manipulation in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, pp109-150.

2. Turner TA: Examination of the equine foot. Vet Clin NA: Eq Prac, 19(2): 309-332, 2003.

3. Turner TA: How to Use Manipulative Tests to Diagnose and Manage Equine Foot Pain in 60th Annual Meeting Am Assoc Eq Practnr, 2014:59-61.
4. Turner TA, Waldsmith JK, Wilson JH: How to assess saddle fit in horses. 50th Annual Meeting Am Assoc Eq Practnr, 2004: 196-201.
5. Baxter GM, Stashak TS: Perineural and Intrasynovial Anesthesia in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, 173-202.
6. Valdes-Martinez A, Park RD: Radiology in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, 207-337.
7. Redding WR: Ultrasonoraphy in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, 338-376.
8. Schramme M, Redding WR: Magnetic Resonance Imaging in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, 416-450.
9. Pease AP, Redding WR: Computed Tomography in Baxter GM(ed) Adams and Stashak's Lameness in Horses. West Sussex, Wiley-Blackwell, 2011, 451-459.
10. Turner TA: Diagnostic thermography,Vet Clin N.A.: Equine Pract. 17(1): 2001, 95-114.
11. Dyson SJ, Pilsworth RC, Twardock AR, Martinellin MJ: Equine Scintigraphy, Newmarket, EqVetJ; 2003.

GIZMOS and GADGETS, WITCHCRAFT or WIZARDRY?

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TAKE HOME MESSAGE: The horse industry likes gizmos and gadgets and there are companies that make products to appeal to this interest. What evidence is there that any of these things have an effect let alone a beneficial one? We evaluate the evidence of some modalities to determine if they are witchcraft or wizardry.

INTRODUCTION: A number of physical therapies are used to manage pain in horses with musculoskeletal disorders.[1,2] These can be broadly categorized as thermal modalities (e.g., cold, heat, therapeutic ultrasound), electrotherapy modalities (e.g., TENS, FES), acupuncture, manual therapies (e.g., manipulation or massage), or exercise. In practice, therapists use a combination of modalities to address the treatment needs of patients based on the findings of clinical examination. In the past, some therapies have been particularly espoused in different countries or by different groups of therapists. Increasingly, however, an evidence-based approach to management requires the selection of therapeutic modalities for which there is scientific evidence indicating effectiveness. However, there are still many specific therapies or specific dosage regimes that have not been subjected to scientific investigation. The aim of this review is to provide a current perspective on research related to the mechanism of action and therapeutic effectiveness of each of the broad therapeutic categories listed above, specifically related to the management of musculoskeletal pain.

THERMAL MODALITIES: A variety of methods produce cooling and heating of the tissues to manage musculoskeletal pain in acute and chronic musculoskeletal conditions. The effects and effectiveness of cryotherapy, superficial heating, deep tissue heating, and therapeutic ultrasound are discussed.

Cryotherapy. Cryotherapy is the use of cold for the treatment of inflammatory pain has been recognized for hundreds of years. Cold is applied in a variety of ways to reduce pain and swelling, including ice bags, ice baths, cold packs, cold baths, and ethyl chloride spray to the skin.[1,2] Cold treatment decreases skin and joint temperature, decreases blood flow, and increases joint stiffness.[3] In addition, it is quite clear that cold is analgesic. Topical application of cold decreases skin, muscle, and intraarticular temperature.[3]

Measuring local pain thresholds after treatment with ice gives varying results, with the effectiveness lasting from 30 minutes to 12 hours.[1,2] Cold also slows the conduction velocity of peripheral nerves. This being the case, decreased nociceptive information transmitted through primary afferents centrally to the spinal cord would result in a decrease in behavioral signs, and a decrease in neuronal activity of dorsal horn neurons. Because an ice bath decreases secondary hyperalgesia, it is inferred that there would be a decrease in activity of central neurons and a reduction of the expanded receptive fields. Alternatively, it has been suggested that application of cold to an arthritic joint serves as a counterirritant by bombarding central pain pathways with painful cold impulses and

activating descending inhibitory pathways. Support for this is based on the observation that cooling the skin alone, as with ethyl chloride spray, increases pain threshold.[2]

Several studies demonstrate immediate or short-term effects of ice on pain in humans with rheumatoid arthritis and low back pain.[2] Ice packs in combination with exercise were compared to hot packs in combination with exercise in patients with rheumatoid arthritis of the shoulder. Significant reductions in pain occurred for both groups but there was no difference between groups. Comparisons have been made between the effects of ice low back massage for pain to TENS and found a significant reduction in pain with both techniques, with TENS showing longer lasting relief (23 hours) compared to ice (12 hours). An analgesic effect has been demonstrated from both heat and cold treatment in patients with rheumatoid arthritis, without differences between groups. Thus, short-term effects of ice are clearly analgesic and there may be some long-term benefits. However, cold treatment does not appear superior in effect to other noninvasive treatments. It may be that application of cold, like heat, is useful as an adjunct therapy to allow a patient to exercise with reduced pain.

Superficial heat. The use of heat to relieve pain of musculoskeletal origin is common.[1,2] Heat can be applied superficially by application of moist hot packs, immersion in hot water baths, use of infrared light, or paraffin wax application. The most common method utilized for treating musculoskeletal conditions is moist hot packs. Moist hot packs are applied over the area of pain for 20-30 minutes and only heat the superficial tissues.[3]

Many of the local physiologic effects of heat have been studied thoroughly. For instance, heat increases skin and joint temperature and blood flow,[3] and decreases joint stiffness.[1,2] Activity of local cartilage-degrading enzymes is influenced by joint temperature. As found in patients with rheumatoid arthritis, when temperature increases from the normal of 33 °C to 36 °C these enzymes are considerably more active. Temperatures above 41 °C decrease the activity of these enzymes. However, the data on superficial heat treatment are not consistent with respect to decreasing pain or increasing function. Some studies show decreases, some show increases, and some show no change in arthritic pain or associated symptoms.

The use of superficial heat is common and has been studied by several different groups. Superficial heat increased the pressure pain threshold over approximately 50% of trigger points immediately after treatment in patients with myofascial pain who had at least one trigger point.[2] Increases in pressure pain thresholds were also noted in patients with temporomandibular joint dysfunction. Comparisons between whirl-pool or paraffin wax baths alone or with exercise, showed significant pain relief in all groups with no differences between groups, although both groups had increased range of motion and decreased pain. No difference between groups was observed when superficial heat was compared to cold treatment.

Muscle spasms or guarding can result in a pain response or contribute to the pain experienced. Muscle spasms can cause local ischemia activating nociceptive afferent fibers. Reducing muscle spasms and guarding would then be expected to reduce pain by reducing ischemia and preventing activation of nociceptors. Elevating muscle temperature to about 42 °C decreases the firing frequency of Type II muscle spindle afferent fibers and increases the firing frequency of Type Ib Golgi tendon organ afferent

fibers. However, Type Ia muscle spindle afferent fiber firing frequencies are also increased in response to elevation of muscle temperature. Type II muscle spindle afferent fibers are tonically active and respond to muscle length. Type Ia muscle spindle afferent fibers respond dynamically and respond to velocity of change in muscle length. When activated, the Type Ia and Type II muscle spinal afferent fibers cause an excitation of the agonist muscle and inhibition of the antagonist muscle. Golgi tendon organs respond to muscle stretch and when activated inhibit the agonist muscle and excite the antagonist muscle. Thus, increasing muscle tissue temperature could reduce muscle spasm by decreasing activity of Type II muscle spindle afferents and increasing activity of Type Ib Golgi tendon organ afferents. This may be a viable explanation for the use of deep heating modalities. Alternatively, heat could decrease pain indirectly. Increasing skin temperature or deep tissue temperature would cause vasodilation of the tissue, increased metabolism, and increased blood flow, all of which assist with healing and repair.[3] Improving healing and repair would result in increased removal of inflammatory compounds, known to activate and sensitize primary afferent fibers. This would result in less input being transmitted to the spinal cord and higher brain centers, and thus decreased perception of pain.

Therapeutic ultrasound. Consists of inaudible acoustic vibrations delivered at a frequency between 0.75 and 3.0 MHz and intensity between 0.5 and 3 W/cm2. The lower the ultrasound frequency, the deeper the penetration of sound waves. There are three primary benefits to ultrasound. The first is the speeding up of the healing process from the increase in blood flow in the treated area.[3] The second is the decrease in pain from the reduction of swelling and edema.[1] The third is the gentle massage of muscles, tendons or ligaments in the treated area because no strain is added and any scar tissue is softened. These three benefits are achieved by two main effects of therapeutic ultrasound. The two types of effects are: thermal and non-thermal effects. Thermal effects are due to the absorption of the sound waves. Non thermal effects are from cavitation, microstreaming and acoustic streaming. The thermal effects have been documented in horses,[3] the non-thermal effects have not. Heating occurs predominantly at tissue interfaces.

Extracorporeal Shockwave Therapy. ESWT are abrupt, high amplitude pulses of mechanical energy, similar to soundwaves, generated by an electromagnetic coil or a spark in water.[4] “Extracorporeal” means that the shockwaves are generated externally to the body and transmitted from a pad through the skin. With ESWT, reduced pain and faster healing are reported. The exact physiological mechanisms at this stage are poorly understood, but it appears that the cells undergo microtrauma which promotes the inflammatory and catabolic processes that are associated with removing damaged matrix constituents and stimulates wound healing mechanisms.

Electrical stimulation. Electrical muscle stimulation (EMS), also known as neuromuscular electrical stimulation (NMES), is the elicitation of muscle contraction using electric impulses.[1,2] EMS has received increasing attention in the last few years because of its potential to serve as a strength training tool for healthy subjects and athletes, a rehabilitation and preventive tool for partially or totally immobilized patients, a testing tool for evaluating the neural and/or muscular function in vivo, and a post-exercise recovery tool for athletes. The impulses are generated by a device and

delivered through electrodes on the skin in direct proximity to the muscles to be stimulated. The impulses mimic the action potential coming from the central nervous system, causing the muscles to contract. The electrodes are generally pads that adhere to the skin. Electrical stimulation is reported to have many benefits: (1) Pain relief caused by decreased spasticity of muscle, (2) Improved range of motion caused by reduced muscle tension, (3) Reduction in swellings caused by injury, (4) Reduction of scar tissue during healing, (5). Re-education of muscle function to prevent further injury, (6) Strengthening of muscles and tendons, (7) Reversal of muscle wasting, and (8) Decreased rehabilitation time after injury and surgery.

There are different types of electrical stimulation, among the most common are transcutaneous electrical nerve stimulation (TENS) is the use of electric current devices to assist with short-term or long-term pain relief. TENS units are designed to produce analgesia of pain and reduce responses of dorsal horn neurons to painful stimuli. The TENS systems activate the descending inhibitory pathway from the brain stem to the spinal cord. However, the means of reducing pain varies between the specific types of systems and includes activating spinal cord gating mechanisms, endogenous opiates, serotonin receptors, noradrenaline receptors, and muscarinic receptors. Regardless, the electrical signals cause muscles under the electrodes to contract.

Another type of electrical stimulation is functional electrical stimulation (FES).[5] FES is the application of an electrical current through surface electrodes to produce a controlled muscular contraction. A microprocessor generates a train of impulses, which imitate the neural signals that pass between the spinal cord and the peripheral nerves in healthy muscle, producing a muscle contraction. FES is utilized to disturb spastic hyperexcitability, returning the muscle to its balanced contraction and relaxation phases, therefore reducing pain. FES devices are designed to stimulate motor nerves but peripheral nerves are also stimulated when motor nerves are activated, so there is a combined effect. FES provides a means to mobilize muscle, tendon, and the associated ligaments through the generation of controlled muscular contractions. FES can be used for stimulation of deeper tissues, and therefore, the attainment of strong muscular contractions is possible. Stronger contractions have been shown to be more effective in reducing pain, and the benefits have proven to be longer lasting than other forms of electrical stimulation.

Pulsed electromagnetic field therapy (PEMF), is a reparative technique most commonly used in the field of orthopedics for the treatment of non-union fractures, failed fusions, congenital pseudarthrosis and depression.[1,2] More recently, PEMF has been used as a more for healing other types of connective tissue including muscle.

There are a number of other modalities that we have assessed but need more research to determine the benefits. Among these are compression devices, vibrating plates, photon or laser light therapy. We have tested the effects these modalities have on circulation and there appear to be benefits but controlled research is needed. Regardless, of the modality, in order to achieve the goal of improving flexibility and muscle condition, it is the author's opinion, the horse must remain in at least low intensity exercise.

REFERENCES

1. Porter M: The New Equine Sports Therapy. Lexington, The Blood Horse, 1998; pp 11-199.
2. Bromiley M: Equine Injury, Therapy and Rehabilitation. Oxford, Blackwell Publishing, 1993; pp 76-159.
3. Turner TA, Wolfsdorf K, Jourdenais J: Effects of heat, cold, biomagnets and ultrasound on skin circulation in the horse. In Proceedings of 37th Annual Meeting of Am Assoc of Equine Practnr, 1991: 249-257.
4. Verna M, Turner TA, Anderson K: Scintigraphic, radiographic, and thermographic appearance of the metacarpal and metatarsal regions of adult healthy horses treated with non-focused extracorporeal shockwave therapy-a pilot study. Vet Therapeutics, 6(3): 268-276, 2005.
5. Schils S, Carraro U, Turner TA, et al: Functional electrical stimulation for equine muscle hypertonicity: Histological changes in mitochondrial density and distribution. J Eq Vet Sci, August 2015.

PRACTICAL EQUINE REHABILITATION FOR THE PRACTITIONER

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TAKE HOME MESSAGE: Injections and surgery are the most common sports medicine techniques used by veterinarians. However, veterinarians are learning the difference between success and failure of these treatments is aftercare. Rehabilitation is based on healing, improving flexibility and physical conditioning, strengthening the injured tissue, and then slowly returning to full activity.

INTRODUCTION: Human physical therapy describes the care and services provided by or under the direction of a licensed physical therapist. In human medicine, physical therapists are regarded as clinicians. They engage in an examination process that includes taking the patient/client history, conducting a systems review, and performing tests and measures to identify potential and existing problems. Based on their judgments, physical therapists provide interventions, conduct reexaminations, modify interventions as necessary to achieve anticipated goals and expected outcomes. They are the only professionals who provide physical therapy examinations, evaluations, diagnoses, prognoses, and interventions based on the physical therapist's examination and evaluative process. In veterinary medicine, only a veterinarian can make a veterinary diagnosis and it is the veterinarian's responsibility to coordinate all phases of treatment and aftercare.

VETERINARY REHABILITATION: Certification programs in equine physical rehabilitation are offered to veterinarians, veterinary technicians, physical therapists, chiropractors and physical therapist assistants by these organizations: The Animal Rehab Institute in Loxahatchee, Florida http://animalrehabinstitute.com/ or the University of Tennessee, Knoxville, TN http://equinerehab.utk.edu/index_certificate.php

Equine rehabilitation is the application of an all-new diagnostic algorithm to our horses. It focuses upon soft tissues rather than bone and joint. It involves special tests that allow for determination of specific tendinopathies and soft tissue abnormalities. The use of objective outcome measures will be absolutely necessary if this area is to grow. The emphasis in rehabilitation therapy is on meeting goals that are functional for the patient. The goals of rehabilitation include the restoration, maintenance and promotion of optimal function and quality of life as they relate to movement disorders. Equine rehabilitation is in its infancy. It is an exciting time but we must progress carefully and look toward those with experience in this specific area. However, this does not mean that we ignore those who have been working in the field of rehabilitation for decades. These individuals may not have formal background in equine rehabilitation but have learned by trial and error what works and what doesn't. These people can include veterinarians that have had a special interest in rehabilitation over the years, but can also include technicians who have been working on the race track or with performance horses in charge of the rehabilitation protocols.

During rehabilitation from lameness, physical therapy may include manual techniques, an exercise-based conditioning program to activate and strengthen specific muscles, aquatic therapy and the use of various modalities, such as therapeutic ultrasound and laser. This talk addresses specific areas of equine rehabilitation that fall within the realm of veterinary physical therapy and that are supported by evidence-based research. These techniques are based in movement and locomotor sciences and the underlying concept of motor control, which is central to understanding rehabilitation from musculoskeletal injuries.

Who should be doing the rehabilitation work? The person to oversee the protocol should be the veterinarian, however the veterinarian should seek advice from those who know the science of rehab and look to develop a team approach. The veterinarian will not be the one doing the rehab protocol on a daily basis, but rather the one to help organize the general protocol. The owner or a technician will typically be doing the day to day work of the rehabilitation, not unlike in the human health care system.

Rehabilitation takes time. In chronic cases, it took time for the problem to show up therefore it is going to take time to fix the problem. Not only does the primary problem need to be solved, but rehabilitation to improve the secondary problem of poor biomechanics must also be solved so that there is a lower chance of reinjury. In acute cases, such as trauma, the time required for rehabilitation may be shorter if there are no secondary mechanical problems that inhibit the healing process.

Rehabilitation utilizes manual therapies and therapeutic modalities. Some basic research has been done on some modalities but more work is necessary to determine the effects these therapies have on the equine body. Through this type of basic research the ground work will be laid not only to determine when it would be appropriate to use a particular therapy but also the intensity of treatment as well as the duration. For instance, under what circumstances should therapeutic ultrasound be used? Electrical stimulation? Shockwave? Magnetic or electromagnetic therapy? Massage? It will be the combination of basic research and clinical experience that will be needed to develop protocols for these modalities. We have already discussed these modalities in a previous paper so the veterinarian can make therapeutic decisions.

How will the process look? The process starts with an accurate diagnosis and an accurate assessment of the injury. In addition, an overall assessment of the horse's secondary or compensatory problems must be performed. This should also include an assessment of hoof balance. Making an accurate diagnose may require different imaging modalities including MRI, CT, ultrasonography and radiography. In addition, a physiologic imaging modality like thermography or scintigraphy may be very useful in identifying the secondary problems.

The next step is to determine the most appropriate therapy and determine if any modality will help the healing process. This must be followed by development of a protocol to restore function. This is not merely healing of the original injury but restoration of range of motion and strength. This would require correction of secondary

issues as well as correction of hoof balance issues. This will require the development of new techniques to measure range of motion of horses as well as measuring strength. This is not to mention the difficulties of determining the healing of secondary issues.

Rehabilitation of performance horses is unique because the rider position must also be part of the solution. Rehabilitation requires the integration of many people to make it work, which is not unlike most areas of veterinary medicine. Some of these people that will need to understand the rehabilitation process include the: owner, trainer, saddle fitter, farrier, nutritionist and barn manager.

Rehabilitation is not just the use of machines and modalities, it is the knowledge of how to use them in an appropriate protocol.

Treatment and Prevention of Equine Back Pain. Back pain is recognized as a common and important cause of poor performance in athletic horses, which has stimulated clinical and research interest in the diagnosis and treatment of equine back pain. There is extensive literature describing the clinical manifestations, pathoanatomical diagnoses, treatment and rehabilitation of back pain in people. It is known that low back pain is associated with persistent inhibition of activity in the deep spinal stabilizer muscles, specifically the *multifidus* and *transversus abdominis.* Within a matter of days after an injury such as a herniated disc, neurogenic atrophy is evident in the *multifidi* at the affected spinal level on the ipsilateral side.[1] After back pain resolves the atrophied muscles do not usually resume normal activation patterns unless specific therapeutic exercises are prescribed. The use of these exercises has been shown to reduce the one-year recurrence rate from 80% to about 30%.[2]

Back pain in horses appears to follow the human model in that affected horses have atrophy of *multifidus* on the affected side(s) and resolution of back pain is not necessarily accompanied by reactivation or hypertrophy of *multifidus*. This is in contrast to the response of the superficial and easily palpable *longissimus* muscle which may go into spasm as it attempts to compensate for the loss of spinal stability. However, the attachments of *longissimus* dictate that it is more effective in moving the spine than in stabilizing the individual inter-vertebral joints. Loss of the stabilizing influence of *multifidus* allows micro-motion of the inter-vertebral joints which predisposes to the development of degenerative joint disease.

Anecdotal reports suggest that dynamic mobilization exercises (baited stretches) performed in flexion or lateral bending are beneficial in athletic horses with back pain. The principle of these exercises is that a bait, such as a piece of carrot, is used to entice the horse to move the chin to a specific position.[3] The exercises are performed in cervical flexion with the chin moving to the chest, between the carpi or between the fore fetlocks, in cervical extension and in lateral bending to the left and right sides with the chin moving to the girth, hip, flank or tarsus/hind fetlock.

The beneficial effect of these exercises is based on the need to activate the deep spinal musculature (*multifidi*) to stabilize the thoracolumbar-sacral spine as the head and neck assume a variety of extreme positions. The benefits have been confirmed in two research studies both of which used ultrasonography to show significant increases in cross-sectional area of the *multifidus* muscles in response to performing baited

stretches regularly over a period of time. In the first study 8 school horses performed baited stretches on 5 days/week for 3 months without any other exercise. There were significant increases in *multifidus* CSA bilaterally at T10, T12, T14, T16, T18 and L5, together with significant improvements in left: right symmetry at each of these spinal levels.[4] A second study conducted in Thoroughbreds in race training found a significant increase in *multifidus* CSA at T16 in horses that performed baited stretches daily for 6 weeks in addition to their regular training program but not in controls subjected to the same training program without baited stretches.[5]

It is recommended that baited stretches be used both prophylactically and therapeutically. Three to five daily repetitions of each exercises appears to be adequate to restore or maintain activity and strength of *multifidus*.

Proprioceptive Stimulation. Following recovery from orthopedic injury, gait deficits may persist due to soft tissue stiffness, muscle atrophy, altered neuromotor coordination patterns or inhibition of specific muscles. These deficits do not necessarily resolve spontaneously with normal exercises and training.[6] During rehabilitation, physical therapy is used to restore function in terms of range of joint motion (ROM), muscle activation, and muscular coordination and strength. Muscle strength and endurance are improved by increasing the intensity or number of repetitions, respectively, of exercises targeted at recruiting specific muscles. Sometimes a muscle becomes inhibited as a consequence of a minor trauma involving pain or capsular distension. This type of inhibition usually involves the deep stabilizing muscles of a joint rather than the prime movers. The result is joint instability that predisposes to degenerative changes due to micro motion of the joint. A well-documented example in the human field is the development of patellofemoral pain following a minor pathology such as distension of the femorotibial joint. This leads to altered knee joint proprioception and changes in the activation sequence of the different heads of quadriceps femoris. Vastus medialis obliquus, which is a deep stabilizer of the knee, shows delayed onset of activity leading to a wide range of effects including maltracking of the patella and loss of strength in the external rotators and abductors of the hip. Evidence-based studies support the use of physiotherapeutic treatment targeting vastus medialis obliquus by use of taping, bracing, and various forms of therapeutic exercise.[7]

In horses little is known about the deep stabilizing musculature in locations other than the spine and there are no evidence-based reports of the effect of therapeutic exercises on specific muscles in the limbs. However, there have been reports of the effect of techniques that change the range of motion of the joints through stimulation of muscle activity.

Trot Poles. Trotting over poles is used therapeutically to restore full ranges of joint motion in the limbs. The kinematics and kinetics associated with trotting over poles on the ground (11 cm high) and raised poles (20 cm high) have been reported.[8] Peak heights of the fore and hind hooves increased significantly and progressively from no poles (fore: 13.8±3.8 cm; hind: 10.8±2.4 cm) to low poles (fore: 30.9±4.9 cm; hind: 24.9±3.7 cm) and to high poles (fore: 41.0±3.9 cm; hind: 32.7±4.0 cm). Peak hoof heights when trotting over poles were higher than with tactile bracelets or leg weights. All joints of the fore and hind limbs contributed to the increase in hoof height through increased swing phase flexion.

There were no changes in peak vertical force or in the degree of fetlock extension when trotting over poles suggesting that loading of the musculoskeletal tissues is comparable with that associated with trotting on level ground at the same speed. This makes it unlikely that trotting over poles will jeopardize the recovery process by over-loading the limbs. Peak forelimb braking force increased and the transverse impulse changed from being directed medially when trotting on level ground to laterally directed when trotting over poles. This was interpreted as a mechanism to enhance balance and suggests that the adductor musculature is being recruited.

Unlike the use of proprioceptive stimulation devices in which the effects decrease over time due to habituation, the horse is required to elevate the hooves to ensure clearance whenever poles are present. The need to raise the limbs sufficiently to clear the poles and to adjust the step length so the hooves are placed accurately also requires visuomotor coordination which suggests an application in rehabilitating neurological cases.

References

1. Hodges P, Holm AK, Hansson T, et al. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine 2006;31:2926-2933.
2. Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine 2001;26:E243-248.
3. Stubbs NC, Kaiser LJ, Hauptman J, et al. Dynamic mobilization exercises increase cross sectional area of musculus multifidus. Equine Vet J 2011;43:522-529.
4. Stubbs NC, Clayton HM. Activate your horse's core. Sport Horse Publications, 2008:Mason, MI.
5. Tabor GF, Johansson C, Randle H. The effects of dynamic mobilization exercises on the multifidus muscle in thoroughbred racehorses, In: 8th International Equitation Science Conference, Edinburgh, Scotland, 2012:64.
6. Van Harreveld PD, Lillich JD, Kawcak CE, et al. Clinical evaluation of the effects of immobilization followed by remobilization and exercise on the metacarpophalangeal joint in horses. Am J Vet Res 2002;63:282-288.
7. Crossley K, Bennell K, Green S, et al. A systematic review of physical interventions for patellofemoral pain syndrome. Clinical journal of sport medicine: official journal of the Canadian Academy of Sport Medicine 2001;11:103-110.
8. Brown S, Stubbs N, Kaiser L, et al. Swing phase kinematics and kinetics of horses trotting over poles. Equine Vet J, DOI: 10.1111/evj.12253, Epub 2014 Apr 9

Demonstration and Discussion of Equine Rehabilitation Equipment

The group will be divided into 4 smaller groups. Each group will get visit each station for approximately 10 minutes to hear and see a brief presentation about the specific piece of equipment. The main considerations with each piece of equipment include cost, labor involved, each of maintenance, safety, potential benefits and lessons learned.

Group 1 – Vibrating plates and Solarium Treatments

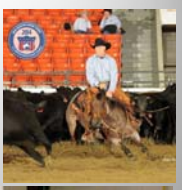
Group 2 – Treadmills and Eurocisers

Group 3 – Water Treadmills and Cold saltwater spa

Group 4 – Unmounted exercises to strengthen core muscles and improve balance

After each group has rotated through each demonstration, the entire group will gather for questions and answers.

Evaluating hind abnormal foot conformation…



…or an idea for seeing/preventing a potential cause for behavioral, performance or lameness in our horses

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Hind foot conformation

Stand at the side and follow a visual line from the hind foot coronary band upward & forward

Stands with limbs properly under the horse

Stands like "goat on a rock"

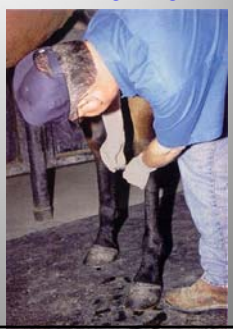
Function of elongating toe and/or lowering heel

(Bailey,A How shoeing contributes to Heel Collapse (in the Modern Horse) BGLS 2007)

Follow the visual line of hind coronets-makes one examine more thoroughly

Evaluating and treating long toe/low heel hind feet abnormal foot conformation. Clinically maybe related to the horse getting:

- Sore heels
- Sore suspensories – high and low
- Sore fetlocks
- Sore hocks – hock injections only work a few weeks
- Sore backs
- Decreased performance or LAME



Painful gluteal muscles as determined by palpation might be related to elongated toes (breakover distance)

Clinical signs

Back pain
Intermittant hind end lameness
Behavior issues – stopping, bucking,etc
Joint injections don't work as well as they used too?

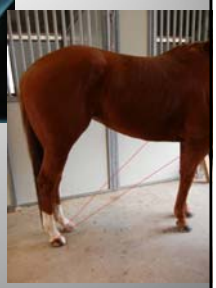
Typical case we see and treat with shoeing changes

Pre shoe

Post shoe 5 mm BD

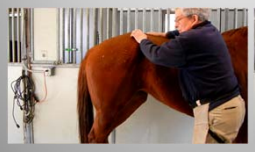
5 year old TB
Show hunter

Pre shoe 30 mm Breakover Distance



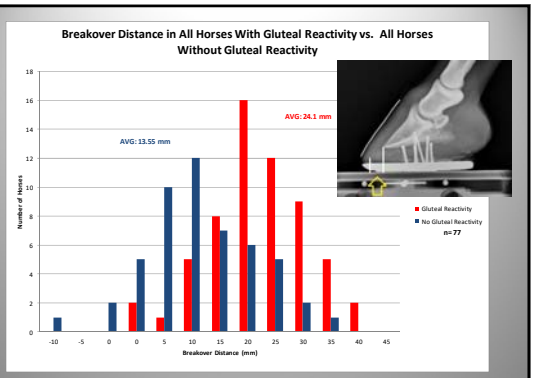
RH shod LH pre shoeing

Significant gluteal muscle pain from elongated toes in the previous horse and then one month later (actually 1 week)



BD 30mm

BD 5 mm



From Mansmann, RA, James,SJ, Blikslager,AT & vom Orde, K. Long Toes in the Hind Feet and Pain in the Gluteal Region:An Observational Study of 77 horses. JEVS. Dec 2010,pp.720-726

What happens to long toe/low heeled horses – vicious cycle!

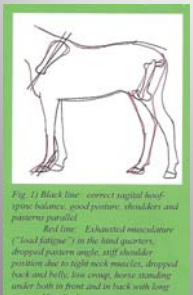


Fig. 1) Black line: correct sagittal hoof-spine balance, good posture, shoulders and pasterns parallel.
Red line: Exhausted musculature ("load fatigue") in the hind quarters, dropped pastern angle, stiff shoulder position due to tight neck muscles, dropped back and belly, low croup, horse standing under both in front and in back with long toes and low heels.

- Long toes can fatigue the horse
- Fatiguing the horse can create a "long toe low heel" stance

From article in 2011 The European Journal vol 152. 2011

Radiographs need to be consistent

to compare from year to year, month to month or day to day

- Same measured marker – we use 75mm soldering wire
- Push tape against the hoof wall
- Can use barium, but messy. Good for selective cases
- Same film focal distance
- Foot up against the cassette

Radiographs need to be consistent

to compare from year to year, month to month or day to day

- Flat surface
- Central beam about 1 cm above shoe (so that is the height of your block – central beam from the ground + 1cm)
- Same for any system; digital or otherwise

This wooden block can be used for a variety of views.

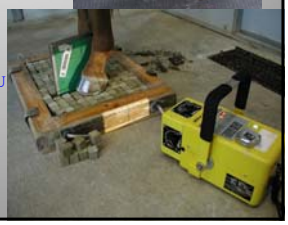
This block is our most recent, made from rubber matting which has good traction & quiet for the horses

Farriers can help their veterinarian make a decent block

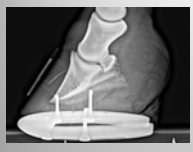
Hind feet are more difficult to radiograph mainly because horses don't like standing on blocks and tend to toe out

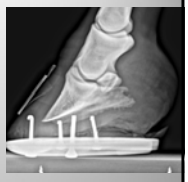
- Sedating most horses helps
- The lower the block the easier to get a horse to stand on them, but have to have enough height for central beam to split the shoe and distal border of P-3
- It is amazing how much we have learned from doing lateral radiographs of hind feet. (still don't know normal yet though!)
- Prior to 2005 only 3% of all lateral radiographs taken at NCSU were hind feet
- And 25% of all laminitics have hind feet involvement?!

So missing 22%?



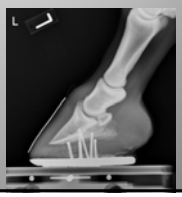
Goal is to have the shoe become a single bar for easy consistent measurements

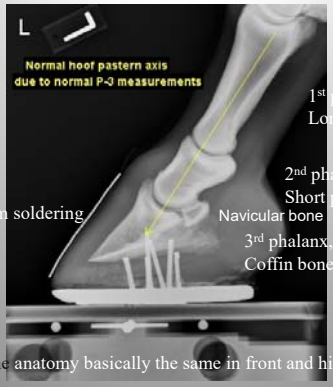




- The higher the machine the more oblique the branches of the shoe and less the sole depth would be?
- Probably not having the branches equal changes break over evaluation?

Take home message –
"The shoe should be a flat bar (line)"





1st phalanx
Long pastern bone

2nd phalanx
Short pastern bone

75mm soldering wire

Navicular bone

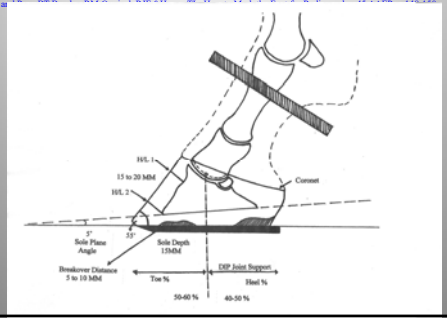
3rd phalanx, distal phalanx
Coffin bone, P-3

Bone anatomy basically the same in front and hind limb

What is normal?

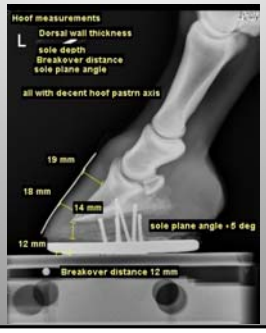
Five measurements that are either **FIVE** or **Factors** of **FIVE**

Picture adapted from Redden, R. Understanding Laminitis. The Blood Horse,Inc, Lexington, Ky. 1998. P.44



What is normal?

Five measurements that are either **FIVE** or **Factors** of **FIVE**



This is not an uncommon long toe/low heel radiograph that we see with a combination of issues:

Palmar or plantar process reaction

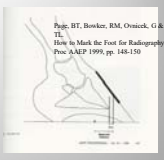
- An elongated break over distance that is causing levering against the DDFT and heel
- The sole depth under the palmar (plantar) processes is less resulting in a negative sole plane angle
- **Frequently there appears to have some reaction to the palmar (plantar) processes. (*Long white arrow*)**

Scrold,MT and Moss, JA. Two Common Pathologies of Hind Feet. The Farrier Journal 352: Dec 2011, pp2-3

- The distal dorsal wall thickness is less thick than the proximal dorsal wall **(reverse laminitis??)**
- The heel support of the DIP joint is less than 40 % thus "driving" the entire hind foot support forward
- Distal phalanx angle - 41°- what is the normal distal phalanx angle

Measuring Break Over both on the horse & radiographically

- Identify true apex of the frog
- Place marker thumbtack or barium from 14 g needle
- Can measure from radiograph
- Can verify via measurement on the horse



This is not an uncommon long toe/low heel radiograph and then how we begin to treat:

- The wall/sole at the toe half grows faster than the wall/sole in the heel half of this type of foot
- Tend to have greater cup thus more wall to be trimmed off
- The trim itself helps reestablish the sole plane angle

Has conformation gotten worse over time?

Did the North have better farriers?

Painting by Richard Stone Reeves

From Stanley Arthurs painting 1922

ANATOMY AND PATHOPHYSIOLOGY OF LAMINITIS

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We use the term laminitis to describe the clinical and pathological consequences of disturbances in the attachment between inner hoof wall and distal phalanx that is normally provided by the digital lamellae. Importantly, the lamellae normally act to suspend the bone within the hoof capsule and it is the largely unrecoverable loss of this suspensory function that leads to the lameness, dysfunction and morphological derangements characteristic of chronic laminitis. Pathologic alterations in the lamellae can be mild and insidious, with slow and progressive lengthening of lamellae due to stretch and cellular proliferation (as in many cases of endocrine-associated laminitis) or may be characterized by rapid loss of tissue mechanical integrity due to widespread cellular adhesion and cytoskeletal failure (more common in sepsis-associated and supporting limb laminitis). Once the suspensory function of the lamellae is lost, the pain, morphological derangement and progression of digital pathology are largely consequences of novel mechanical forces now acting between the distal phalanx, sole and ground surface, together with the proliferative and dysplastic response of the lamellar epidermis to injury. These chronic processes are common regardless of the inciting cause. Over the last decade however, researchers have recognized that there are key differences (and some similarities) in the initial events that lead to laminitis depending on the inciting cause. A focus on these early events is leading to a better understanding of why laminitis occurs in different clinical situations and is helping to identify therapeutic targets.

Are there common pathways to lamellar dysfunction?

In all 3 types of laminitis (sepsis-associated, supporting-limb laminitis [SLL] and endocrinopathic laminitis), the lamellar epithelial cell is central to the events that lead to lamellar failure. In each case lamellar failure is caused by a combination of:

1. Loss of the normal cell shape (stretching) of the lamellar epithelial cells and
2. Failure of epithelial cell adhesions (both between cells, and to the dermis via the basement membrane).

Endocrinopathic laminitis is dominated by cell stretch with less evidence of adhesion loss, whereas sepsis-associated laminitis and SLL is dominated by adhesion loss. Cell stretching/loss of shape is caused by disruption of the cytoskeleton, and adhesion loss is due to disruption of hemidesmosome/desmosome dynamics. These events could occur as a result of either aberrant cellular signaling or a lack of cellular energy to maintain the dynamic cytoskeletal components and adhesions. The unique environment of the

lamellar epithelium means that the cells are normally subjected to profound mechanical stresses and therefore even minor events affecting regulation of the cytoskeleton and/or adhesion complexes could rapidly escalate and cause loss of mechanical integrity.

Disruption of energy supply as a cause of epithelial cell adhesion loss/stretch could occur as a result of perfusion failure (ischemia) or could be associated with more subtle tissue energy dysregulation/imbalances. Traditionally ischemia was hypothesized to play a central role in all types of laminitis, however to date there is only evidence for ischemia playing a role in SLL. There is preliminary evidence of subtle oxidative energy failure in sepsis-associated laminitis (see below) and lamellar energy balance has not yet been evaluated during endocrinopathic laminitis development. Since maintenance of epithelial cell cytoskeleton and adhesion complexes involves dynamic processes, aberrant cellular signaling may be involved in disrupting the normal balance and triggering laminitis. There is evidence emerging (from the Belknap laboratory at OSU in particular) that lamellar signaling in models of all 3 types of laminitis converge at the level of the mTOR signaling pathway, which plays a central role in cellular growth and homeostasis. The mTOR signaling pathway regulates epithelial-mesenchymal transition (EMT) which is important is many disease processes and dysfunction of mTOR signaling is implicated in an increasing number of conditions including cancer and diabetes. Since cytoskeletal rearrangement and cell adhesion dissolution are initial events EMT (and laminitis), aberrant mTOR signaling may be responsible for lamellar structural failure by causing disruption of these same cellular processes in lamellar epithelial. Furthermore, the role of mTORC1 as a metabolic sensor and regulator of cellular energy metabolism may be critical in laminitis pathophysiology, underlined by preliminary data demonstrating perturbations of energy metabolism in models of the SRL and SLL forms of laminitis.

Sepsis-associated laminitis

Laminitis occurs as a consequence of a range of conditions in horses where systemic inflammation is a feature; particularly when this inflammation is driven by bacteria or bacterial products liberated into the bloodstream (sepsis). Gram negative sepsis and endotoxaemia accompany metritis, pneumonia and colitis/enteritis in horses and these conditions are most often complicated by the development of acute laminitis. Experimental alimentary carbohydrate overload models traditionally used to study laminitis feature clinical signs that are characteristic of sepsis and studies have confirmed the presence of endotoxin in the blood and a systemic inflammatory response with these models. It is not surprising that endotoxin, usually of gastrointestinal origin, is an important trigger of sepsis in horses considering their exquisite sensitivity to it, but interestingly investigators have not been able to induce laminitis with experimental infusion of endotoxin alone. Nevertheless, the presence of clinical signs of endotoxaemia/sepsis is an established risk factor for the development of laminitis in hospitalised horses.

Sepsis is a malignant systemic inflammatory process triggered by bacteria and their products (including endotoxin) in the blood with a resultant cascade of haemodynamic alterations, coagulopathy and metabolic alterations. In most species including humans, the development of organ failure ("multiple organ dysfunction syndrome" [MODS]), most commonly affecting the lungs, liver and kidneys, has a major effect on survival in cases of sepsis/SIRS. Laminitis appears to be a form of end organ dysfunction/failure that is ultimately most important in terms of recovery for the adult horse with sepsis. The pathophysiology of organ dysfunction in sepsis (and sepsis associated laminitis) is still poorly understood. Efforts to determine the mechanisms that lead to sepsis-associated laminitis have mirrored those in human MODS research, with the investigation of circulatory derangements, local inflammatory processes, apoptosis and non-ischaemic derangement of cellular energy metabolism receiving the most attention. Although there is some evidence of microvascular dysfunction in MODS and laminitis there is no evidence of true ischaemia. The central role of inflammation in the pathogenesis of acute laminitis has been highlighted experimentally, with endothelial activation, cytokine and chemokine upregulation, and leukocyte emigration into the lamellar tissue occurring early during the development of experimentally induced laminitis. There is little evidence that apoptosis or oxidative damage play a primary role in laminitis development. Since the lamellae have a unique mechanical role, the lamellar basal epithelial cell adhesions and the integrity of the extracellular matrix have received special research attention, however recent evidence suggests that enzymatic degradation of extracellular matrix components by e.g. matrix metalloproteinase (MMP) enzymes appear to play only a secondary role. There is evidence of non-ischaemic energy failure (mitochondrial dysfunction) in models of human sepsis that is thought to play a role in MODS, but there is debate over whether this may represent a downregulation of cellular metabolic pathways that is actually protective in tissues during sepsis. Similar disruption of energy metabolism in sepsis-induced laminitis has not been definitively documented in the early developmental period, however preliminary results from a study in the author's laboratory suggest that there is dysregulation/failure of oxidative energy metabolism in the late developmental/early acute phase of experimentally-induced sepsis-associated laminitis: this may contribute to the failure of lamellar epidermal basal cells to maintain their critical intercellular and cell-matrix adhesions. Although insulin dysregulation may be a feature of sepsis in horses, it is clear from experimental models that laminitis develops in the absence of clinically significant hyperinsulinaemia. There is evidence that mTORC1 growth factor signaling is upregulated in experimental sepsis-associated laminitis – in this case through IL6 activation. These pathways are consistently blocked by hypothermia experimentally, which also ameliorates the laminitis lesion.

Endocrinopathic laminitis

Endocrinopathic laminitis encompasses laminitis associated with obesity, insulin dysregulation, pasture-associated laminitis, equine metabolic syndrome (EMS), pituitary

pars intermedia dysfunction (PPID) or glucocorticoid administration. This type of laminitis appears to be the most common worldwide. Recent research has shown that prolonged hyperinsulinaemia can induce laminitis in healthy ponies and horses. In contrast to sepsis-associated laminitis, lamellar inflammation does not appear to be a major feature and evidence of systemic or gastrointestinal inflammation is not apparent in models or natural disease. The histological changes seen in the lamellae are dominated by epithelial cell stretch rather than adhesion failure, and as the pathology progresses there is increased mitotic activity and cellular proliferation. Current evidence suggests that overstimulation of the growth factor receptor IGF-1R by excess insulin is responsible for triggering a proliferative response in lamellar epidermal cells which involves disruption of normal cell adhesions. Interestingly, excessive activity of the same growth factor receptor, IGF1R, is a major cause of epithelial-mesenchymal transition (EMT) in epithelial cancers and this is mediated through mTORC1. It is therefore likely that the cytoskeletal and adhesion dysregulation of endocrinopathic laminitis is due to mTORC1 activation occurring though activation of IGF1R by excessive insulin. The contribution of cellular energy failure through interference with perfusion, glucose uptake or metabolic pathways has not been specifically studied to date but appears unlikely on current evidence.

The concentrations of insulin in blood (and therefore tissue) vary widely in naturally occurring endocrinopathies and peak in association with soluble carbohydrate ingestion in particular. Episodes of hypersinulinemia in natural cases are also much less profound and more transient compared to those achieved in the protocol of insulin infusion used to induce laminitis experimentally. Transient hyperinsulinemic insults in natural cases are likely to contribute to the insidious and sometimes subclinical disease course of laminitis in many endocrinopathic cases. More severe and profound hyperinsulinemia upon exposure to carbohydrate-rich pasture for instance can still result in more severe acute laminitis in prone horses through these mechanisms.

Supporting-limb laminitis

Although the severity and duration of lameness are considered risk factors, the development of supporting limb laminitis (SLL) is still unpredictable, both in terms of timing and also with respect to which cases will succumb to it (and what degree of pain in the limb with the primary condition is necessary for SLL development). Despite making significant advances in our ability to treat complicated fractures and other painful limb conditions in horses, SLL remains the primary limiting factor to treatment success in these cases. This form of laminitis tends to be commonly associated with rapid and severe failure of the lamellae, with subsequent distal displacement (‘‘sinking’’) of the distal phalanx (DP) within the hoof capsule. The incidence has been estimated at approximately 10-15% of horses that present for painful limb problems (or require limb casts) in North American studies, however the threat of SLL likely leads to a reduced

propensity to even attempt treatment in many complicated painful limb conditions, with many of these horses instead being euthanased.

Mechanical and vascular mechanisms have been considered by authors as potential contributors to the pathophysiology of SLL but there is very little published evidence. It is well accepted that in the standing horse, the body mass is divided between the fore and hind limbs in a 60:40 ratio. Peak ground reaction forces increase to be equivalent to 0.25 x bwt at the walk, 0.5 x bwt at the trot, and up to 3 x bwt at the gallop. It therefore seems unlikely, from a mechanical perspective, that compensatory load redistribution in the standing horse bearing weight on a single limb could exceed the mechanical ‘‘strength’’ of the lamellae. The lamellar tissue has a high requirement for glucose, yet there is no means for local glycogen storage, therefore reduced supply of blood glucose may rapidly lead to energy failure. Almost 30 years ago it was first demonstrated that there was a valve-like mechanism in the digital arteries of the loaded limb leading to a cut-off of arterial blood supply during the loading phase. This work has been repeated recently by the author’s group using 3D computed tomography (CT) studies, demonstating arterial attenuation and occlusion under load in cadaver limbs that affects contrast fill in the arterial vasculature within the hoof capsule. Recent studies from the author’s group utilising microdialysis, (a technique that allows real-time detection of local energy metabolites and blood flow in the lamellar interstitium) showed that lamellar perfusion and energy balance are largely determined by the frequency of limb load cycling (weight shifting frequency), which appears to override other manipulations such as attempts modulate vasomotor tone pharmacologically. These studies showed that the act of walking had a marked positive effect on lamellar perfusion and interstitial glucose concentrations, more so than just repeated unloading of the limb in a static horse, indicating that the weight bearing phase itself (and perhaps break over) may play an important role in lamellar perfusion. Preliminary microdialysis studies using an experimental model of preferential weight bearing show that a combination of increased load and decreased limb load cycling frequency can cause reduced perfusion and negative energy balance consistent with ischaemia in the lamellar dermis. The mechanism of this is not clear from preliminary data but it appears more complicated than simple load dependent arterial occlusion: the results of these studies will be discussed.

CURRENT APPROACHES TO TREATMENT AND PREVENTION OF LAMINITIS

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There has been very little variation in our approach to the treatment of acute and chronic laminitis over the course of recorded history, mostly due to the fact that the normal function of the lamellae in suspending the distal phalanx within the hoof is largely unrecoverable once disrupted. Therapeutic efforts in laminitis should be focused on prevention and early intervention in acute cases. We now recognize that there are important differences in the mechanisms that lead to laminitis depending on the inciting cause, and laminitis may be classified as being sepsis-associated, endocrinopathic or supporting limb laminitis, with the preventative/therapeutic strategies and priorities being different for each.

Sepsis-associated laminitis

Therapeutic efforts to control the primary disease and systemic inflammation in cases of equine sepsis are paramount. Binding of circulating endotoxin using polymixin B and hyerimmune plasma and the use of NSAIDs to control downstream inflammation are important treatment strategies particularly in cases of gut-derived sepsis. Prophylactic continuous cooling of the feet has been demonstrated in several experimental studies and one clinical study of naturally occurring colitis to be protective and this is the only therapy to have withstood scientific rigor in human or veterinary research for the prevention of end-organ damage in sepsis. Continuous cooling of the feet using ice and water immersion of the distal limbs until after the abatement of clinical evidence of systemic inflammation is recommended. There is experimental evidence that cooling during the acute phase of laminitis (after lameness develops) can help to limit progression. The therapeutic mechanisms are not completely clear, however therapeutic cooling has profound effects on inflammatory mediators and a profound local hypometabolic effect.

Endocrinopathic laminitis

The key to prevention of endocrinopathic laminitis is early identification of horses at risk. Testing for evidence of insulin dysregulation (including insulin resistance as well as the propensity for post-prandial hyperinsulinemia) using insulin testing pre and post oral sugar challenge is the most effective way to identify horses and ponies at risk of laminitis development early. Management to reduce the laminitis risk in these cases can then

include a combination of dietary control, pasture access management, weight loss and exercise, which can dramatically reduce the risk of laminitis development or progression. Cases that have a profound hyperinsulinemic response to oral sugar ingestion may benefit from medications such as metformin that blunt this insulin response. Since the systemic effects of metformin on insulin sensitivity appear inconsistent, the use of targeted administration (prior to meal/turnout) appears most effective and rational in order to reduce postprandial hyperinsulinemia in these cases. Testing for pituitary pars intermedia dysfunction (PPID) in appropriately aged horses (at least >5 years) can help to identify this condition before the development of irreversible laminitis pathology. It is important to recognize that although laminitis may be the only important clinical manifestation of PPID in some horses, the recognition of clinical signs of laminitis (lameness) often only occurs after the development of irreversible laminitis pathology, which can be insidious and gradual. Treatment of PPID with pergolide can be quite effective in preventing laminitis development (or its progression) however dosage adjustment guided by frequent monitoring of ACTH is required for adequate control in most cases. It is impossible to adequately control chronic laminitis in cases where there is inadequate control of an underlying endocrinopathy.

Supporting-limb laminitis

It appears that cyclic loading and unloading of the feet plays an essential role in digital homeostasis. In a horse that is preferentially weight bearing on one limb, it is currently not clear whether static or dynamic manipulations of the supporting limb (using orthotics or other devices) are sufficient to improve lamellar perfusion and energy balance without intermittent complete unweighting of the limb, however studies specifically evaluating this are underway. The key to supporting limb laminitis prevention is likely to be the development of strategies to monitor and then regulate load cycling frequency in the supporting limb of patients at risk. Monitoring should include some form of serial assessment of limb load cycling – human pedometer devices and fitness tracking devices that incorporate accelerometers can track limb load cycling over time and may be of use clinically. In horses at risk, regular encouragement to walk may be beneficial, however there is insufficient data to support specific recommendations at this stage and the logistics of this may depend on the nature and severity of the primary condition. Strategies to reduce weight on the supporting limb may include partial sling support and periodic forced or encouraged recumbency. Although sedation may help to encourage recumbency, it also reduces voluntary exercise and limb load cycling in stabled patients and therefore may be contraindicated.

ICING (THERAPEUTIC HYPOTHERMIA)

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Introduction

There have been a multitude of treatments suggested and used for laminitis in horses, dating back to the beginning of recorded history. Amongst those, a traditional treatment for acute laminitis was to stand the affected horse in a cool stream. The effect of cooling the feet (cryotherapy/therapeutic hypothermia) has only recently been tested in experimental models of laminitis and it is the only therapeutic modality for laminitis that has stood up to repeated scientific scrutiny. The effects of hypothermia on tissue are complex, powerful, and difficult to replicate pharmaceutically. The value of therapeutic hypothermia in the modern clinical setting was first recognised and utilised in humans undergoing cardiac bypass surgery with the objective being protection and preservation of organs (particularly the heart and brain) during planned operative ischemia [1]. Therapeutic hypothermia has since been studied extensively and has achieved variable clinical adoption as a preventative as well as therapeutic agent in human patients with traumatic brain injury, stroke, cardiac arrest and neonatal encephalopathy [2-4]. There is some evidence from rodent sepsis models that therapeutic hypothermia may help to prevent and treat acute lung injury [5-7], an important manifestation of end-organ damage in human sepsis. Protocols for whole-body and regional hypothermia are currently evolving, with ideal methods, temperature and duration still not clear. Hypothermia can have detrimental effects, which are perhaps most important where cooling of the target organ requires cooling of the whole body, and particularly when cooling is more profound. The equine distal limb offers a unique opportunity for the use of therapeutic hypothermia: the target organ (lamellar tissue) can be cooled selectively, in isolation from other tissues. The equine distal limb is also adapted to extreme environmental cold, allowing more profound local hypothermia without apparent side effects and with excellent tolerance from the horse.

Therapeutic mechanisms of hypothermia

Analgesic and hypometabolic effects

The major effects of hypothermia on tissue reported in the human literature include analgesia, reduced metabolism and a vascular response [8]. Cold has a direct effect on peripheral nerves, reducing conduction velocity, raising the threshold for stimulation and increasing the refractory period after stimulation [9]. Tissue metabolic rate and oxygen consumption are directly related to temperature [10] and this hypometabolic effect was traditionally credited as the major basis for the therapeutic effects of hypothermia.

Hypothermia has been best studied in brain tissue for its neuroprotective effects following traumatic and ischemic brain injury [11]. Cerebral metabolism decreases by 6-10% for each 1 ˚C decrease in body temperature [12-14]. A reduced requirement of cooled tissue for oxygen, glucose and other metabolites enhances the survival of cells during periods of ischemia, protecting tissue on the periphery of an injury from secondary hypoxic damage [8] and maintaining tissue viability during organ transplant surgery [15]. Hypothermia reduces apoptosis, mitochondrial failure and inflammation after cerebral ischemia-reperfusion [11]. Reductions in enzymatic activity, including that of collagenases, are observed at lower temperatures [16,17].

Anti-inflammatory effects

Regional hypothermia (cryotherapy) is well established as a treatment for reducing inflammation and providing analgesia in acute musculoskeletal injury and following orthopedic surgery in humans [8,18,19]. The array of potent anti-inflammatory effects exerted by hypothermia is also proving useful in other disease scenarios where inflammation plays a major role: both in the treatment of primary inflammatory conditions and also in protecting tissue from inflammatory damage post-ischemia. The primary anti-inflammatory effects of hypothermia demonstrated in rodent models of human disease include reduced production and activity of pro-inflammatory cytokines [7,20,21], increased production of anti-inflammatory cytokines (particularly IL-10) [7,22], reduced adhesion and extravasation of leukocytes from the microvasculature into affected tissues [23,24], and reduced production of free radicals of oxygen or reactive oxygen species (ROS) by polymorphonuclear leukocytes [25]. In addition, hypothermia had an inhibitory effect on matrix metalloproteinase expression (leukocyte-derived MMP-9) in studies of brain trauma [26] and cardiac arrest [27]. The anti-inflammatory effects of hypothermia are thought to play an important role in reducing the prolonged phase of inflammatory tissue damage in the brain associated with reperfusion after ischemia [28].

Preventing end-organ damage in sepsis

Of particular interest and a topic of much recent research is the potential for hypothermia to protect against end organ damage during systemic inflammatory conditions including sepsis. Mild, whole body hypothermia (5-6˚ C below normal) has been shown to increase survival in rodent models of sepsis [5,29] and the experimental evidence is prompting discussion of clinical application of therapeutic hypothermia in septic human patients [30,31]. Preservation of cellular energy metabolism and mitochondrial function [32], reduced inflammation [6,7,22,33] and inhibition of apoptotic pathways [34] are effects of hypothermia that are proposed to protect against end-organ damage in the liver, lung and heart. Pre-emptive hypothermia (10˚C below normal) markedly reduced the severity of acute lung injury in a rat model of sepsis by reducing neutrophil emigration, inhibiting pro-inflammatory cytokine activity and increasing anti-inflammatory cytokine activity [7]. In a subsequent study, less profound hypothermia (5˚C below normal) applied after neutrophilic inflammation was induced in the lung decreased the severity of acute lung injury, suggesting a potential therapeutic role for hypothermia beyond its preventive effect [6].

Vascular effects

Hypothermia in most tissues generally results in local vasoconstriction [35]. This is largely mediated by sympathetic nervous control; however, a direct constrictive effect on blood vessel walls may occur, particularly when tissue temperature approaches 0˚C [36]. A scintigraphic study demonstrated a significant reduction in soft tissue perfusion when the equine digit was immersed in 4°C iced water for a short period of 30 min [37]. Based on hoof temperature measurement, digital vasoconstriction appears to predominate when distal limb cryotherapy is applied continuously for longer periods [38-41] and when horses are maintained in controlled, low ambient temperature (10 ºC) environments [42]; however intermittent, spontaneous increases in hoof temperature up to 12 ºC during cold water immersion at 5 ºC and up to 25 ºC in a 10 ºC climate controlled room were noted in 4/6 and 4/8 horses in respective studies [41,42]. These 1 to 12 h periods of increased hoof temperature can occur synchronously or asynchronously in the forelimbs (Figure 1.). The phenomenon is similar to that noted in horses living in natural environments below freezing (Chris Pollitt BVSc, PhD, St Lucia, Queensland, unpublished data). The function or reason for this periodic hoof temperature increase in the face of environmental or local hypothermia is unclear. The increases in hoof temperature most likely represent transient increases in perfusion, metabolism or both [43], and may be associated with opening of arteriovenous anastomoses (AVAs). The fact that these periods are not reliably synchronous suggests that the trigger is a local phenomenon, possibly local tissue temperature or accumulation of metabolic intermediates. r In other tissues, periods of transient vasodilation (cold-induced vasodilation [CIVD]) are observed when temperatures are reduced below approximately 18°C [44]. A cyclic pattern of increasing and decreasing blood flow (the hunting reaction) may be noted [45]. The phenomenon has been studied mostly in the human hand, however CIVD has also been noted in the face, forearms and feet [46]. In the human finger, CIVD appears to occur as a result of dilation of the AVAs, whereas cold-induced vasoconstriction appears to occur as a result of constriction of AVAs as well as the arteries supplying the finger [47].

Rationale and evidence for digital hypothermia in laminitis

Our understanding of the pathophysiology of laminitis has evolved dramatically in recent years, as has our understanding of the mechanisms by which hypothermia can alter disease progression. Eighteenth century references to the usage of digital hypothermia for the treatment of laminitis have been cited by Wagner [48] but it was the study of Pollitt and Davies [42] that prompted scientific evaluation of its effects. This study of carbohydrate-induced laminitis, performed in a climate controlled room (10ºC), demonstrated increased hoof temperature (indicative of vasodilation) during the developmental phase in horses that went on to develop laminitis compared with those that did not, prompting the hypothesis that digital vasoconstriction might be protective. At the time of that study, vascular changes (vasoconstriction and ischemia) were thought to be major initiating events in laminitis, regardless of the inciting cause. In recent years, researchers have come to realise that there are important differences in the pathophysiology of laminitis depending on the inciting cause. This has divided laminitis into essentially three forms, based upon the purported basic mechanisms of disease

(sepsis, hyperinsulinemia/endocrinopathy and supporting limb laminitis). The mechanisms that lead to the different forms of laminitis are now better defined (see relevant chapters) allowing more informed exploration of the opportunities for therapeutic intervention with hypothermia in the clinical setting.

Studies evaluating the effects of digital hypothermia on the development of laminitis have been performed exclusively in the oligofructose (OF) experimental laminitis model. These studies have utilised ice and water or refrigerated water immersion of the distal limb leading to internal hoof temperatures of 3-8 ºC, or a 20˚C or more decrease below normal foot temperature. This is dramatically more profound than the 3-5˚C reduction in body temperature that is commonly targeted for whole body hypothermia used in human medicine for a variety of disease processes [1,11]. In an initial study in which hypothermia was initiated at the time of OF administration, continuous cooling of a single limb during the developmental phase dramatically reduced both the severity of clinical signs of lameness and the severity of laminar injury (on histopathology) when evaluated 48 h after the OF dose [39]. That study proved the principle of prophylactic hypothermia as a viable preventative treatment for animals at risk of sepsis-related laminitis. A follow-up study showed that digital hypothermia of all four limbs maintained for 72 hours after OF dosing resulted in reduced severity of lameness and laminar injury evaluated 4 days after cooling ceased [41]. This study demonstrated that the prophylactic effect of digital hypothermia does not merely delay the appearance of laminitis pathology, but rather has a lasting effect. In both studies, pathologic changes to the lamellae were not completely absent in the hypothermic limbs, but were ameliorated to an extent that would translate into a meaningful clinical effect. The current evidence for the use of therapeutic digital hypothermia in laminitis is based solely on these controlled experimental studies with small treatment and control groups (6 to 8 horses): what remained lacking until recently was a clinical trial evaluating the effects of digital hypothermia in the clinical setting. Kullman *et al.* published a multicentre retrospective case series of 130 colitis cases, with an overall acute laminitis incidence of 21% [49]. Horses treated with digital hypothermia in that study had 10 times less odds of developing laminitis compared with horses that did not have their feet cooled, demonstrating that prophylactic cooling is an effective laminitis preventative in clinical colitis cases.

There are several possible mechanisms by which hypothermia protects lamellar tissue from damage and reduce the progression of laminitis in the OF model of sepsis-related laminitis [50-52]. Inflammation and protease activity are implicated in the developmental as well as acute phases of sepsis-related laminitis [53-59]. When applied before the onset of clinical lameness in the OF model, continuous digital hypothermia dramatically inhibited transcription of inflammatory mediators including cytokines, chemokines and cyclooxygenase in lamellar tissue [60]. In addition, hypothermia had an inhibitory effect on lamellar matrix metalloproteinase expression in the OF model [39], a finding consistent with experimental studies of hypothermia in brain trauma [26] and cardiac arrest [27]. In other animal models of sepsis, preservation of cellular energy metabolism and mitochondrial function [32], reduced inflammation [6,7,22,33] and inhibition of apoptotic pathways [34] are effects of hypothermia that protect against end-organ damage in the liver, lung and heart. The contribution of cellular energy failure (non-ischaemic) to the different forms of laminitis is

unclear, but hypothermia of the equine digit may exert protective effects on lamellar tissue by modulating mitochondrial function and reducing metabolic demands.

It is unclear whether digital hypothermia would have a preventative effect on the development of supporting limb or endocrinopathic laminitis, as in the clinical setting both these forms have a developmental phase that is much less predictable than that associated with the septic form, with little evidence of the marked lamellar inflammatory signaling documented in sepsis-related laminitis. However, inflammation, protease activation and pain are features of the acute stage of laminitis, regardless of the inciting cause. It is therefore rational to expect that digital hypothermia might have beneficial effects in acute laminitis, once lameness is already evident. Due to the concern that veterinarians in clinical practice often do not attend cases until the animal is already demonstrating clinical signs of lameness, a recent study was performed in which digital hypothermia was not initiated until after the onset of lameness in the OF model. In this study, a profound therapeutic effect was still observed with digital hypothermia [61], with only mild to undetectable lesions present in the hypothermic limb (Figure 2) in the face of total lamellar failure (complete separation of lamellar epidermis from dermis; “sinking”) in untreated control limbs. As the goal in the treatment of acute laminitis should be to reduce the progression of the lesion (lamellar separation and ultimately mechanical failure of the suspensory apparatus of the distal phalanx), this study documented that digital hypothermia instituted at the onset of clinically apparent lameness is an effective therapy. Digital hypothermia also exhibited analgesic effects in that study, which would clearly be beneficial in the acute laminitis patient.

Digital hypothermia in clinical cases

Case selection

Once established, laminitis causes derangements of lamellar structure that are largely irreversible; therefore efforts should be aimed at prevention. It follows that digital hypothermia is likely to be most effective prior to the onset of clinical signs; however this is often not possible or practical in the clinical setting. Any horse that is at immediate risk of developing acute laminitis is an appropriate candidate for continuous distal limb hypothermia. Conditions associated with a high risk of laminitis development include colitis, enteritis, complicated gastrointestinal obstructions, metritis, pneumonia, and alimentary carbohydrate (grain) overload [62-66]. Horses exhibiting clinical signs consistent with endotoxemia should be considered at particularly high risk [62]. The recent study described above suggests that digital hypothermia is also indicated in acute laminitis [61]. Regardless of the inciting cause, digital hypothermia will provide analgesia and may help to reduce lesion progression in horses with acute laminitis (or chronic laminitis with a recrudescent, acute episode).

Timing and duration of treatment

The ideal target period to initiate treatment is the developmental phase, defined as the period between the initial causative event and the appearance of acute lameness. The duration of the developmental phase is highly variable, lasting between 18 and 72 hours in carbohydrate overload models, up to 5 days in clinical colitis, enteritis and metritis cases, or longer with other diseases such as pleuropneumonia. The difficulty in identifying the developmental phase (current lack of any suitable biomarker) means that the perceived risk of laminitis and the logistics of application (constant digital hypothermia is a very work intensive therapy for owners/technicians) will govern the decision to treat prophylactically in most cases. Resolution of the primary disease may be used as an indicator for ceasing digital hypothermia in individual cases. It is prudent to continue therapy for 24-48 hours after the resolution of clinical signs of sepsis (fever, mucus membrane hyperemia) and normalization of laboratory indicators (leukopenia and perhaps serum amyloid A concentration) of systemic inflammation.

When treating acute cases of laminitis, it is rational to use digital hypothermia as part of a first aid strategy. Cooling for 2-3 days or more may be warranted in the acute laminitis case, although there is currently no evidence to guide cessation of therapy in these cases. Anecdotal evidence from clinical cases suggests that digital hypothermia can be maintained for days to weeks without ill effect. There is no evidence to support the use of intermittent cooling, and all experimental studies have thus far have used continuous digital hypothermia. Work in other species suggests that (ideally) rewarming should be gradual (over 12-24 hours) as rapid rewarming after therapeutic hypothermia may negate the protective effect [11]. However, in most clinical cases, cryotherapy is ceased without gradual rewarming, without apparent negative effects.

Cooling methods

To maximize the clinical effect and minimize the side effects, therapeutic hypothermia should be targeted as much as possible to the tissue of interest. The equine distal limb presents a unique opportunity: distal to the carpus and tarsus, there is no muscle and the major blood vessels are superficial, seemingly ideal for inducing deep hypothermia of the foot. However, the hair coat and hoof provide a barrier to effective conduction of thermal energy in and out of the limb, and blood flowing through the major arteries constantly replenishes the rich vascular network within the corium with warmed (or cooled) blood. The numerous lamellar arteriovenous anastomoses (AVAs) [67], typical of a thermoregulatory organ, mean that rapid increases in net perfusion of the foot with warm blood can occur.

Numerous modalities are used for distal limb cryotherapy in horses, and there are several commercially available devices for this purpose, although most are suited to short term (30-60 min) applications. Published studies focused on limb structures other than the foot have used commercial cold gel wraps and cold cuffs as well as ice water immersion [68,69]. In order to achieve consistently low temperatures within the target (lamellar) tissue,

the requirements for cooling methods appear to differ significantly from those most commonly used for distal limb tendon/skeletal cooling.

Studies in experimental models of laminitis have shown a preventative effect when hoof temperatures (measured by a probe embedded in the hoof wall or on the hoof surface) were maintained consistently at 3-5 C using cold water alone or ice and water slurries immersing the limb up to just below the carpus. This may represent a 20˚C or more decrease below normal foot temperature (depending on the ambient temperature foot response at the time). When applied after the onset of lameness in the OF laminitis model, the same method resulted in hoof wall surface temperatures of 6-8 C, and still achieved a significant therapeutic effect. Recent human medical-related studies suggest a superior effect when mild to moderate (3-5˚C reduction) therapeutic hypothermia is used compared with traditional, more profound (>5˚C reduction) hypothermia in a variety of disease processes [1,11]; however this may be due mostly to reduced side effects at warmer temperatures, particularly with whole body hypothermia. A critical temperature for laminitis prevention and treatment has not been established, although it is possible that even mild decreases in lamellar temperature have some beneficial effect.

Based on currently available data, clinical distal limb cryotherapy should be aimed at achieving hoof wall surface temperatures that are (at least) consistently below 10˚C. In order to effectively cool the lamellae, it appears that the hoof and its solar surface as well as the pastern, fetlock and cannon region must be included to cool incoming arterial blood. A water interface against the limb overcomes the conduction barrier of the hair coat and negates the difficulty in getting consistent contact of a heat exchanger with the uneven surface of the equine distal limb. Immersion of the limb from the upper metacarpus/metatarsus distally in an ice and water mixture effectively achieves this, though constant ice replenishment is labor intensive. In a recent study, temperature was measured directly in the lamellar tissue and the digital vein during cryotherapy application to compare three different cooling methods [40]. Results indicated that immersion in an ice and water slurry to the level of the upper pastern (using a 5 L fluid bag) or the mid to upper cannon (using a wader style vinyl boot) achieved rapid cooling to around 11 ºC (approximately 24 ºC decrease) and the two methods were not significantly different over the 2 hour treatment period. Application methods that use gel or ice packs wrapped around the distal limb/digit tend to perform poorly, even when replaced as frequently as every 30 minutes [70]. A summary of the efficacy of different methods currently available for digital hypothermia is shown in Figure 3.

Although laminitis is more common and more severe in the forelimbs, it is important to cool hindlimbs as well as forelimbs in cases at risk. The authors have received specimens and reports from clinical cases where laminitis has developed exclusively in the uncooled hindlimbs when cryotherapy was only applied to the forelimbs. A tub, 200 cm long, 80 cm wide and 50 cm high, as used in a previous study [41], offers a practical method for prolonged, continuous application of cryotherapy to all four limbs that can be used clinically. A water-tight door at one end for ease of access, and a rubber floor are suggested. Temporary or permanent stocks, together with cross-tying the head may assist in keeping the horse stationary. A refrigerated pump, recirculating water at around

1-2°C, can reduce or replace the requirement for ice (Figure 3). The disadvantages of this system are the inability of the horse to lie down, the need for constant supervision, lack of portability and potential biosecurity issues (particularly in colitis cases). A system capable of cooling all four limbs effectively, that allows ambulation and is linked to a refrigeration system would be ideal for hospitals and requires development. Most of the currently available cooling cuff devices made for the equine distal limb are either untested or unable to reduce temperatures within the digit to the desired levels for laminitis prevention without major modification.

Side effects and contraindications

The equine distal limb appears resilient in the face of extreme, continuous hypothermia: there are no reports in the literature of complications directly related to therapeutic hypothermia of the equine distal limb and no adverse effects are reported in polar climates where the distal limbs of horses and ponies are continuously immersed in snow and sub-zero ambient temperatures. Cold-induced pain, observed in human patients when cryotherapy is applied at 5°C or less [19], is not observed in horse studies. Adverse effects of local therapeutic hypothermia in people are rare but may include frostbite and nerve palsy [8]. The temperatures and duration of exposure required to induce frostbite are unclear. Nerve palsy in humans usually involves large superficial nerves and it may be that compression (often used in association with cryotherapy in human patients) contributes to nerve palsy. Prolonged exposure to cold and moisture (days to weeks) is associated with the development of 'immersion foot' and 'trench foot' in human patients [71]. Local swelling and pain are features of these conditions, which may progress to blistering of the skin, nerve damage and gangrene [72]. There are anecdotal reports of a similar phenomenon in horses being treated with digital hypothermia: dermatitis of the pastern and maceration of the coronary band can occur particularly when cooling for more than 5 days, but in most cases this resolves with no or minimal treatment once the cooling is ceased. On rare occasions the dermatitis progresses to more severe necrosis of the skin (Figure 4.). Profound whole body hypothermia (more than 10˚C below normal) can result in severe side effects associated with cardiac, endocrine and metabolic function [11]. Hypothermia has also been associated with coagulopathy and increased risk of infection [11]. The association between whole body hypothermia and reduced ability to clear infection is based upon the results of one rodent study in the 1960s [73]. More recent studies have not demonstrated adverse effects of hypothermia on bacterial growth in rodent models of sepsis [32] and the benefits of hypothermia are now thought to outweigh the risks in sepsis [31]. It is difficult to extrapolate the results of these studies to the profound local hypothermia achieved in the digit in horses and it may be prudent to avoid this for prolonged periods in cases where laminitis is associated with infection within the foot (eg. laminitis accompanied by sub-solar or sub-mural abscessation, septic osteitis or seedy toe)..

1. Varon J, Acosta P. Therapeutic hypothermia: past, present, and future. *Chest* 2008;133:1267-1274.
2. Crossley S, Reid J, McLatchie R, et al. A systematic review of therapeutic hypothermia for adult patients following traumatic brain injury. *Critical care* 2014;18:R75.
3. Rossouw G, Irlam J, Horn AR. Therapeutic hypothermia for hypoxic ischaemic encephalopathy using low-technology methods: a systematic review and meta-analysis. *Acta paediatrica* 2014.
4. Xiao G, Guo Q, Shu M, et al. Safety profile and outcome of mild therapeutic hypothermia in patients following cardiac arrest: systematic review and meta-analysis. *Emergency medicine journal : EMJ* 2013;30:91-100.
5. Rim KP, Kim K, Jo YH, et al. Effect of therapeutic hypothermia according to severity of sepsis in a septic rat model. *Cytokine* 2012;60:755-761.
6. Chin JY, Koh Y, Kim MJ, et al. The effects of hypothermia on endotoxin-primed lung. *Anesthesia and analgesia* 2007;104:1171-1178, tables of contents.
7. Lim CM, Kim MS, Ahn JJ, et al. Hypothermia protects against endotoxin-induced acute lung injury in rats. *Intensive Care Med* 2003;29:453-459.
8. Swenson C, Sward L, Karlsson J. Cryotherapy in sports medicine. *Scand J Med Sci Sports* 1996;6:193-200.
9. Douglas WW, Malcom JL. The effect of localized cooling on mammalian muscle spindles. *J Physiol* 1955;130:53-71.
10. Fuhrman GJ, Fuhrman, F.A. Oxygen consumption of animals and tissues as a function of temperature. *J Gen Physiol* 1959;42:715-722.
11. Polderman KH. Mechanisms of action, physiological effects, and complications of hypothermia. *Crit Care Med* 2009;37:S186-202.
12. Erecinska M, Thoresen M, Silver IA. Effects of hypothermia on energy metabolism in Mammalian central nervous system. *Journal of cerebral blood flow and metabolism : official journal of the International Society of Cerebral Blood Flow and Metabolism* 2003;23:513-530.
13. Hagerdal M, Harp J, Nilsson L, et al. The effect of induced hypothermia upon oxygen consumption in the rat brain. *J Neurochem* 1975;24:311-316.
14. Palmer C, Vannucci RC, Christensen MA, et al. Regional cerebral blood flow and glucose utilization during hypothermia in newborn dogs. *Anesthesiology* 1989;71:730-737.
15. Griepp RB, Stinson EB, Shumway NE. Profound local hypothermia for myocardial protection during open-heart surgery. *J Thorac Cardiovasc Surg* 1973;66:731-741.
16. Harris ED, Jr., McCroskery PA. The influence of temperature and fibril stability on degradation of cartilage collagen by rheumatoid synovial collagenase. *N Engl J Med* 1974;290:1-6.
17. Zachariassen KE. Hypothermia and cellular physiology. *Arctic Med Res* 1991;50 Suppl 6:13-17.
18. Knight KL. Metabolism and inflammation In: Knight KL, ed. *Cryotherapy in sports injury management*. Champaign, IL: Human Kinetics, 1995;77-84.

19. Ohkoshi Y, Ohkoshi M, Nagasaki S, et al. The effect of cryotherapy on intraarticular temperature and postoperative care after anterior cruciate ligament reconstruction. *Am J Sports Med* 1999;27:357-362.
20. Westermann S, Vollmar B, Thorlacius H, et al. Surface cooling inhibits tumor necrosis factor-alpha-induced microvascular perfusion failure, leukocyte adhesion, and apoptosis in the striated muscle. *Surgery* 1999;126:881-889.
21. Webster CM, Kelly S, Koike MA, et al. Inflammation and NFkappaB activation is decreased by hypothermia following global cerebral ischemia. *Neurobiol Dis* 2009;33:301-312.
22. Scumpia PO, Sarcia PJ, Kelly KM, et al. Hypothermia induces anti-inflammatory cytokines and inhibits nitric oxide and myeloperoxidase-mediated damage in the hearts of endotoxemic rats. *Chest* 2004;125:1483-1491.
23. Prandini MN, Neves Filho A, Lapa AJ, et al. Mild hypothermia reduces polymorphonuclear leukocytes infiltration in induced brain inflammation. *Arq Neuropsiquiatr* 2005;63:779-784.
24. Kamler M, Goedeke J, Pizanis N, et al. In vivo effects of hypothermia on the microcirculation during extracorporeal circulation. *Eur J Cardiothorac Surg* 2005;28:259-265.
25. Novack TA, Dillon MC, Jackson WT. Neurochemical mechanisms in brain injury and treatment: a review. *J Clin Exp Neuropsychol* 1996;18:685-706.
26. Jia F, Pan YH, Mao Q, et al. Matrix metalloproteinase-9 expression and protein levels after fluid percussion injury in rats: the effect of injury severity and brain temperature. *Journal of neurotrauma* 2010;27:1059-1068.
27. Hastbacka J, Tiainen M, Hynninen M, et al. Serum matrix metalloproteinases in patients resuscitated from cardiac arrest. The association with therapeutic hypothermia. *Resuscitation* 2012;83:197-201.
28. Gonzalez-Ibarra FP, Varon J, Lopez-Meza EG. Therapeutic hypothermia: critical review of the molecular mechanisms of action. *Frontiers in neurology* 2011;2:4.
29. Chang YT, Wann SR, Tsai JS, et al. The role of autonomic nervous system function in hypothermia-mediated sepsis protection. *The American journal of emergency medicine* 2013;31:375-380.
30. Crouser ED. Warming up to hypothermia for treatment of severe sepsis*. *Critical care medicine* 2012;40:1020-1022.
31. Acosta-Lara P, Varon J. Therapeutic hypothermia in sepsis: to use or not to use? *The American journal of emergency medicine* 2013;31:381-382.
32. Beurskens CJ, Aslami H, Kuipers MT, et al. Induced hypothermia is protective in a rat model of pneumococcal pneumonia associated with increased adenosine triphosphate availability and turnover*. *Critical care medicine* 2012;40:919-926.
33. Fujimoto K, Fujita M, Tsuruta R, et al. Early induction of moderate hypothermia suppresses systemic inflammatory cytokines and intracellular adhesion molecule-1 in rats with caerulein-induced pancreatitis and endotoxemia. *Pancreas* 2008;37:176-181.
34. Lee JH, Kim K, Jo YH, et al. Therapeutic hypothermia attenuates liver injury in polymicrobial sepsis model of rats via Akt survival pathway. *The Journal of surgical research* 2013;181:114-120.
35. Lehmann JF, de Lateur, B.J.. Cryotherapy In: Lehmann JF, ed. *Therapeutic heat and cold*. 4th ed. Baltimore: Williams & Wilkins, 1990;590-631.

36. Perkins JF, I MC, F H. Sudden vasoconstriction in denervated or sympathectomized paws exposed to cold. *Am J Physiol* 1948;155:165-178.
37. Worster AA, Gaughan EM, Hoskinson JJ, et al. Effects of external thermal manipulation on laminar temperature and perfusion scintigraphy of the equine digit. *New Zealand Veterinary Journal* 2000;48:111-116.
38. Pollitt CC, van Eps AW. Prolonged, continuous distal limb cryotherapy in the horse. *Equine Vet J* 2004;36:216-220.
39. van Eps AW, Pollitt CC. Equine laminitis: cryotherapy reduces the severity of the acute lesion. *Equine Vet J* 2004;36:255-260.
40. Reesink HL, Divers TJ, Bookbinder LC, et al. Measurement of digital laminar and venous temperatures as a means of comparing three methods of topically applied cold treatment for digits of horses. *Am J Vet Res* 2012;73:860-866.
41. van Eps AW, Pollitt CC. Equine laminitis model: Cryotherapy reduces the severity of lesions evaluated seven days after induction with oligofructose. *Equine Vet J* 2009;41:741-746.
42. Pollitt CC, Davies CT. Equine laminitis: its development coincides with increased sublamellar blood flow. *Equine Vet J Suppl* 1998;26:125-132.
43. Hood DM, Wagner IP, Brumbaugh GW. Evaluation of hoof wall surface temperature as an index of digital vascular perfusion during the prodromal and acute phases of carbohydrate-induced laminitis in horses. *Am J Vet Res* 2001;62:1167-1172.
44. Clarke RSJ, Hellon, R.F., Lind, A.R. Vascular reactions of the human forearm to cold. *Clinical Science* 1958;17:165-179.
45. Lewis T. Observations upon the reactions ofthe vessels of the human skin to cold. *Heart* 1930;15:177-208.
46. Daanen HA. Finger cold-induced vasodilation: a review. *European journal of applied physiology* 2003;89:411-426.
47. Bergersen TK, Hisdal J, Walloe L. Perfusion of the human finger during cold-induced vasodilatation. *Am J Physiol* 1999;276:R731-737.
48. Wagner IP, Heymering H. Historical perspectives on laminitis. *Vet Clin North Am Equine Pract* 1999;15:295-309.
49. Kullmann A, Holcombe SJ, Hurcombe SD, et al. Prophylactic digital cryotherapy is associated with decreased incidence of laminitis in horses diagnosed with colitis. *Equine Vet J* 2014;46:554-559.
50. Pollitt CC, Visser MB. Carbohydrate alimentary overload laminitis. *Vet Clin North Am Equine Pract* 2010;26:65-78.
51. van Eps AW, Pollitt CC. Equine laminitis induced with oligofructose. *Equine Vet J* 2006;38:203-208.
52. Bailey SR, Adair HS, Reinemeyer CR, et al. Plasma concentrations of endotoxin and platelet activation in the developmental stage of oligofructose-induced laminitis. *Vet Immunol Immunopathol* 2009;129:167-173.
53. Belknap JK, Giguere S, Pettigrew A, et al. Lamellar pro-inflammatory cytokine expression patterns in laminitis at the developmental stage and at the onset of lameness: innate vs. adaptive immune response. *Equine Vet J* 2007;39:42-47.
54. Blikslager AT, Yin C, Cochran AM, et al. Cyclooxygenase expression in the early stages of equine laminitis: a cytologic study. *J Vet Intern Med* 2006;20:1191-1196.

55. Johnson PJ, Tyagi SC, Katwa LC, et al. Activation of extracellular matrix metalloproteinases in equine laminitis. *Vet Rec* 1998;142:392-396.
56. Leise BS, Faleiros RR, Watts M, et al. Laminar inflammatory gene expression in the carbohydrate overload model of equine laminitis. *Equine Vet J* 2011;43:54-61.
57. Coyne MJ, Cousin H, Loftus JP, et al. Cloning and expression of ADAM-related metalloproteases in equine laminitis. *Vet Immunol Immunopathol* 2009;129:231-241.
58. Visser MB, Pollitt CC. The timeline of metalloprotease events during oligofructose induced equine laminitis development. *Equine Vet J* 2012;44:88-93.
59. Visser MB, Pollitt CC. Lamellar leukocyte infiltration and involvement of IL-6 during oligofructose-induced equine laminitis development. *Vet Immunol Immunopathol* 2011;144:120-128.
60. van Eps AW, Leise BS, Watts M, et al. Digital hypothermia inhibits early lamellar inflammatory signalling in the oligofructose laminitis model. *Equine Vet J* 2012;44:230-237.
61. van Eps AW, Pollitt CC, Underwood C, et al. Continuous digital hypothermia initiated after the onset of lameness prevents lamellar failure in the oligofructose laminitis model. *Equine Vet J* 2014;46:625-630.
62. Parsons CS, Orsini JA, Krafty R, et al. Risk factors for development of acute laminitis in horses during hospitalization: 73 cases (1997-2004). *J Am Vet Med Assoc* 2007;230:885-889.
63. Cohen ND, Parson EM, Seahorn TL, et al. Prevalence and factors associated with development of laminitis in horses with duodenitis/proximal jejunitis: 33 cases (1985-1991). *J Am Vet Med Assoc* 1994;204:250-254.
64. Cohen ND, Woods AM. Characteristics and risk factors for failure of horses with acute diarrhea to survive: 122 cases (1990-1996). *J Am Vet Med Assoc* 1999;214:382-390.
65. Slater MR, Hood DM, Carter GK. Descriptive epidemiological study of equine laminitis. *Equine Vet J* 1995;27:364-367.
66. Alford P, Geller S, Richrdson B, et al. A multicenter, matched case-control study of risk factors for equine laminitis. *Prev Vet Med* 2001;49:209-222.
67. Pollitt CC, Molyneux GS. A scanning electron microscopical study of the dermal microcirculation of the equine foot. *Equine Vet J* 1990;22:79-87.
68. Kaneps AJ. Tissue temperature response to hot and cold therapy in the metacarpal region of a horse. Proceedings of the Fourty-sixth Annual Convention of the American Association of Equine Practitioners 2000;208-213.
69. Turner TA, Wolfsdorf K, Jourdenais J, et al. Effects of heat, cold, biomagnets and ultrasound on skin circulation in the horse. Proceedings of the Thirty-Seventh Annual Convention of the American Association of Equine Practitioners 1992;249-257.
70. van Eps AW, Orsini JA. A comparison of seven methods for continuous therapeutic cooling of the equine digit. *Equine Vet J* 2014.
71. DeGroot DW, Castellani JW, Williams JO, et al. Epidemiology of U.S. Army cold weather injuries, 1980-1999. *Aviation, space, and environmental medicine* 2003;74:564-570.
72. Ungley CC, Channell GD, Richards RL. The immersion foot syndrome. 1946. *Wilderness & environmental medicine* 2003;14:135-141; discussion 134.

73. Webb WR, Deguzman VC, Grogan JB, et al. Hypothermia: its effects upon hematologic clearance in experimentally induced staphylococcal bacteremia. *Surgery* 1962;52:643-647.

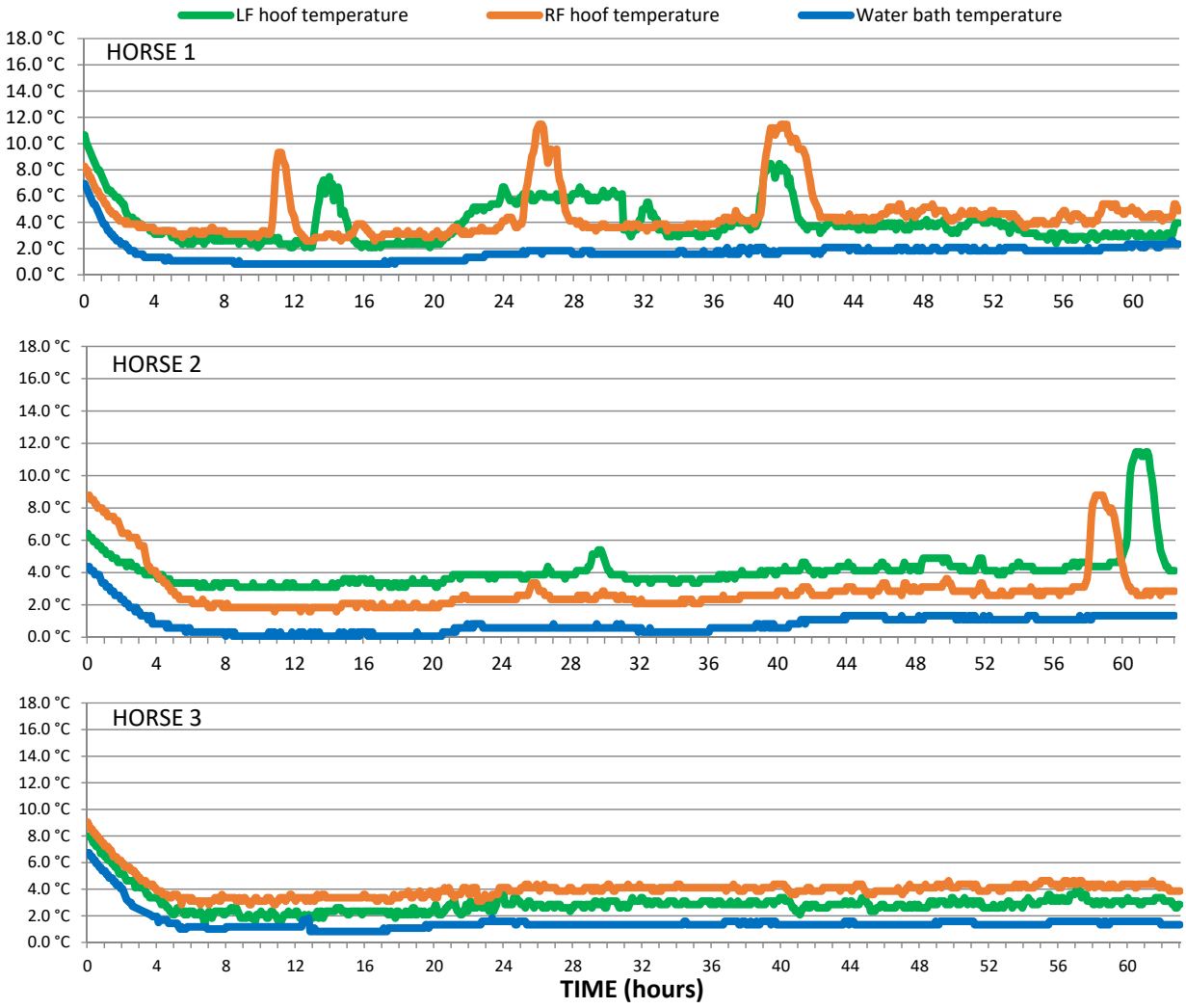


Figure 1. : Hoof temperature traces from both front limbs three horses during cold water immersion of all four feet over a 3 day period. Intermittent, spontaneous increases in hoof temperature are noted regularly in some horses (Horse 1), rarely in others (horse 2) and not at all in some horses (Horse 3). These 1 to 12 h periods of increased hoof temperature occur sometimes synchronously in the forelimbs, and sometimes asynchronously and are also seen in horses living in natural environments below freezing (Chris Pollitt BVSc, PhD, St Lucia, Queensland, unpublished data). The increases in hoof temperature may be associated with opening of arteriovenous anastomoses (AVAs). The fact that these periods are not reliably synchronous suggests that the trigger is a local phenomenon, possibly local tissue temperature or accumulation of metabolic intermediates (e.g. lactate).

Figure 2. Digital hypothermia can protect lamellar tissue from complete separation/mechanical failure. The top images represent the histological appearance of an uncooled hind limb (A) and a cooled forelimb (B) from the same horse: a clinical case of colitis that was euthanized due to the development of laminitis in the uncooled hind limbs. A similar effect has been shown in experimental models, even when cooling is delayed until after the onset of laminitis: complete separation of lamellar epidermis is noted in the uncooled forelimb (C) compared with almost no appreciable structural change in the cooled limb (D) (VAN EPS AND BELKNAP, SUBMITTED).

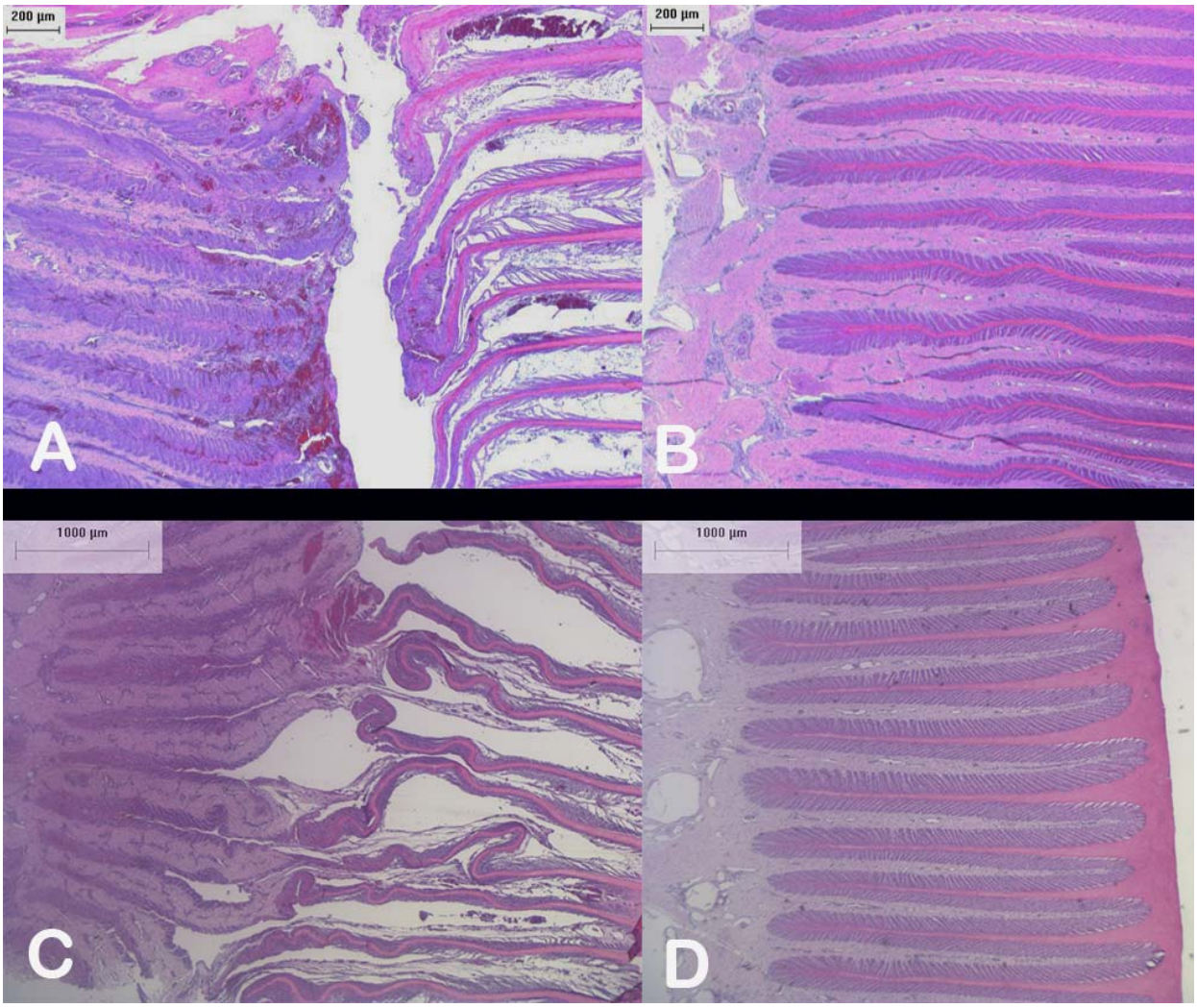
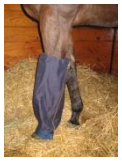
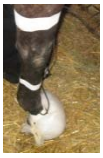
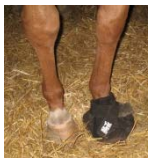
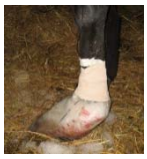


Figure 3. Comparison of several different methods used for digital hypothermia over an 8 hour study period. Where ice packs and crushed or cubed ice used, this was replenished every 30 minutes. Hoof temperature was measured using probes embedded in the dorsal hoof wall. Methods with a water interface that include the distal limb and foot achieved lower hoof temperature (Andrew van Eps, unpublished data)

	Method	Mean hoof temperature (˚C)	Mean reduction from control group (˚C)
	Ice boot distal limb (not including foot)	**25.7**	**6.6**
	Frozen gel pack wrap distal limb (including foot)	**19.8**	**12.5**
	Rectal sleeve tied around coronet filled with crushed ice	**17.9**	**14.4**
	Frozen gel pack boot (foot only)	**16.2**	**16.1**
	5 L Fluid bag filled with ice	**5.5**	**26.8**
	Water bath to upper metacarpus (1 ºC refrigerated water, all 4 limbs)	**3.9**	**28.6**

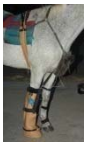
	Ice boot filled with ice and water to upper metacarpus	**2.7**	**29.6**

Figure 4. Complications from digital hypothermia in horses are extremely rare, although with longer application (>5 days) there are anecdotal reports of dermatitis and coronary band maceration. Occasionally, severe dermatitis can progress to necrosis of skin and deeper structures. This photograph depicts a lesion on the dorsal metacarpal region of a gelding that had been treated for colitis and had digital hypothermia applied for 9 days to all four limbs using cubed ice and wader style boots. The ice boots were removed on day 9 due to developing cellulitis/dermatitis. This progressed to necrosis and required debridement of skin and part of an extensor tendon. The lesion healed eventually and the horse returned to athletic activity. The probability of this type of complication may be increased with longer application times (>5 days) and when ice is applied directly to the skin (without a water interface). Photograph courtesy of Dr Joy Tomlinson (University of Pennsylvania).