The complexity of identifying performance-limiting pain in the equine athlete

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One of the most critical things the equine clinician can do to maintain an athlete is constant reassessment and diagnosis of any low-grade chronic or acute injuries. Often time's owners will notice a horse to have either poor behavior or be not training up to its potential. These cases provide the greatest challenge to the equine practitioner. Often time's if our diagnostic results are negative we will recommend advanced imaging such as nuclear scintigraphy to identify subtle, deep lesions that can be difficult to characterize. In addition, any new lesions picked up on regular examinations should be addressed and discussed with the owner and trainer in order to characterize their significance and management.

The lameness examination is the cornerstone for appropriate diagnosis of equine musculoskeletal disease. Veterinarians need to be able to recognize if the horse is lame, which leg or legs the horse is lame in and be able to grade the degree of lameness for comparison after diagnostic analgesia is used and for assessment at follow-up examinations. However, with increasing information about disease and pain in the axial skeleton, it is becoming more and more difficult to identify the exact cause of performance limitation in the athlete. The goal of this lecture is to discuss objective changes in horses gait during lameness and clinical tools that can be used to characterize the horses' lameness. Subjective assessments of the axial skeleton will also be discussed. It is essential to understand that the lameness examination is an artistic experience acquired by years of clinical practice along with integration of the objective information that is known about lameness. Although recommendations can be made to characterize lameness, a clinician needs to develop their own set of skills that can optimize their ability to see and characterize lameness in horses. It is also important to know that obvious lameness often times may not need to be present in order to be performance limiting. Some lamenesses are not amenable to diagnostic analgesia, furthermore some subtle lamenesses although treatable may not solve the performance limiting problem.

With experience comes the ability to characterize the lameness as it relates to pain and specific body parts. Although some people can be adamant that they can characterize the site of lameness subjectively, our approach usually has been to use this as an indicator to drive diagnostic analgesia to confirm the diagnosis not in place of diagnostic anesthesia. Similar gait deficits can exist for various conditions but it is important to document those specific movements that may indicate pain in a particular area.

Documentation of the history of the lameness is important as is the expectations of the owner and trainer for the level of use in competition of the horse. It is important to document the rider's experience and any previous lamenesses and treatment that have been used.

The first phase of the lameness examination is conformation examination in which both experience and science play a role. The horse should be evaluated from all sides and the conformation of the horse should be characterized in light of their intended use.

The second phase of the lameness examination is the static examination. During this examination, hoof testers should be applied to the feet, especially around the nail holes, sole, frog and the hoof wall. However, care must be taken in remembering that hoof testers have been shown to be about 50% accurate when correlated with caudal heel pain. The hoof capsule should also be statically examined for conformation and coronary band characteristics. Wear of the shoe should also be characterized and digital pulse should be appreciated both before and after trotting. In some instances, subtle hoof pain can demonstrate a significant increase in digital pulse after trotting. Joints and tendon sheaths should also be palpated and synovial effusion should be differentiated from joint capsule or tendon sheath thickening. Significant synovial effusion can be present yet not have a clinical impact on the presenting lameness and in some cases, especially with subchondral bone disease; synovial effusion may not be present. Muscle symmetry should be appreciated and soft tissues and bones should be palpated to determine geometry and pain during palpation. Isolated pain on palpation is of value for further investigation, however, for proximal suspensory ligaments, for example, there can be significant damage at the origin without much response to palpation. Conversely, significant pain can sometimes be elicited in the proximal suspensory area without significant damage. In the latter case some have speculated that the pain response is due to stimulation of the adjacent nerves. Range of motion of the joints and limbs should be appreciated although reduction in range of motion is not always pathologic. It must be remembered that some young horses in active work can respond dramatically to passive flexion such as the fetlock joints of Thoroughbreds and the hind limbs of Western Performances horses.

The third phase of the lameness examination is the dynamic examination. Horses should be walked to evaluate hoof landing and limb movement and the stride length and the cranial and caudal phase of the stride should be characterized. A neurologic assessment should also be made during that same time. Most of the assessment of lame horses is done at a trot and in North American a 0-5 grading scale is used and in Europe a 0-10 grading scale is used. The same scales can also be used during an examination in circles and in response to flexion. The American Association of Equine Practitioners (AAEP) lameness grading scale is most typically used in the United States with grade 0 being sound; grade 1 being difficult to observed and not consistently apparent regardless of circumstances (such as weight carrying, circling, inclines and hard surfaces); grade 2 is difficult to observe at a walk or trotting a straight line but is consistently apparent under certain circumstances (such as weight carrying, circling, inclines and hard surfaces); grade 3 being consistently observable at a trot under all circumstances; grade 4 being an obvious lameness with marked head nodding, hitching or shorten stride; and a horse with a grade 5 lameness typically shows minimal weight bearing. At a trot stride characteristics should be characterized although overall stride length cannot differ between limbs or else the horse would be moving in a circle. Therefore, if a horse has a decreased cranial phase then the caudal phase makes up for the difference in both limbs. In some instances the

stride may differ in its lateral to medial motion such as horses with knee pain that typically abduct to reduce flexion in the knees. Hoof placement should be characterized as well. The hardness of the surface can stimulate a pain response variable with the site of pain within the limb. Hard ground is thought to make horses with foot, bone and joint pain worse, where soft ground is felt to make the lameness worse in a horse with soft tissue pain, including those soft tissue problems in the foot.

Flexion tests are commonly used to subjectively characterize the site of pain and the severity. The use of flexion tests has become contentious however; it is still used commonly in practice. It is impossible to isolate just one area or tissue. For instance, digital flexion can aggravate pain in the proximal suspensory ligament region, and "stifle flexion" will include the tarsus and digit to some extent. That is not to say that these flexion tests are not of some value, but rather that they should not be relied upon solely for identifying the site of lameness. Some level of worsening lameness in response to flexion is normal usually for 3-5 steps and it must be remembered that in some young athletes flexion response may be worse without that site being the primary site of pain. One method of assessing response is graded as normal, mild, moderate or severe. The lameness can also be re-scored after the flexion test.

Circling horses is often beneficial. Good control and consistent circles are needed on both hard and soft ground. Some generalizations also include: foot pain being worse on the inside of the circle and on hard ground; soft tissue pain being worse with the leg on the outside of the circle; and lameness dues to lesions on the medial side of the limb being worse with the limb on the outside of the circle. In some cases the lameness seen on the circle can be worse than baseline.

With hind limb lameness the entire pelvis typically rises before the lame limb contacts the ground. You most commonly see differences in hip motion with increased push off from the sound limb at the end of the stride. In horses with more severe lesions, grade 3 and above, the head will often go forward sometimes confusing this with forelimb lameness. Multiple limb lesions can sometimes be difficult to see at a trot. In general, horses with bilateral forelimb lameness will show a shuffling gate with short strides. Horses with bilateral hind limb lameness will appear "short strided" with almost a smooth rocking horse appearance. Horses with contralateral limb lameness will typically demonstrate a rough rocking horse lameness and horses with ipsilateral limb lameness look very similar with the head nod consistent with lameness in both limbs. However, it should be noted that horses with hind end lameness may drift away from the lame leg.

Objective measurement techniques have been used and commercial systems are currently available. The Lameness Locater® (Equinosis, Columbia, MO) has been useful to some extent in evaluating lame limbs but the sensitivity/specificity of the device for mild clinical lamenesses is in the early phases of validation.

In most cases it's best to use diagnostic analgesia to further characterize the site(s) of pain. Diagnostic analgesia is performed and then the lameness re-characterized sometime later. After diagnostic analgesia, horses should be re-examined within a few

minutes and in some cases need to be re-examined outwards of 30 minutes after. Significant migration of the local anesthetic can be observed as the duration post-block increases.

In some cases however, because of trainers schedule and the mild level of lameness trainers may ask that basic lameness examination be performed with flexion tests and possibly some basic perineural blocks however, a thorough examination may not be possible. In this case, the goal is to treat the horse and monitor its response to treatment. A further goal in using this method is to reduce the risk of further injury as much is as possible. In using this technique there is some inherent danger that an undiagnosed injury would not be appropriately treated or characterized and consideration to the risk of worsening of the injury should be made.

Diagnostic nerve and joint blocks have been used not only to identify the sight of pain in a limb, but also to confirm the significance of diagnostic imaging findings and to confirm the need for surgical intervention for a lesion. Although diagnostic nerve and joint blocks have been described for decades, and specific sites of anesthesia have been characterized with each block, there is recent evidence that various blocks may not be as specific as once believed.

The recent evidence of reduced specificity with each block comes from the possibility that 1) carbocaine can be inadvertently injected into a synovial structure; 2) carbocaine can diffuse into an adjacent structure; and 3) carbocaine can travel proximally within a neurovascular structure to desensitize other structures. Other mechanisms may exist, but regardless, results of diagnostic anesthesia should be questioned if lesions do not exist on diagnostic imaging techniques.

Foot

The equine foot can suffer from lesions within several different structures, and consequently treatment options can be different based on the site of lesion. In addition, many of these structures are within close proximity to each other, allowing for rapid diffusion of carbocaine between structures. Palmar digital nerves run in close proximity to the coffin joint and can be desensitized with with intra-articular anesthesia. Intra-articular anesthesia can also diffuse into the navicular bursa. The positive side of this is that intra-articular administration of medication can also diffuse into other structures. Diagnostic anesthesia of the palmar digital nerves can also have an unpredictable pattern of anesthesia. Although this block is meant to desensitize the palmar 1/3 of the foot, it can desensitize the sole, dorsum of the foot and the coffin joint.

Although diagnostic blocks can lack specificity, there are also lesions within the foot that are clinically significant, yet cannot be desensitized with conventional blocks. As an example, some significant lamenesses in the foot may not improve until and abaxial sesamoid, or even a low 4-point nerve block are performed. As an example, recent studies have shown that lesions within the deep digital flexor tendon are commonly seen

on MRI. Some of these lesions may be proximal enough to resist desensitization with a palmar digital nerve block.

In some instances, a palmar digital nerve block may desensitize structures proximal to the level of injection. For instance, proximal first phalangeal lesions have been desensitized with a palmar digital nerve block.
Injection of carbocaine into the digital sheath can also desensitize various structures within the foot, pastern and fetlock areas. The digital sheath is a large structure that is runs in close proximity to the navicular bursa, the coffin joint and the pastern joint.

Care must also be taken when injecting carbocaine into a joint where the injection site lies next to a neurovascular structure. For instance, the palmar pastern site for injection is near the neurovascular structure and extravasation of carbocaine from the joint can potentially desensitize the nerve in that area.

Fetlock Joint

Intra-articular administration of carbocaine can block any sites of joint pain including synovium, joint capsule, and soft tissues near the joint, such as suspensory ligament branch, intersesamoindean ligament and collateral ligaments. Carbocaine can also potentially extravasate from the injection site and desensitize the adjacent nerve. However, intra-articular anesthesia rarely desensitizes the subchondral bone. In cases of subchondral bone pain, a low 4-point block or a palmar metacarpal (forelimb) or lateral palmar (hindlimb) block must be performed.

Care must be taken in interpreting a low 4-point nerve block as the proximal metacarpal or metatarsal areas can be desensitized. Carbocaine can migrate proximally up the neurovascular bundle and partially desensitize the area. In most cases, a lameness in the proximal metacarpal/metatarsal areas will improve 20-30% with a low 4-point block. On rare occasions an abaxial sesamoid nerve block can also desensitize the proximal metacarpal/metatarsal regions.

Proximal Metacarpus/Metatarsus

Desensitization of the of the proximal metacarpus can be accomplished by direct infiltration of the suspensory area, however, distal outpouchings of the carpometacarpal joint can be inadvertently injected and the middle carpal joint desensitized. The lateral palmar nerve block was developed to overcome this problem, but in some cases lesions within the carpus have been desensitized.

The same is true of the hindlimb, where infiltration of the proximal suspensory area can inadvertently infiltrate the lower hock joints. Desensitization of the deep branch of the lateral plantar nerve was thought to be more specific, however, carbocaine injected into this site can infiltrate the tarsometatarsal joint.

Carpus

Injection of carbocaine in the carpal joints can desensitize primary joint lesions, however, similar to the fetlock joint, subchondral bone lesions may not be desensitized. The distal outpouchings of the carpometacarpal joint, and its communication with the middle carpal joint, can potentially allow intra-articular carbocaine to desensitize the proximal metacarpal area.

The carpal canal is a large structure that spans the caudal aspect of the carpus from the distal 1/3 of the radius to the proximal 1/3 of the third metacarpal bone. Consequently, lesons within this area can be desensitized with intrathecal administration of carbocaine.

Tarsus

Intra-articular injection of carbocaine into the tarsometatarsal and distal intertarsal joints can potentially diffuse into the proximal suspensory area, or inadvertently block the deep branch of the lateral plantar nerve. The proximal intertarsal and tibiotarsal joints communicate in most instances and both can be desensitized by injection in each.

Stifle

In some instances intra-articular anesthesia of the stifle joints can either be nonresponsive (for instance with subchondral bone lesions) or can make a lameness worse. The exact cause of the latter is unknown.

Elbow

Carbocaine can extravasate from the elbow joint and inadvertently desensitize the radial nerve, leading to signs of radian nerve paralysis.

Summary

The advancement of volumetric imaging such as MRI and CT has allowed for a more complete understanding of the effects of diagnostic anesthesia in the lower limb of horses. Consequently, without these imaging modalities, care must be taken to correlate the findings with conventional diagnostic imaging techniques such as ultrasound and radiographs. Response to treatment can also be used to assess the true diagnostic value of a nerve or joint block. Horses that fail to response to treatment should be further evaluated to identify the true site of pain.

Overall, it is best to stay consistent, determined and disciplined when evaluating the lame horse. There is often some bias in saying that a horse is better after diagnostic analgesia especially in very prolonged lameness examinations when a horse does not block out consistently. Discipline must be used to continue aggressively working up these cases to eliminate that bias. Although the literature can be useful for helping to characterize lameness, an open mind must be kept as not all horses respond the same as those in the literature. It is best that individual clinicians develop their own guidelines for management

of these cases as lameness characteristics and response to treatment can change quickly.