How to Evaluate Clinically Indistinct Gait Deficits to Differentiate Musculoskeletal and Neurological Causes

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1. Introduction
Gait deficits are commonly encountered in equine practice. They occur for a wide variety of reasons, but their effect on the performance horse is to impair its ability to safely and comfortably exercise, train, and compete to its potential. Equine veterinarians are expected to identify more and more causes of gait deficits that are produced by conditions other than osteoarthritis or appendicular inflammation and injury. Horse owners and trainers are presenting their horses for evaluation earlier in the progression of these conditions. The presentation of the horse when clinical signs are less severe requires our ability to identify the etiology of a gait deficit that may be clinically indistinct. The successful diagnosis of such gait problems is facilitated by organization and expansion of the diagnostic protocol. The suggested protocol in this abstract will assist the equine veterinarian to categorize the deficit to a body system and to subsequently pursue a specific diagnosis of the gait deficit within the affected body system. Using the proposed diagnostic protocol, an accurate diagnosis will be facilitated and earlier therapeutic intervention will produce better outcomes, even in the situation when the gait deficits are clinically indistinct or unusual.

2. Materials and Methods
Although many horses presenting with gait deficits show obvious lameness or neurological deficit with a routine examination, in performance horse practice, many or most of the horses presented for gait evaluation exhibit ambiguous clinical signs and problems. Whereas it may be common for an equine veterinarian to identify a subtle gait deficit, a reliable identification of its etiology is often much more challenging. In particular, a common problem is the difficulty in determining whether a gait deficit is caused by typical skeletal, tendinous, or ligamentous causes of lameness or a neurological deficit. In addition to these etiologies, primary and secondary muscular problems have been well identified and must now also be included in the differential diagnosis of gait deficits. Figure 1 is an algorithm suggested to assist the equine veterinarian with decisions in the clinical and diagnostic evaluation of horses with a gait deficit that does not have an obvious etiology. It has been used extensively to evaluate horses with well-defined gait deficits but has also been applied to horses that were presented specifically for evaluation of clinically indistinct and uncategorized gait problems.
Fig. 1. Algorithm suggested to assist the equine veterinarian with decisions in the clinical and diagnostic evaluation of horses with a gait deficit that does not have an obvious etiology.

**TABLE 1: GAIT DEFICIT; LAME OR NEUROLOGIC?**

1. History, signalment, complete physical examination
2. Gait Evaluation (relative to performance history) is there a deficit?
   - **NO**
     - Ophthalmic Exam
     - Evaluate Axial Skeleton Diagnostics
     - Repeat Physical Exam with Thoracic and abdominal palpation and percussion, imaging/cytology
     - Behavioral?
     - *NORMAL FINDINGS*
   - **YES**
     - **CLEARLY LAME**
     - Evaluate Lameness
     - Axial Lameness
     - Appendicular Lameness

3. **Lameness Examination**
   - **INCONCLUSIVE**
   - **Complete Neurologic Examination**
     - **NEUROLOGIC IMPAIRMENT**
       - Localize
     - **NO NEUROLOGIC IMPAIRMENT**

4. **Peripheral**
   - Muscle atrophy (denervation?)
   - EMG evaluation
   - EPM test
   - Muscle biopsy?

5. **Central**
   - Neuroanatomic Localization
   - Axial Skeleton Evaluation
   - Generalized Wasting
   - Cervical/skull/spinal Radiographs
   - CSF and Serum EPM Test
   - CSF Cytology and Protein
   - EMNED evaluation/biopsy
   - Myelogram
   - Vitamin E

6. **Exercise Test**
   - Exercise Intolerance
     - evaluate airways
     - muscle enzymes
     - muscle biopsy
     - cardiac exam
     - blood potassium electrolytes/UEB
   - Lameness
     - muscle enzymes vitamin E/selenium
     - biopsy of atrophic muscle
     - examine under saddle
     - reevaluate axial skeleton
   - **INCONCLUSIVE**
     - evaluate aorta/iliac arteries (u/s)
     - muscle enzymes/biopsy
     - nerve/joint blocks
     - re-evaluate axial skeleton
   - **Lameness Exam Under Saddle**
     - UNCLER
     - IMPROVED
     - NERVE/JOINT BLOCK
   - LAME
     - NERVE/JOINT BLOCK
     - IMPROVED

ON THE FLAT: unable to pick up lead, unable to change lead, unable to get lead behind, can’t hold lead in circles
OVER JUMPS: backs off at jump, swaps lead before jump, lands on cross canter, jumps crooked/twists

Evaluate pelvis, lumbosacral joint/ligaments, sacroiliac joints, acetabulum (ultrasound, blocks, nuclear scintigraphy) OR empirical therapy OR repeat algorithm from Lameness and/or Neurologic Examinations
When presented with the problem of an uncategorized gait deficit, it is important for the veterinarian to start by gathering information regarding the history and signalment of the horse. Although this information may seem to be irrelevant to a lameness examination, it often proves to be very important when non-appendicular causes of gait deficits are being considered. The history should include detailed information from the rider of the horse regarding any changes in behavior and performance that he/she has noticed during any exercise. Although the rider’s experience may affect the utility of this information, the details regarding what the rider experiences may be helpful to identify whether the horse is affected with a neurological or musculoskeletal problem. The breed and age of the horse also affect the prioritization of the possible diagnostics by focusing on problems that are associated with certain ages, breeds or physical characteristics (muscle disorders, OCD, arthritis, cervical myelopathy, etc.). An initial rapid gait evaluation is advisable to determine whether there is an easily detected gait deficit. This evaluation is worthwhile because gait deficits in performance horses are often subtle or imperceptible. A rapid cursory examination permits the veterinarian to get an idea whether he/she is in for a “marathon” examination or whether the lameness is one he/she feels can be identified, localized, and specifically diagnosed in a relatively short period of time. This will facilitate budgeting of time necessary to complete the examination in a thorough manner and will help keep the client from becoming frustrated. The initial cursory examination may include evaluation on various surfaces, at various gaits or speeds and in various patterns of travel (straight line, circle etc.) to demonstrate a gait deficit.

As is standard, the subsequent lameness examination includes a complete set of flexion tests and thorough palpation of each limb, as well as palpation and manipulation of the neck, back, and pelvis of the horse. Further evaluation on the surface producing the greatest exacerbation of the lameness may be necessary. Hind limb deficits seem to be the most common type of subtle lameness in performance practices. Up to 30 min, or more, of time may be spent watching horses with subtle gait deficits on lunge lines, moving in both directions, at a trot, and at the canter. Occasionally there may be a change in the gait over time. Deterioration of the gait with time may help indicate whether there is a possible muscular problem or a condition such as aortoiliac thrombosis. Some horses with chronic joint disorders may improve over the period of exercise but the deficit often does not disappear. Naturally, if lameness is identified, the next step is to attempt regional anesthesia using any number or combination of nerve and/or joint blocks deemed to be necessary. If no gait deficit is detectable by a lameness exam, the horse should receive further diagnostic evaluation to evaluate for other potential explanations of abnormal gait. Evaluations for ocular abnormalities, reduced neck and spinal mobility, muscle tremor or splinting, evidence of other diseases including pleuritis and peritonitis, and consideration of behavioral abnormalities may be worthwhile. Horses with subtle gait deficits frequently produce inconclusive findings in the lameness examination and with these other diagnostics. A gait deficit may be merely suspected but unappreciated by the examination, or it may be identified but uncharacterized because of unusual clinical signs. Clinically indistinct deficits may remain un categorized by limitations imposed by a clinical environment of the examination and/or at the gait(s) evaluated. Although the findings of a neurological evaluation are also subjective and therefore potentially unclear, at this point, a complete neurological examination is necessary to evaluate for an alternative explanation of the uncategorized gait deficit. The neurological evaluation should include examination of the entire nervous system. This means more than only a gait evaluation. It should incorporate ophthalmological examination, cranial nerve evaluation, reflex evaluation, postural evaluation with body righting, neck and spinal palpation and mobility assessment, evaluation of the gait with head elevation, obstacle avoidance, blindfolding, and gait evaluation up and down inclines. A complete neurological examination is important because gait evaluation alone, as the sole component of the neurological examination, often produces findings that are also difficult to interpret. However, the detection of another neurological deficit (in addition to a suspected neurological gait deficit) through a complete neurological examination will have significant impact on the characterization of an ambiguous gait deficit detected in either or both the lameness and neurological examinations.

If a neurological deficit is identified, it should be evaluated to determine neuroanatomical localization of the lesion or lesions. Localization must begin with determining whether a neurological problem is central or peripheral in location. Diagnostic testing should depend on this localization and the differential diagnoses for a lesion(s) in this location. Cavalier testing and interpretation of a horse for equine protozoal myeloencephalitis (EPM) should be avoided because it will often lead to treatment failure and client frustration. Clinical signs compatible with EPM are important to identify before testing for this disease. A confounding factor for assigning clinical signs of EPM is that the clinical signs attributed to EPM are extremely broad and may themselves be clinically indistinct. Because it is an infectious disease, one might expect the signs of EPM to progress with time. However, using the evaluation of disease progression as a diagnostic tool would not be an advisable means of differentiating EPM from a musculoskeletal problem or from another neurological disease that may also be progressive. Therefore, testing only horses exhibiting clinical signs compatible with a central neurological deficit is the most appropriate and reliable approach. The interpretation of results of EPM testing must be made with an understanding of local seroprevalence, the possibility of blood contamination of
a cerebrospinal fluid (CSF) sample, and the recognition that other neurological diseases may render CSF positive by damage to the blood–brain barrier.\(^1\) Positive EPM tests are often used to justify treatment of horses with subtle gait deficits. Not infrequently, the test used to justify treatment is performed on serum samples only. However, the diagnosis and treatment of horses for EPM should be recommended only when the horse shows compatible central nervous system signs, other causes have been ruled out, and both the serum and CSF are positive.\(^1\) Although treatment of a horse based solely on a positive serum test result is not recommended, an incomplete understanding of the testing limitations and the frustration often experienced by the veterinarian and the client in dealing with these difficult cases often lends itself to treating for EPM instead of continuing diagnostic evaluation. EPM remains one of the few neurological diseases affecting the horse’s gait that cannot be definitively diagnosed but can usually be effectively treated. Therefore, empirical treatment with evaluation of response to therapy will likely continue to be used and, in fact, demanded by performance horse clients. Unfortunately, many horses that have received empirical EPM treatment have not received complete neurological and musculoskeletal gait evaluations. Treatment of this group of horses will result in a significant extent of treatment failures. The time and money spent in pursuing ineffective treatment will likely lend itself to client frustration. For this reason, before treating for EPM, it is worthwhile to rigorously evaluate for and rule out other plausible causes of gait deficits in horses that show indistinct gait abnormalities.

Cervical radiography is a very important part of evaluating a horse that displays spinal ataxia or reduced neck mobility or neck pain. However, survey cervical radiography is also recommended for many or most horses that exhibit indistinct and/or uncategorized gait deficits. Interpretation of the cervical radiographs must be made carefully. Radiographic changes of the articular facets may occur without clinical signs in the lower cervical joints (C5–C6, C6–C7) as a horse ages and/or with athletic use.\(^2\) The presence of any bony change in the neck does not confirm myelopathy, and significant changes have been noted without clinical signs of nervous impairment or neck pain.\(^2\) Clinical signs from lameness, musculoskeletal, and neurologic evaluations of the horse have significant impact on how the cervical films may be interpreted. Suspicious radiographic findings that may be compatible with the clinical signs or a gait deficit should ideally be confirmed by myelography or by evaluating a response to treatment, such as intra-articular injection of the cervical facets. Although EPM and cervical myelopathy seem to be the most commonly diagnosed diseases affecting the equine spinal cord,\(^3\) numerous other infectious, degenerative, and inflammatory conditions also occur with clinical signs that can be clinically indistinguishable from EPM and cervical myelopathy. Other diseases may warrant evaluation of vitamin E levels, CSF cytology and protein, motor neuron disease evaluation, and muscle biopsy and radiographs of other areas of the axial skeleton and skull. Peripheral neuropathies or central neuropathy with lower motor neuron pathology may be associated with denervation atrophy (lower motor neuron signs) of a focal muscle or region of muscle.\(^4\) Clients have often attributed muscle wasting to EPM. However, more diffuse and symmetric muscle wasting is often seen in the epaxial and femoral musculature caused by cachexia and systemic disease.\(^4\) Disuse atrophy or atrophy caused by upper motor neuron lesions in the central nervous system may also occur in a localized muscle or muscle group. However, the severity of atrophy caused by upper motor neuron lesions is expected to be less severe than those caused by a lower motor neuron lesion. If no skeletal or neurological deficits are detected by thorough evaluation, an exercise test may be worthwhile. A protocol for an exercise test has been proposed by Valberg.\(^5\) During this test, the horse is exercised for a period of 15–20 min and can be simultaneously evaluated for changes in gait. Exercise may be performed on a lunge line or with a rider, depending on the clinical situation. The rider is asked to keep the horse on a loose rein. A blood sample for muscle enzyme evaluation is drawn between 4 and 6 h after the exercise test. Besides muscle enzyme evaluation for muscle disorders, the exercise test allows evaluation for exercise intolerance, airway noise, exercise-induced coughing, lameness that is induced or worsened by exercise, and metabolic or electrolyte abnormalities (if the test is prolonged and other blood and urine samples are submitted).

When the results of both the lameness evaluation and the neurological evaluation produce inconclusive results, the horse should be examined under saddle. The addition of weight to the horse and the response to rider often helps elucidate the cause of the gait deficit. Horses that have pelvic, sacroiliac, or lumbosacral pain may display perceivable clinical signs only when there is a rider on the horse.\(^6\) A horse with sacroiliac pain may fail to hold a lead in the hind legs and/or may display poor hindlimb impulsion at the trot and at the canter.\(^6\) Although a neurological problem may produce similar clinical signs, these signs, by themselves, seem to be more likely to suggest a musculoskeletal problem than a neurological problem. Neurological problems that produce ambiguous clinical signs such as toe dragging, poor engagement, and poor impulsion are usually associated with other abnormalities that are detected in association with various testing performed in the neurological evaluation such as head elevation, navigating an incline or a step up and down, blindfolding, and tail pull. These neurological deficits may be manifest by hypermetria, exacerbation of proprioceptive deficits, misjudging, stumbling, induction of instability, spasticity of the.
limbs, inability to avoid obstacles, circumduction in circles, or excessive pivoting in tight circles. The point is that a diagnosis of a neurological etiology of a gait deficit should be corroborated by some abnormal or arguably abnormal findings encountered in the neurological examination.

If a neurological problem was considered and the horse required evaluation under saddle for further elucidation of the cause of the gait deficit, the problem is likely to be in the hind end. Other than adding the weight and signals of the rider, the evaluation of the horse under saddle permits removal of the “clinical situation” by permitting the horse to move in the environment and footing to which it is accustomed. Evaluation of the horse can also be made both on the flat and over jumps if necessary. Repeating flexion tests may be worthwhile with the rider on the horse. When evaluating horses on the flat, a variety of gait deficits may be observed. Many of the gait deficits that become apparent with a rider involve the horse’s use of its hind end and may manifest as an inability to pick up and/or maintain a lead. Lead changes may be repetitively missed and this may be unilateral or bilateral, in both the forelimbs and hindlimbs, or only in the hindlimbs. The side on which the lead change is missed has not been a reliable indication of the affected side. Over a jump, only the rider may experience resistance of the horse to approach the jump, or it may be obvious to the observer. The horse may swap the lead before the jump, an experienced horse that normally lands correctly may land on a cross canter, the horse may jump to the side of the jump, may jump crookedly, or may twist its body over the jump. If a gait deficit is detected with these evaluations, an attempt to block the problems should be made. However, when a gait deficit exists and there is no obvious lameness to block, sequential nerve blocking up each hind leg may be necessary to facilitate identification of the problem. This may take several appointments and several days to complete. It is important to relate this to your client so that the expectations and requirements for the continued evaluation may be met. In the present population of horses, the preponderance of horses that required evaluation under saddle exhibited improvement with diagnostic nerve blocking. Evaluating both hind legs and blocking identical structures in both limbs is sometimes necessary. Sequential blocking of nerves and joints is also important. Because of the number of structures often blocked in 1 day, an apparent response to a nerve or joint block should be substantiated by repeating only the presumptively effective block well after any other joint or nerve blocks have worn off. Sites in the hindlimb that were disproportionately responsive to this approach in the present population of horses included the tarsi, the high suspensory, and the stifle joints. All joints should ideally be blocked separately. If diagnostic imaging with nuclear scintigraphy is required after joint and nerve blocking, up to 2 wk may be needed until the site of a block does not appear on a scintigraphic image. On occasion, clinical suspicion may suggest blocking of the sacroiliac joints. Blocking these joints was effective in facilitating a diagnosis of sacroiliac pain in five horses. However, many or most veterinarians no longer perform this block regularly because of the tendency to induce ataxia. Empirical treatment is sometimes worthwhile if the horse has compatible clinical signs and has received complete diagnostic evaluation that has failed to indicate another problem.

3. Results
By following the proposed diagnostic guidelines for the evaluation of gait deficits that require etiological categorization, the equine veterinarian can expect to provide a comprehensive diagnostic protocol to his/her clients when evaluating horses that exhibit clinically indistinct gait deficits. The veterinarian will expand his/her familiarity with less common diagnostic protocols for diseases that have the potential to affect the gait of the horse. Organized application of the presented guidelines facilitates earlier and accurate diagnosis, leading to more effective therapy. Using this diagnostic protocol over the past 15 yr, numerous horses have been evaluated for all types of gait deficit, and 112 horses have been specifically evaluated for clinically indistinct gait deficits of uncategorized etiology. Utilization of the proposed guidelines for the evaluation of this group of horses led to the determination that nearly 65% (71/112) exhibited a primary skeletal, ligamentous, or tendinous lameness conditions. The problems identified in these horses included tarsal lameness, stifle lameness, high suspensory lameness, fetlock lameness, and lameness caused by problems affecting the axial skeleton, including cervical arthritis, overriding dorsal spinous processes, pelvic lameness affecting the sacroiliac joints, and presumptive peripheral nerve impingement. Because of the apparent high incidence of appendicular and skeletal problems as a cause of ambiguous gait deficits, it is important to make great effort to rule out skeletal, ligamentous, and tendinous disorders in the evaluation of horses with these clinically indistinct signs. In the present population of horses, this has necessitated frequent re-evaluation at various gaits and under varying conditions, thorough and systematic blocking of the limbs, a willingness to repeat blocks, and to watch the horse under saddle while executing athletic maneuvers. Expanded diagnostic imaging modalities are often necessary to help evaluate horses with subtle lameness. Besides typical radiography, these modalities have predominantly included ultrasonography, nuclear scintigraphy, and magnetic resonance imaging.

Horses that were diagnosed with gait deficits caused by neurological impairment comprised ~35% (38/112) of the horses presented for gait evaluation. These horses were determined to be neurologically...
impaired based on the results of the complete neurological examination and various gait evaluations. Most of the horses evaluated were graded between 1 and 3 of 5. Ten percent (4/40) of the neurologically impaired horses appeared to display perceptible clinical signs only with manipulative neurological evaluation (head elevation, navigating an incline or a step, circling, etc.). The diagnosis of the neurologically affected horses included cervical myelopathy and/or cervical arthritis (29/38), EPM (2/38), equine motor neuron disease (1/38), presumptive equine degenerative myelopathy (1/38), and other infectious or inflammatory neurological conditions including trauma (2/38), stylohyoid osteopathy (1/38), and otitis media/interna (2/38). Six horses that showed cervical osteoarthritis without neurological signs responded to cervical facet injections with clinical improvement and returned to performance.

Horses that were determined to exhibit a primary muscle disorder have, in this 15-yr period, been the most infrequently encountered (~3% or 3/112). Although the most common breeds presented (pure warm blood breeds) can exhibit type 2 polysaccharide storage myopathy (PSSM), there were apparently no such horses presented for evaluation of a gait deficit. Quarter Horses were not commonly presented for gait evaluation, but of the horses determined to have a muscle disorder in this period, there was one Thoroughbred and two horses of Quarter Horses breed or Quarter Horse lineage. The relatively low number of horses affected with muscle disorders may be explained by the fact that muscle disorders of all types may be managed by owners, trainers, and veterinarians as sporadic exertional rhabdomyolysis (“tying-up”) and/or lack of fitness. If this is the case, some horses may be affected with myopathies that are successfully managed using regular exercise programs and high-fat/low-carbohydrate diets that have been advocated for many if not most muscle disorders. Another primary gait problem might also mask a muscle disorder that might otherwise have been detected using the present diagnostic protocol. In this 15-yr period, no horse was diagnosed with aortoiliac thrombosis as the etiology of perceived gait deficits.

4. Discussion
The proposed algorithm in Fig. 1 offers guidelines to the diagnostic approach of a horse with clinically indistinct gait deficits of uncategorized origin. These guidelines are organized and comprehensive and have proven to be helpful in the determination of the etiology of such gait deficits. This has produced successful results in the majority of examined horses, facilitating the horse’s return to performance. On occasion, the results of the evaluation have led to the recommendation for discontinuation of riding a neurologically impaired or otherwise unsound horse, facilitating safety for both the horse and its rider. The outlined approach has increasingly facilitated the identification of both lameness and neurological disease that appear clinically indistinct. With continued experience using the proposed diagnostic guidelines, the equine veterinarian can expect to continue to improve in the identification and diagnosis of disorders that cause clinically indistinct gait deficits.

The algorithm for evaluation of clinically indistinct gait deficits focuses on step by step evaluation of the horse that leads to categorization of the gait deficit and detailed examination of the body systems that can impact the gait of the horse. This involves complete evaluation of the musculoskeletal and neurological systems, accompanied by manipulative tests to facilitate clinical detection of a subtle deficit. When following the proposed algorithm, ancillary procedures and tests are recommended to help further clarify clinical findings when etiology of the signs is poor. Finally, it is suggested that evaluating a horse under saddle and under less clinical conditions may also help to clarify a gait deficit. It is important to be willing to repeat diagnostic tests and physical evaluations to reassess a condition, to affirm a presumption, and to confirm a finding. This may require retuning to an earlier point within the diagnostic algorithm and being open to other possible explanations of the clinical signs detected. The cases that benefit from this diagnostic algorithm are not restricted to horses that have indistinct gait deficits. Occasionally, a gait deficit is clearly present, but its etiology is not. The evaluation of horses with clear gait deficits but an indistinct etiology is as likely to benefit from the proposed approach as are the horses with indistinct gait deficits.

Case 1 Summary
A 13-yr-old experienced Salle Fancais gelding presented with poor performance and presumptive lameness several months in duration. The horse competed regularly as a children’s hunter and in equitation classes. The complaint was primarily from the rider. The complaints were that the horse displayed hesitation at the jumps, was missing lead changes, and was knocking rails more frequently. The horse had been examined by several veterinarians and had received two rounds of therapy for EPM. The stifles had been treated with intra-articular injections. The hocks were also injected early in the course of the applied therapies. No improvement was recognized. On presentation, the initial lameness examination showed ambiguous and subtle gait deficits. A complete neurological evaluation failed to show evidence of neurological deficits. Physical examination and ophthalmological examination findings were within normal limits, and there seemed to be no sensitivity or resistance to head, neck, spinal, and pelvic palpation and manipulation. With continued exercise, there was no change in gait deficit. However, once a rider was mounted,
the gait deficit appeared to worsen. Impulsion of
the hindlimbs appeared to be reduced, and the horse
could not maintain a lead. When asked to
jump, the horse overtly refused on two occasions,
and the rider reported the sensation of the horse's
hiccoughing in hand when asked to jump on other
occasions. Given these findings, hind end lame-
ness was suspected. The horse received sequen-
tial nerve and joint blocking over the next few
days. These blocks failed to change the gait def-
cit. After regional anesthesia of the limbs, the
sacroiliac joints were blocked. Because the horse
did not appear to become ataxic, the rider was
again asked to mount the horse for evaluation.
The sacroiliac blocks appeared to significantly improve
the gait deficit by resumption of lead
changes, impulsion, and ability to hold a lead.
A nuclear scintigraphic scan was requested 2.5 wk
later, but the findings were inconclusive. The
horse was successfully treated for sacroiliac pain
after nuclear scintigraphy. He returned to regu-
lar successful training and competition.

Case 2 Summary
A 10-yr-old Thoroughbred gelding presented for a
history of an undefined gait deficit reported to
begin after a fall over a jump. The horse com-
peted regularly as an adult amateur hunter. After the fall, the horse was reported to be consist-
tently hitting jumps and exhibiting a reduced
level of performance. The lameness examination
showed no significant abnormalities. The physi-
cal and ophthalmological examinations were
within normal limits. Physical evaluation and
manipulation of the cervical/axial skeleton was
within normal limits. However, because of the
history of trauma, cervical radiographs were ob-
tained. Cervical radiographs showed mild osse-
ous changes in the caudal cervical facets. These
changes did not seem to be associated with clinical
signs or any abnormal findings with manipulation
of the neck or spine. A complete neurological ex-
amination was interpreted to be within normal
limits. Physical examination and blood work re-
sults after exercise failed to show any indication of
myopathy. The horse was subsequently evalu-
ated under saddle. During this examination, it
was noticed that the horse was exhibiting poor
hindlimb engagement and was dragging its toes
(more than what the owner felt was normal).
A neurological examination was repeated, and the
manipulative tests were again evaluated. After
this evaluation, it was believed that the horse
exhibited mild proprioceptive deficits with head
extension and incline navigation. These conclu-
sions were tentative. Further cervical spinal di-
agnostic imaging was considered, but EPM was
also considered. The less costly and invasive
CSF and serum EPM testing was pursued before
pursuing myelography. The results of the EPM
testing showed strong positive serum and CSF
immunoblot. Given the testing limitations, be-
fore pursuing myelography, polymerase chain re-
action (PCR) testing of the CSF for EPM was
requested. The results of the PCR were positive.
Because PCR contamination was considered to be possible, it was decided to treat the horse for EPM
and to re-evaluate for response to therapy. Re-
evaluation of this horse indicated significant im-
provement in performance, improvement in jump-
ing, improved engagement of the hind legs, and
reduced toe dragging. After apparent re-
response to therapy, it was concluded that the horse
had been affected with EPM. Although there was
apparent improvement, the owner also reported
that she did not feel that the horse had returned to
its previous level of athletic performance. It was
considered that the fall could have occurred be-
cause of the effects of EPM and/or that there could
be cervical myelopathy or osteoarthritis either as a pre-existing condition or induced by the fall.
It was recommended that if the horse began to
show stiffness or other clinical signs, the neuro-
logical and lameness examinations should be re-
peated, and injection of the cervical articular
facets may be warranted. The horse continued to
show successfully without this need.

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