THE LAMENESS QUIZ
OBSERVATIONS AND CHARACTERISTICS OF LAME HORSES

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The purpose of this lecture is to reinforce your ability to recognize gait deficits during lameness examination. Please attend the lecture and make your own observations of the video segments shown before reading these notes. I will show a series of brief video segments and give you relevant clinical history. I will ask you to formulate your initial thoughts and then review the cases, adding other examples when appropriate to illustrate the characteristics of lameness as they appear to me. Below you will find a summary of my thoughts regarding the observations and characteristics of lame horses, but it is my hope you will formulate your own!

Severe, bilateral forelimb lameness can mimic odd gait deficits of the hindlimbs. While not common horses with bilateral condylar fractures or dorsal cortical fractures of the third metacarpal bone, or those with bilateral slab fractures of the third carpal bone may appear to exhibit hindlimb gait abnormalities. You may, in fact, be summed to examine the horse because of hindlimb, rather than forelimb lameness. I have made this observation in a miniature horse and a 3-year-old STB filly with bilateral severe shoulder lameness as well.

Careful observation during lameness examination may reveal loss of support of a particular part of a limb, a marked conformational abnormality, asymmetry or some other interesting relevant abnormality, which can be critical to determining the source of pain causing lameness. In the example show a horse has injured the origin of the gastrocnemius muscle and lost support of a hock, causing a dynamic sickle-hock conformational change. Horses with gastrocnemius origin injury may also exhibit an unusual hindlimb gait deficit (unusual internal rotation of a hindlimb), but this observation should be made carefully since in some horses bilateral internal rotation of the hindlimbs is normal. Other important observations are straight-hock conformation (often present in horses with chronic, recurrent suspensory desmitis), fetlock drop, and pelvic asymmetry (pelvic fractures, muscle atrophy). A rare observation is scapular elevation (occurs with subscapular injury or in horses with injury/disease of the nerve roots supplying the thoracic limbs).

Circling remains a very important, basic manoeuvre during lameness examination. Many horses with forelimb and hindlimb lameness will be worse with the limb on the inside of a circle. Worse while turning is common in horses with a source of pain causing lameness in the distal aspect of a forelimb. Caution should be used in over-interpreting observations regarding circling and there are many common misconceptions. Carpal region pain may cause horses to show lameness with an affected limb on the outside of a circle (horses may be better with the limb on the inside of a circle). Upper limb pain is exacerbated with a limb on the inside of a circle when horses are trotted in hand.

Horses with prominent unilateral forelimb lameness will appear lame in the contralateral hindlimb. For example, a horse with a grade 3 right forelimb (RF) lameness has a shortened cranial phase of the stride, head elevation (unloading) during protraction of the RF and a head nod down while loading the LF, a straight-forward RF lameness. The horse must shorten the cranial phase of the stride in the contralateral hindlimb, the left hind (LH), a stride-to-stride gait compensation to maintain symmetry, and appears “lame” in the LH (as judged by a shortened
cranial phase of the stride). Apparent LH lameness resolves once the source of pain causing RF lameness is abolished using diagnostic analgesia. Importantly, a pelvic hike consistent with LH lameness is not seen. The horse has no need to unload (pelvic hike up) the LH and load (pelvic drop, settling) the RH, differentiating this situation from genuine LH lameness. Ipsilateral coexistent lameness is much more common than is contralateral coexistent lameness. In horses that gallop, canter, or pace coexistent ipsilateral forelimb and hindlimb lameness is the norm. Coexistent RF/RH and LF/LH is much more common than contralateral coexistent lameness with one exception — trotters. Trotters most often have coexistent contralateral lameness, LF/RH and RF/LH. Load sharing between the diagonal pairs is a simple explanation. However, another explanation involves the issue of interference at high speeds. For instance, a trotter with LF lameness reduces the cranial phase of the stride in the LF. Prolongation of the caudal phase of the stride puts a trotter at risk of interference of the LF with the LH; a protective mechanism is to shorten the cranial phase in the LH, so the trotter looks like it is “running behind”, or on the left lead. Simulating left lead gallop chronically overloads the RH and eventually coexistent LF/RH lameness develops. If there is coexistent ipsilateral lameness (LF and LH) there will be a head and neck nod commensurate with LF lameness and a pelvic hike associated with LH lameness and the cranial phases of the stride in both limbs will be reduced. Diagnostic analgesia must begin in the hindlimb since hindlimb lameness can mimic ipsilateral forelimb lameness and cause a head and neck nod.

Single-limb lameness occurs commonly in forelimbs and hindlimbs and clinical recognition is generally straight-forward. Analgesia is used to localize the source of pain causing lameness and a diagnosis is established. In horses with chronic, degenerative conditions low-grade mild lameness may go unrecognized for weeks to months. Horses are effective at compensating for mild, chronic low-grade pain and compensatory, coexistent lameness often develops in predictable patterns. Bilateral lameness is most common, and if symmetrical, can be a source of poor performance that goes unrecognized. Bilateral front foot pain is a common cause of poor performance that leads to compensatory hindlimb lameness problems or gait abnormalities. Veterinarians are summoned to evaluate the horse for suspected hindlimb lameness, riders often complain the horse “feels off behind”, only to find the horse to be lame up front as a result of chronic foot pain. Horses may trot apparently without lameness in a straight line but lameness is easily recognized while circling the horse, and is particularly prominent on a hard surface. Palmar digital analgesia in one limb produces obvious contralateral forelimb lameness. The horse’s apparent hindlimb gait deficit resolves once pain is abolished in the forelimbs. Thus, forelimb lameness mimics hindlimb lameness. Severe bilateral forelimb lameness can cause substantial hindlimb gait deficits, some of which resemble neurological abnormalities. Rarely, miniature horses or ponies develop severe bilateral forelimb lameness as a result of scapulohumeral joint dysplasia and osteoarthritis (OA), causing unusual hindlimb gait deficits. Bilateral mild laminitis, palmar foot pain, bilateral metacarpophalangeal or carpal OA, and bilateral slab fractures of the third carpal bone can cause horses to exhibit unusual hindlimb gait deficits.

Hindlimb lameness can mimic forelimb lameness – Horses with substantial (lameness grade >2.5-3) hindlimb lameness can look like they are lame in the ipsilateral forelimb but may only be lame behind (or be lame in both hindlimb and ipsilateral forelimb, see above). A horse with LH lameness will have a pelvic hike up during protraction of the LH and settling during weight-bearing of the RH and will have a shortened cranial phase of the stride in the RF. However, while trotting the horse will appear to have a head and neck nod similar to LF lameness, since load is shifted forward (LH and RF move together) and the head and neck nod down when the RF is
weight-bearing. If only the forelimbs are watched the examiner will mistakenly diagnose LF lameness.

Racehorses with multi-limb lameness develop a typical short, choppy, uncomfortable gait and appear as Dr. R Pilsworth would describe, “all jarred up”. These horses appear to be sore all over or “foot sore”. Subchondral bone pain arising from mal-adaptive bone remodelling of the distal aspects of the third metacarpal/metatarsal bones is the most common cause of this multi-limb gait deficit. And, the hindlimb component has the most profound influence on the development of this abnormal gait. An examiner can usually pick out a “lamest limb” and blocking begins in this limb. The lamest limb can sometimes be determined while watching the horse carefully during deceleration at a trot in hand. Typically, lateral plantar metatarsal analgesia in one limb results in pronounced contralateral hindlimb lameness, much more obvious than in the initially assessed lamest limb. Lateral plantar metatarsal analgesia in the second hindlimb results in marked improvement – the horse may then show forelimb lameness or reverts to showing clinical signs in the original hindlimb.

Horse with authentic upper forelimb pain move with a marked shortened cranial phase of the stride often have pain originating from the shoulder region, but keep in mind, shortening of the cranial phase of the stride occurs in nearly every horse with lameness, forelimb and hindlimb. In horses with an upper forelimb source of pain there is often exaggerated head and neck excursion while walking, and in fact head and neck excursion while walking may be more pronounced than while trotting. In the hindlimb, horses that walk with a shortened caudal phase of the stride could have an upper limb source of pain, and most commonly have coxofemoral or another severe source of pelvic pain, but, horses with severe hind digit pain walk similarly. Plaiting occurs in horses with bilateral hindlimb pain originating from the pelvic region, but can occur in a horse with bilateral hindlimb pain from a source much more distal in the limb, and is by no means pathognomonic for pelvic pain. I have seen it however in horses with bilateral ilial stress fractures, sacroiliac pain and in horses with coxofemoral joint pain. If one considers the stifle region “upper limb” then toe-drag is often seen in horses with stifle region pain; however, another common hindlimb source of pain, proximal suspensory desmitis often causes horses to develop toe-drag as well. So, if there is a difference in gait between walking and trotting, I think upper limb. If there is an exaggerated head and neck nod or excursion, I think upper limb. If in hindlimb lameness there is a shortened caudal phase of the stride I keep very much in mind the upper limb.

Hemarthrosis or hemorrhagic synovitis can be a cause of intermittent, severe lameness, and the most commonly affected joints/sheaths are the antebrachiocarpal joint, the carpal sheath and the tarsocurural joint.

Racehorses exhibiting a marked “cross-extensor test” (during a flexion test the horse is worse on the supporting limb, rather than the limb undergoing flexion) often have carpal pain, although this observation is not pathognomonic for that region.