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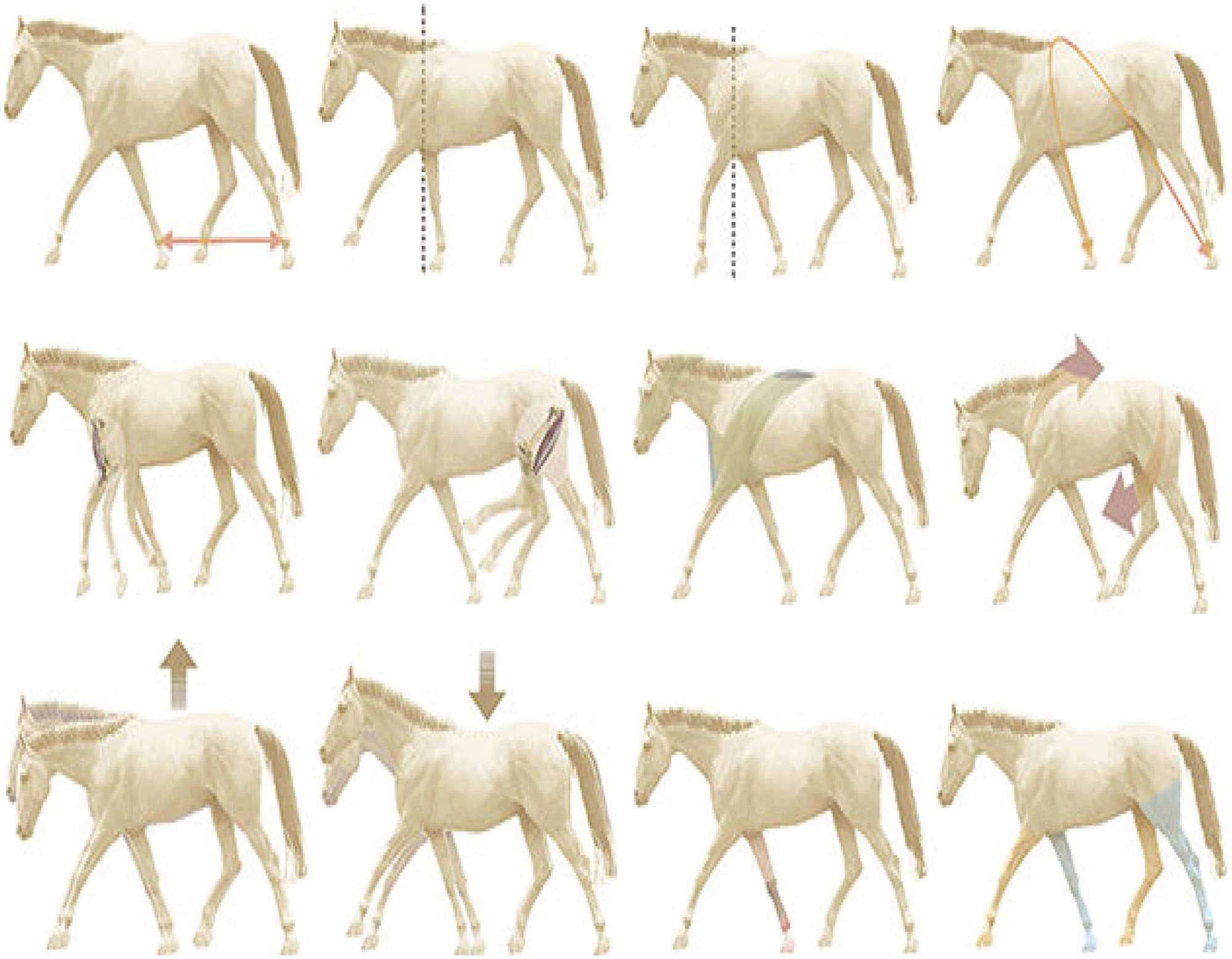
QUINE LAMENESS
FOR THE LAYMAN



Tools for Prompt **Recognition**, Accurate **Assessment**, and Proactive **Management**

G. ROBERT GRISEL, DVM

Equine Lameness *for the* Layman

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Accurate Assessment, and
Proactive Management**

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For my family

Peter, Ben, Lisey, Kate, and Fionn

for their patience and support

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Introduction

The words, "This would be a lot easier if the horse could just tell us where it hurts!" have been muttered many times over the course of my veterinary career. The horse's inability to verbally disclose the reasons for inadequate performance seems to imply that we, as equine professionals, have to be exceptionally clever with regard to deciphering the details of any related problem. Of course, the savvy veterinarian understands that the inherent "mystery" of equine lameness may also figure into a successful career.

Ironically, my many years as an equine practitioner have taught me that lame horses actually *do* tell us where it hurts. Of course, it took me a while to realize that horses went to the trouble to impart this information. And like many of us, I initially lacked the innate ability to visually "tune into" the lame horse, primarily because I was accustomed to listening with my ears instead of my eyes.

Indeed, the lame horse actually goes to great lengths to tell us that there is a problem. Most will even reveal the likely cause to anyone who is willing to pay a little extra attention. As we'll see, horses utilize a form of "sign language" that they convey by altering the movement of their body and limbs. Those of us who learn to assimilate this form of nonverbal communication will become effective observers of equine lameness. Of course this demands that we first explore the interplay between motion and meaning.

The key to informative visual assessment lies in our ability to recognize patterns of movement. The horse repeats both normal and abnormal actions with each stride, thereby affording us the opportunity to develop and confirm our impressions over time. *Pattern recognition* will be the basic technique that we utilize during our study of the lame horse throughout the course of this book. This skill requires that we develop the ability to "listen" with our eyes and refrain from overthinking what we see. Fortunately for us, lameness recognition can indeed be learned.[1]

The primary objective of this book is to provide a structured and systematic approach to visually interpreting physical gestures made by the lame horse. These gestures come in a variety of visual forms, each of which retains a unique appearance. Fortunately for us, horses are quite expressive in their movement and tend to adhere to explicit patterns of gait that can be readily distinguished from one another. Horses of different breeds that do different things in different parts of the world, for instance, will usually display a specific form of lameness in the same way. The following chapters provide you with the tools necessary to identify common visual markers displayed by lame horses. We will also discuss *when* and *where* to look for these markers so as to glean the maximum amount of clinical information possible from your assessment. Acquiring basic knowledge of the common forms of lameness (Section III), pertinent equine anatomy (Section IV), and common visual markers (Section V) will help as you navigate the examination process (detailed in Section VI).

As you might expect, some of the ensuing content is quite advanced; several visual markers are very subtle and their interpretation somewhat sophisticated. Notwithstanding, it will help you to consider the material solely within the context of how it will affect what you see with your eyes. Video commentaries are provided along the way to help you identify and characterize various patterns of movement, one feature at a time. Each commentary can be accessed directly on your smartphone by scanning the QR ("Quick Response") code located within that aspect of the book's narrative pertaining to the topic of interest. (There is also an index of video links and codes beginning on page 229.) Once you learn to recognize distinct patterns of movement, you'll be able to distinguish between numerous forms of equine lameness.

Naturally, the intention is not to create a world in which horse owners are diagnosing lameness. A *diagnosis* is what we pay the veterinarian to provide for us. Rather, the information in this guidebook should be used to develop an appreciation and basic understanding of equine locomotion with the goal of being better prepared to recognize, classify, and rate your horse's lameness when it occurs. The benefits associated with acquiring these basic skills will also be highlighted. Assessment will become especially fun and productive once you've learned to differentiate the nature and severity of what you're seeing. You'll be surprised at how much of the horse's language you're able to interpret. You'll also be surprised to discover how much your horse has been trying to convey all of these years.

The information compiled in this book is based on personal experience. Observations have been developed and refined while performing approximately 30 equine lameness examinations per week over a 25-year period. Albeit the techniques described herein relate to horses, they could also be implemented to assist in the visual assessment of other quadrupeds and humans. Of course, they are most helpful when evaluating animals that can't speak (verbally) to us.

Glossary

Throughout the pages ahead, you will find words in **bold** in the text. These highlighted words correspond with the definitions provided below. You can familiarize yourself with them now, or refer back as you come upon unfamiliar terms.

A

Abaxial: Away from the center of the body or limb.
Abduction: The movement of a body part *away* from the midline of the horse.
Acute: Of recent or abrupt onset. "This is an *acute* lameness; it just developed this morning."
Adduction: The movement of a body part *toward* the midline of the horse.
Adhesion: The abnormal adherence of one anatomic structure to another.
Amphiarthrodial Joint: A fibrocartilagenous junction that allows limited motion between articulating bones.
Annular Ligament: A fibrous band that encircles the superficial and deep digital flexor tendons as they pass behind the fetlock joint and proximal sesamoid bones.
Anterior: Toward the front of the horse. More appropriately denoted as *cranial.* "The head is *anterior* to the neck."
Appendicular: Refers to the limbs.
Arthritis: Joint inflammation.
Arthrotherapy: Treatment directed at improving joint health and function.
Artificial Gait: A gait that is either inspired through generations of breeding or learned through training.
Ascending Movement: Upward motion of one or more body parts.
Atrophy: Degeneration (or "wasting away") of body tissue (e.g. muscle) due to lack of use or underlying pathology.
Axial: Toward the center of the body or limb.
Axial Lameness: Altered movement stemming from one or more structures associated with the axial skeleton. "Temporomandibular synovitis (TMJ), cervical arthrosis and 'kissing spines' all have the potential to generate *axial lameness* in the horse."
Axial Skeleton: Consists of the horse's head, vertebral column (within the neck, thorax, and lumbar regions), and sacrum.

B

Bar Shoe: Characterized by a closed (rather than open) heel.
Beat: The number of beats associated with a gait refers to the number of individual footfalls that occur before the sequence repeats. Two feet striking the ground surface simultaneously generate a single beat.
Bilateral Lameness: Altered movement manifesting on both (right and left) sides of the horse.
Biomechanical Lameness: Altered movement(s) made in an attempt to accommodate restricted or exaggerated action of one or more parts of the horse's anatomy (usually the limbs). Many forms of biomechanical lameness have no inflammatory component and accordingly do not hurt.
Biomechanics: The structure and function of biological systems.
Breakover: The action of the hoof as it pivots over the toe to lift and move the respective limb forward.
Bursa: A synovial sack that facilitates tendon movement over bone via lubrication.

C

Caudal: Toward the tail. Refers to anatomy of the head and body parts above the carpi (knees) in the forelimbs and tarsi (hocks) in the hind limbs. "The pelvis is *caudal* to the neck."
Centerline: An imaginary line that splits the horse into right and left halves (see also Midline).
Central Neurologic Lameness: Altered movement stemming from abnormal function of the brain and/or spinal column.
Cervicothoracic: The region adjoining the neck and chest.
Chronic: Of long or indefinite duration. "This horse has a *chronic* lameness; it first developed last year."
Circumduction: The circular or conical movement of a limb relative to the horse's body. During protraction, the *circumducting* limb moves away from the midline before moving back toward it, thus tracing a semicircle. This gait deficit is most often associated with neurologic disease in the horse.
Collagen Fibers: Small, inelastic reticular fibrils comprised of insoluble protein. Collagen fibers are found in skin, bone, ligaments, tendons, and cartilage, and comprise nearly one-third of all body protein.
Collateral: On either side. "The medial (inside) and lateral (outside) *collateral* ligaments of the fetlock joint function to maintain stability."
Collection: A movement performed by the horse in which more weight is assumed by the hind limbs relative to the forelimbs.
Columnar: A pattern of muscle tissue in which individual fiber bundles (called *fascicles*) run parallel to the long axis of the structure and its respective tendon. This pattern allows for extensive range of motion but generates nominal force.
Compensating Limb: The limb that "takes the brunt" of the primary problem associated with another limb. "The horse's left front limb often serves as the *compensating limb* for the right hind limb."
Concentric Muscle Action: Contraction or shortening of muscle fibers.
Contraction: The process of becoming shorter or shrinking.
Contralateral: Located on the other side of the horse. "The right front limb is *contralateral* to the left front and left hind limbs."
Contralateral Counterpart: The comparable limb located on the other side of the horse. "The right hind limb is the *contralateral counterpart* to the left hind limb."
Contralateral Limb: A limb located on the other side of the horse. "The left front limb is *contralateral* to the right front and right hind limbs."
Correct Lead: Leading with the inside forelimb while turning or circling.
Cranial: Toward the head. Refers to body parts above the carpi (knees) in the forelimbs and tarsi (hocks) in the hind limbs. "The neck is *cranial* to the pelvis."
Cross-Firing: Striking the forelimb with the opposite (diagonal) hind foot. Occurs in pacers.

D

Daisy Cutter: A horse that demonstrates a flat stride with very little elevation of the limbs during protraction.
Descending Movement: Downward motion of one or more body parts.
Desmitis: Inflammation of ligament tissue.
Desmopathy: Ligament disease.
Diagonal Gait: A method of movement in which the forelimb and hind limb on opposite sides of the horse mimic each other in action.
Diagonal Pair: The pair of limbs that mirror each other in action. "The left hind and right front limbs comprise one *diagonal pair* and the right hind and left front limbs comprise the other."
Diarthrodial Joint: A freely-movable joint characterized by the presence of a fibrous capsule, synovial membrane, lubricating (synovial) fluid and fibro- or hyaline cartilage layers which line opposing bony surfaces.
Differential List: A distinguishing list of diseases or conditions that present similar symptoms or clinical signs.
Distal: Toward the free (lower) end of the limb. "The foot is *distal* to the fetlock joint."
Distal Tarsitis: Inflammation associated with the lower joints of the horse's tarsus (or hock).
Dorsal: Toward the front of the limbs (below the levels of the carpus and tarsus) and toward the upper aspect of the head, neck, back, and pelvis. "The horse's withers are *dorsal* to the ribs."
Dorsal Plane: Passes through the head, body, or limb parallel to its dorsal surfaces.
Dorsal Subluxation: Abnormal repositioning or dislocation of a limb structure in a forward direction or a body structure in an ascending direction.
Dorsiflexion: Bending of the spine in a way that moves either end away from the ground surface.
Dorsolateral: Toward the front and outside of the lower limb (below the levels of the carpus and tarsus). "The outside toe quarter is *dorsolateral* to the center of the navicular bone."
Dorsomedial: Toward the front and inside of the limb (below the levels of the carpus and tarsus). "The inside toe quarter is *dorsomedial* to the center of the navicular bone."
Drifting: Orientation of the horse's spine in a way that is not consistent with its trajectory (directional path of movement).

E

Eccentric Muscle Action: Relaxation or lengthening of muscle fibers.
Etiology: Cause or origin.
Excursion: Excessive up-and-down movement of one or more of the horse's body parts.
Extrinsic: Originating from outside of the horse's body or respective body part.

F

Fascia: A thin sheath of fibrous tissue encasing muscle or other organ.
Fibrocartilagenous Joint: Formed via the presence of an intervertebral disc.
Fibro-Osseous Junction: The site at which a ligament or tendon attaches to bone.
Fibrotic Myopathy: Pathologic condition characterized by the presence of scar (or fibrotic) tissue within one or more muscle bellies. Scar tissue can deleteriously affect the pliability of the muscle(s) within which it develops, thereby having the potential to precipitate biomechanical lameness.
Fibrotic Tissue/Fibrosis: Scarring that typically forms pursuant to an injury and local tissue damage.
Flexor Tendonitis: Inflammation of the superficial and/or deep digital flexor tendons.
Flexural Deformity: Excessive bending of one or more joints in response to disproportionate tension of the flexor apparatus.
Flight Path: The track of a limb or foot as it advances through the air during the non weight-bearing phase of the stride.
Flight Phase of Stride: The phase of a horse's stride during which the respective limb is airborne (not in contact with the ground surface).
Forging: Striking of a forelimb with the ipsilateral hind limb (on the same side of the horse). Usually occurs as the toe of the hind foot strikes the heel of the ipsilateral forefoot at the trot. Also known as *overreaching.*
Fracture: A break or fragmentation. "Blunt trauma resulted in *fracture* of the underlying bone."
Fusion: The coalescence of two or more structures to form a single entity.

G

Gateado: A smooth and supple quality possessed by superior-moving Peruvian Paso horses.
Greater Trochanteric Bursitis: Inflammation of the greater trochanteric bursa, which is a synovial sac that lubricates the middle gluteal muscle tendon as it courses over the greater trochanter of the femur just outside of the hip joint. This condition is often referred to as *whorlbone* in horses.
Ground Reaction Force (GRF): The force exerted by the ground surface on a horse's limb that is in contact with it. Vertical (up-and-down), transverse (side-to-side), and sagittal (front-to-back) ground reaction forces are experienced by each limb during the latter's respective stance phase of the stride.

H

Hyperflexion: Flexion of a joint beyond normal or expected limits.
Hypermetric: Movement (such as flexion of the limbs) beyond normal limits.
Hypoflexion: Flexion of a joint short of normal or expected limits.
Hypometric: Movement (such as flexion of the limbs) shy of normal limits.

I

Innervation: Nerve supply.
Interference: A lateral gait deficit in which one foot contacts the inside of the opposing limb during flight. This is most commonly observed in horses that wing-in or plait, due to the close proximity of contralateral limbs during this activity. Also known as *brushing.*
Inter-: Between.
Intermittency: Alternately appearing and disappearing.
Intermittent Upward Fixation of the Patella (IUPF): A condition characterized by inadvertent engagement (or locking) of the patella over the medial trochlear ridge of the femur (an action epitomizing the primary component of the hind stay apparatus).
Intermuscular: Between muscle bellies.
Interphalangeal: Between two phalanges.
The horse has three phalanges in each limb: the first phalanx (also known as the long pastern bone or P1), the second phalanx (also known as the short pastern bone or P2), and the third phalanx (also known as the coffin bone, pedal bone, or P3). There are two interphalangeal spaces: one between P1 and P2 and one between P2 and P3.
Intra-: Within.
Intramuscular: Within a single muscle belly.
Intrinsic: Originating from within the horse's body or respective body part.
Ipsilateral: Located on the same side of the horse. "The right front limb is *ipsilateral* to the right hind limb."

J

Joint: A point of articulation between two or more bones. Joints serve to absorb the force of impact, transfer the force via cartilage to bone, and to allow a variable degree of movement between bones.

K

Kinematics: The geometry of movement.
Kinesiology: The science of movement.
Kinetics: Forces that cause movement.
Kyphosis: Excessive ventroflexion of the spine, often referred to as a *roached back.*

L

Laminitis: Inflammation of the (laminar) tissues that bond the horse's hoof to the underlying (pedal) bone. Laminitis is often bilateral and more commonly associated with the horse's forelimbs.
Lateral: Away from the median plane. "When you look at a horse's left side you see the *lateral* surfaces of the left limbs."
Lateral Gait: A method of movement in which the forelimb and hind limb situated on the same side of the horse mimic each other in action.
Laterality: Dominance of one side of the brain or body over the other side.
Lead: The forelimb not assigned to the working diagonal pair determines the lead at the canter, gallop, and run. The lead forelimb protracts farther than the other forelimb at these gaits.
Ligament: A band of fibrous connective tissue that attaches bone to bone or bone to tendon. Ligaments serve to stabilize structures relative to one another.
Lordosis: Excessive dorsiflexion (or extension) of the spine, often referred to as a *swayback.*
Luxated: Full dislocation or displacement of one object relative to another. This term usually refers to the abnormal positioning of bones relative to one another across a joint.

M

Medial: Toward the median plane. “When you look at a horse’s left side you see the *medial* surfaces of the right limbs.”
Median Anatomy: Parts of the horse’s body that are located near to or along the median plane (or midline).
Median Movement: Motion associated with the horse’s axial anatomy comprising the head, neck, chest, abdomen, rump, and tail (everything excluding the limbs).
Median Plane: Divides the horse’s body into right and left halves.
Metricity of Stride: Refers to the degree of movement with respect to the horse’s stride.
Midline: An imaginary line that splits the horse into right and left halves (see also Centerline).
Motor Nerves: Nerves that serve to incite muscle contraction or gland activity.
Multifactorial Lameness: Altered movement arising from more than primary source of pathology. “A horse with current right front foot and right stifle joint pain would be expected to exhibit *multifactorial lameness.”*
Muscle: Tissue comprised of fibers that have the ability to contract and relax, thereby generating movement or maintaining posture of the bones to which it is attached.
Musculoskeletal: Refers to the horse’s bones, muscles, ligaments, tendons, and joints.
Myopathy: Muscle disease.
Myositis: Inflammation of muscle tissue.

N

Natural Gait: A gait that the horse demonstrates within the first few days of life and is not inspired by generations of breeding.
Negative Palmar/Plantar Angulation: Abnormal positioning (angulation) of the third phalanx (P3, coffin, or pedal bone) within the hoof capsule; the front of the bone is elevated relative to the back of the bone and ground surface.
Neurologic Lameness: Altered movement(s) made in response to a lack of neuromuscular input and/or in an attempt to maintain balance.
Neuropathy: A disease or condition affecting the nervous system.

O

Off Side: The horse's right side, also known as the *far side.*
On Side: The horse's left side, also known as the *near side.*
Oscillation: The repetitive up-and-down movement of one or more of the horse's body parts.
Ossification: The process of assuming the characteristics of bone. Sometimes denoted as *calcification.*
Overreaching: Striking of a forelimb with the ipsilateral hind limb (on the same side of the horse). Usually occurs as the toe of the hind foot strikes the heel of the ipsilateral forefoot at the trot. Also known as *forging.*

P

Pain-Mediated Lameness: Altered movement(s) made in an attempt to avoid pain. Pain is invariably a consequence of local inflammation and/or nerve compression. As you might surmise, pain-mediated lameness *hurts.*
Palmar: Toward the back of the forelimb below the level of the carpus. "The navicular bone sits *palmar* to the coffin joint."
Palmarolateral: Toward the back and outside of the forelimb below the level of the carpus. "The outside proximal sesamoid bone is positioned *palmarolateral* to the fetlock joint."
Palmaromedial: Toward the back and inside of the forelimb below the level of the carpus. "The inside proximal sesamoid bone is positioned *palmaromedial* to the fetlock joint."
Palpation: The examiner's use of fingers and hands to physically perceive abnormality(ies) on or within the horse's body and limbs.
Pathognomonic: Distinctively peculiar to a specific disease or condition.
Pathology: A disease process or the study of disease. Anything that is *pathologic* is abnormal.
Peak Vertical Force (PVF): The maximum vertical ground reaction force, which is encountered by each limb during mid-stance.
Pennate: A pattern of muscle tissue in which individual fiber bundles (called fascicles) attach obliquely (in a slanting array) to a common centralized tendon that runs the entire length of the structure. This pattern allows for higher force production but maintains a smaller range of motion.
Perineural: Near or adjacent to nerves.
Periosteum: A dense layer of connective tissue that covers bone.
Peripheral Lameness: Lameness associated with the horse's extremities (limbs).
Peripheral Neurologic Lameness: Altered movement stemming from abnormal function of nerves outside of the brain and spinal column.
Periphery: The aspects of the horse located away from the center or midline. "The *peripheral* nervous system comprises the nerves and ganglia outside of the brain and spinal cord."
Pisos: A term describing the quality of gait (e.g. timing, extension, animation, smoothness, elegance, and forward motion) demonstrated by the Peruvian Paso horse.
Plaiting: Technically means *braiding.* With respect to equine ambulation, plaiting refers to the way in which the limbs track during protraction; one foot is placed directly in front of the other. Often referred to as *rope walking.*
Plantar: Toward the back of the hind limb below the level of the tarsus. "The hind navicular bone sits *plantar* to the coffin joint."
Plantarolateral: Toward the back and outside of the hind limb below the level of the tarsus. "The outside proximal sesamoid bone is positioned *plantarolateral* to the hind fetlock joint."
Plantaromedial: Toward the back and inside of the hind limb below the level of the tarsus. "The inside proximal sesamoid bone is positioned *plantaromedial* to the hind fetlock joint."
Posterior: Toward the rear of the horse. More appropriately denoted as *caudal. "*The horse's pelvis is *posterior* to the withers."
Prognosis: A forecast of the course of a disease or affliction. "High-motion joint disease often carries a guarded *prognosis* for future soundness in the performance horse."
Protraction: The movement of a body part (e.g. a limb) in a forward direction.
Proximal: Toward the attached (upper) end of the limb. "The carpus is *proximal* to the fetlock joint."

R

Retinaculum: A fibrous retaining band that houses and stabilizes multiple tendons as they course along the horse's limb.
Rostral: Toward the nose. Refers only to anatomy of the head. "The horse's eyes are *rostral* to the ears."

S

Sagittal Plane: Any plane parallel to the median plane. For instance, a plane dividing the right and left sides of a hoof.
Sensory Nerves: Nerves that serve to carry sensory information to the brain for processing.
Sheath: A "sleeve" that envelops tendons as they course over or under joint surfaces.
Solar Surface: The (bottom) aspect of the foot that contacts the ground surface.
Speedy Cutting: Striking the hind limb with a forefoot. Usually observed in disciplines that entail making sharp turns at high speeds (e.g. barrel racing and cutting).
Stance: The phase of a horse's stride during which the respective limb is in contact with the ground surface.
Stay-Apparatus: An arrangement of muscles, tendons, and ligaments that work together to "lock" major joints of a limb in the extended position. The horse enjoys stay-apparatuses in both the fore and hind limbs, thereby allowing it to remain standing with minimal muscular effort. The mechanism is transiently employed during locomotion and fully employed during periods of upright sleep.
Striking: Contacting a limb with a hoof.
Subacute: Of fairly recent onset. "This is a *subacute* lameness; it developed last Tuesday."
Subluxated: Partial dislocation or displacement of one object (usually bone) relative to another.
Suspensory Desmitis: Inflammation of the suspensory ligament body and/or its branches.
Synarthrodial Joint: A fibrous junction that allows little or no motion between articulating bones.
Syndesmodic Joint: An immovable joint in which bones are joined by dense connective tissue.
Synovial: Associated with a joint, tendon sheath, and/or bursa (which are all *synovial* structures).

T

Telemedical Evaluation: Remote analysis performed through the use of telecommunications technology (e.g. electronic devices such as smart phones and computers).
Tendon: A band of fibrous collagen tissue that attaches muscle to bone. Tendons serve to move structures relative to one another.
Tendon Sheath: A "sleeve" that facilitates tendon movement over or under joint surfaces via synovial lubrication.
Tendonitis: Inflammation of tendon tissue.
Tenobursitis: Inflammation of tendon tissue within the confines of a synovial bursa.
Tenopathy: Tendon disease.
Termino: A desirable action in the Peruvian Paso horse; outward swinging of the forelimb that emanates from the shoulder joint.
Thread: The character of transitions demonstrated by the Peruvian Paso horse from the walk up and through the faster gaits.
Torsion: Twisting or wrenching of the horse's body or body part.
Transverse Plane: Passes through the head, body, or limb perpendicular to the part's long axis.

U

Unilateral Lameness: Altered movement manifesting on one (either right or left) side of the horse, but not both.

V

Ventral: Toward the lower aspect (bottom) aspect of the head, neck, belly, and pelvis. Does not refer to the horse's limbs. "The horse's sternum is *ventral* to the withers."
Ventroflexion: Bending of the spine in a way that moves either end toward the ground surface.

W

Winging-In: The foreleg swings to the inside (toward the horse's midline) during protraction. Also known as *dishing.*
Winging-Out: The foreleg swings to the outside (away from the horse's midline) during protraction. Also known as *paddling.*
Wrong Lead: Leading with the outside (rather than the inside) forelimb while turning or circling.

SECTION I

Our Responsibility to the Performance Horse

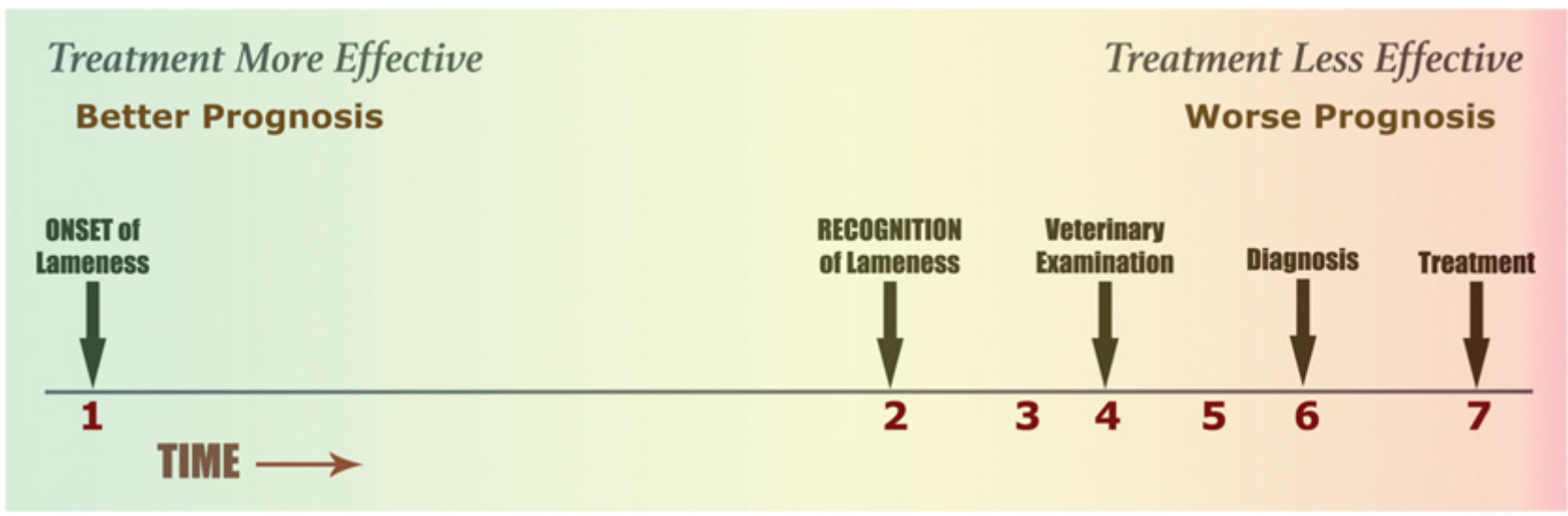
Biological tissue **pathology** does not come without cost. Even if the problem is mild, its persistence over an extended period of time can have permanent consequences with regard to an animal's eventual comfort and performance. As time passes, our ability to successfully manage lameness becomes more challenging due to the natural progression of primary and secondary pathologies. Physiologic abnormalities that can be successfully treated (and in some cases reversed) early on may acquire varying degrees of permanence if left unaddressed for enough time. From this standpoint, the duration of lameness has an indirect relationship with the horse's **prognosis** for future performance. Most veterinarians know this and would, therefore, prefer to treat **acute** severe inflammation (that recently developed) as opposed to **chronic** mild inflammation (that has been present for a long time).

The key to successful management lies in our ability to detect the problem during our "window of treatment opportunity"—that is, the phase when treatment will still be curative or at least highly effective. As many of us have learned the hard way, recognizing a problem after it has already reached the chronic phase makes successful management more difficult and, in some cases, impossible. We would all agree that management certainly gets more expensive as time goes on.

The key to maintaining long-term soundness in the horse entails proactive prevention (prophylaxis) as opposed to reactive treatment.

Once a problem has been recognized, we tend to be fairly proficient with regard to seeking professional consult, performing the necessary diagnostics, and implementing appropriate treatment. Veterinary research in the field of equine sports medicine has primarily been focused on improving diagnostic and treatment techniques that are implemented after the existence of a problem has already been confirmed. A major management dilemma, therefore, lies with the length of time between the *onset* of a problem and its *recognition* (fig. I.1).

I.1 Phases of Lameness Management



1. The onset of lameness. This represents the moment or period when the problem first occurs.

2. Local recognition of lameness. This represents the moment when the primary caretaker (owner, trainer, barn manager, friend, farrier) discovers the problem.

3. Veterinary confirmation of lameness. This represents the moment the veterinarian first becomes aware of the problem.

4. Clinical examination. This may be performed in the field or in a hospital setting. Physical assessment, passive and active soundness evaluations, palpation, flexion testing, and local anesthesia (blocks) are common techniques implemented during hands-on examination.

5. Diagnostic imaging and testing. Radiography, ultrasonography, thermography, nuclear scintigraphy, and magnetic resonance imaging (MRI) are imaging modalities commonly employed in modern work-ups of the lame horse. In some cases, clinical pathology (blood work) and histopathology (biopsy) are also performed to identify specific forms of disease.

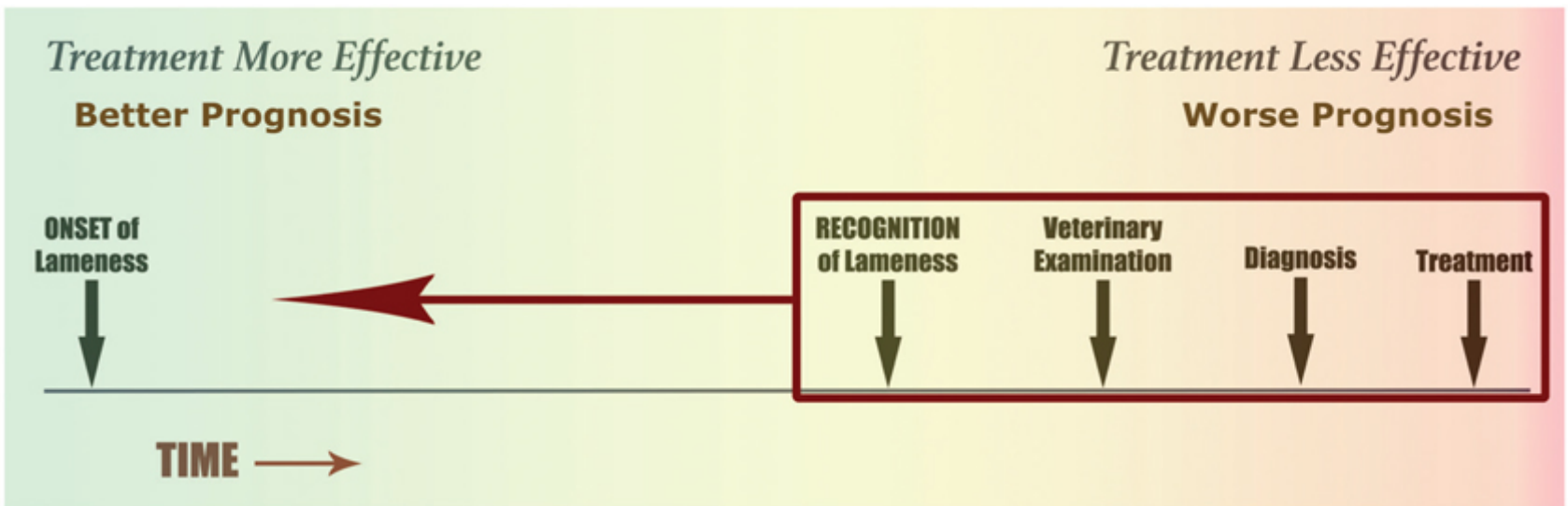
6. Diagnosis. The results of clinical examination, diagnostic imaging, and other tests often enable the veterinarian to reach a diagnosis, which designates the specific cause of the horse's lameness.

7. Treatment. Once a diagnosis is made, an appropriate treatment plan can be formulated for the horse. Corrective shoeing, systemic arthrotherapy (joint supplementation), local arthrotherapy (joint injections), extracorporeal shock wave therapy (ESWT), regenerative therapy (e.g. stem cells), and chiropractics are strategies frequently employed in the treatment of equine lameness.

We can shorten the length of time between the onset of lameness and its recognition by:

- Improving the ability of the local caretakers (horse owner, trainer, farrier) to visually detect subtle lameness.
- Improving dialogue between the local caretaker (horse owner) and veterinarian during the early stages of compromised performance.

I.2 Earlier Recognition of Lameness



Once the veterinarian is made aware of a performance issue, he or she can initiate the process of lameness confirmation, either through on-site evaluation or remote video review. The advent of **telemedical (remote) evaluation** allows the veterinarian to more quickly and easily become a part of the recognition process, a task previously restricted by time, distance, and expense. This concept typifies the foundation of *equine sports wellness* within the veterinary industry.

1

The Responsibility of the Horse Owner in Successful Management of Equine Lameness

Veterinary examinations are not performed on horses that are considered to be sound by their owners. It is the horse owner, not the veterinarian, who is best situated to initiate the processes of lameness diagnosis and treatment. Accordingly, observant horse owners make *better* horse owners. Unfortunately, most horse owners and trainers are not proficient at lameness recognition.[2] Consequently only problems that are obvious, chronic, or advanced tend to receive medical attention.

The utilization of basic visual assessment techniques can help horse owners detect lameness more quickly, thereby starting the diagnostic process sooner and improving the horse's prognosis for future soundness. Local trainers, farriers, and friends can also assist the owner in prompt lameness recognition. Remember, a veterinary degree is not required to formulate an opinion as to the existence, location, and possible cause(s) of a horse's lameness.

At the end of the day, equestrians want to stay in the saddle as long as possible and spend as little money as possible doing it. But this relies on our ability to serve as the frontline "sensors" for lameness in our horses. The faster we're able to recognize a problem, the faster the veterinarian can initiate the diagnostic, treatment, and recovery processes.

Horse owners who adopt a *proactive* approach to detecting lameness in their own horse tend to be more successful in whatever equine discipline they undertake. Those that can recognize subtle gait deficits will recognize small problems *before* they become big problems. The more timely problems are recognized and addressed, the less likelihood they have of becoming long-term or permanent issues. There is also less opportunity for other primary or secondary problems to develop. With fewer areas of the horse being affected, our visual depiction of asymmetry becomes appreciably less complicated.

Your ability to detect lameness will help you to:

- Keep your horse in consistent work.
- Save you money by staying ahead of problems that would otherwise incur increased diagnostic and treatment costs.
- Improve your horse's chances of performing better for longer.

The primary objective of this book is to shorten the time frame between the onset of your horse's problem and your recognition of it.

The ability to localize the potential source of lameness is also very useful to the horse owner. The recognition of gait deficits consistent with a shoulder problem, for instance, tells the owner that the horse is not suffering from yet another foot bruise. With this knowledge, appropriate measures for further diagnostics and treatment can be initiated swiftly. Competence at differentiating problems that pose performance-limiting risk from those that do not is extremely valuable to equestrians at all levels.

2

The Responsibility of the Veterinarian in Successful Management of Equine Lameness

Thirty years ago, visual examination was the primary tool that veterinarians employed to evaluate lame horses. Back then I was fortunate enough to work under the tutelage of legendary veterinarians like Ted Stashak (Colorado State University) and Marvin Beeman (Little Large Animal Clinic), who would extract vast amounts of information during their clinical lameness examinations. Through meticulous inspection and a methodical approach, these practitioners would formulate a visual impression of the horse's movement for the purpose of discerning clinical significance(s). The ability to "decode" individual gait characteristics enabled them to more quickly and accurately diagnose problems. Exceptional clinicians like Drs. Stashak and Beeman (affectionately referred to as "leg men") realized that horses with similar pathology would display consistent patterns of abnormal movement (or *gait deficits*). This approach to evaluation was more representative of "art" than medicine, but often allowed the practitioner to accurately interpret the relationship between certain physical gestures and probable sources of lameness.

Nowadays, veterinarians don't rely nearly as much on visual examination during their diagnostic workup of the lame horse. This is largely attributed to recent advancements in diagnostic imaging and inertia (motion) sensing. A variety of supplemental tools (also known as "toys") allow today's veterinarian to reach a diagnosis with more confidence and less subjectivity.

Unfortunately, the improved technology also tempts today's veterinarian to spend less time and energy on visual examination and more time retrieving electronic data from a machine. Although we gain valuable information via the use of advanced diagnostic modalities, we can also sacrifice time and direction by compromising the visual aspect of the clinical evaluation.

We rely on the veterinarian to accurately diagnose and treat lameness. Equine veterinarians can do both the horse and the horse owner a further service by getting involved in the recognition phase of lameness management. Through the use of regular telemedical evaluation (remote video analysis)[3,4], the veterinarian can help the owner avoid allowing subtle issues to go unchecked.

This strategy can also facilitate post-treatment management of lame horses by providing the veterinarian with regular "virtual views" of their patients in motion. Adjustments to the treatment strategy can be made as necessary depending on updated visual impression(s). Of course, this tool becomes more valuable as the veterinarian's ability to scrutinize footage continues to improve.

> *The clinical examination, although subjective, is still the most important diagnostic modality available because the information gathered during this portion of the evaluation tells the veterinarian where to look. And a veterinarian who knows where to look for the problem is more likely to find it.*

SECTION II

What Is Lameness?

Lameness in the horse has been around for as long as the horse has been around. Before horses were domesticated, lameness became a factor when it precluded animals from keeping up with the herd or avoiding predation.

Nowadays lameness is, if nothing else, the most common reason why horses are unable to perform their job.[5] This form of debilitation is much less of a hindrance to humans who have devised ways to survive by doing things that involve very little (if any) physical exertion (such as writing a book). Every modern horse, on the other hand, is a bona fide athlete. In order to perform its job properly, the equine athlete must be physically comfortable and sound. This is true for racehorses as well as trail horses. Even the retired horse has to ambulate comfortably. This concept becomes clearer as we begin to compare the careers of average horses with those of professional human athletes.

3
The Definition of Lameness

Lameness can be defined as any alteration of the horse's normal gait. A lame horse alters the way it moves to avoid **torsion** around painful **joint(s)** and to center **ground reaction forces.** A bruised foot, for example, will prompt the horse to change the way it moves its head, body, and limbs. These changes reflect the horse's attempt to adjust the location and intensity of **peak vertical forces** experienced by the affected limb. This sounds logical, but what does this mean from a visual standpoint?

Perhaps it would be easier to start by defining *soundness.* For the purpose of this book, we will define soundness as regular and symmetric movement of the horse (**VL 3a**). Lameness, therefore, could be counter-described as irregular or asymmetric movement. Any visible difference in movement between the horse's right and left sides would signify lameness. We generally think of lameness involving the limbs, but horses exhibiting irregular movement of the head, neck, or body (which comprise the **median anatomy)** would also be labeled as unsound based on this interpretation (**VL 3b**).

VL 3a
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VL 3b
Scan/Click to view video.
www.getsound.com/tutorials/3b

The lame horse alters its gait to avoid pain, accommodate some biomechanical restriction in movement, and/or maintain balance.

Depending on the degree of asymmetric movement, lameness might be easier or more difficult to discern with the naked eye. Veterinary studies suggest that, on average, the human eye requires approximately 25% asymmetry in movement in order to detect lameness.[6] As we'll learn, however, knowing *where to look, when to look,* and *what to look for* can dramatically enhance our ability to detect even slight variations in movement.

The reality is that every horse is probably lame to some degree; only those displaying enough abnormality to be perceived by the observer's eye will receive further attention. Put more simply, *our ability to distinguish abnormal movement from normal movement is primarily what separates unsound horses from sound horses, respectively.*

4
The Art of Seeing Lameness

Foremost, we should consider the basic rationale behind why horses limp. Is it pain? Is it some biomechanical restriction that prohibits normal movement? Pain might be an inciter in some instances, but the uncomfortable horse actually limps in an attempt to *avoid* pain. The resulting gait abnormality, therefore, is a voluntary action executed by the horse to circumvent the problem as opposed to an involuntary product of the problem. This is an important distinction, because as observers we don't directly appreciate the source of the horse's pain. Rather, *we see how the horse alters its movement to escape it.*

Visual assessment of the horse's physical adjustments relies on the art of *pattern recognition,* which is relatively simple once we know *where* to look, *when* to look and *what* to look for. This concept is familiar to us, because all of us practice pattern recognition in one form or another in our daily activities/jobs. Horse trainers, for instance, regularly evaluate recurring patterns of body language expressed by riders, both correct and incorrect. Farriers, on the other hand, are accustomed to interpreting various patterns of hoof wear. In all cases, abnormal patterns can give us insight with regard to what might be going on behind the scenes. With practice, we will learn to use our pattern recognition skills to depict visual markers of lameness that were previously imperceptible.

*• In the case of **pain-mediated lameness,** we perceive adjustments that the horse makes in an attempt to avoid pain.*

*• In the case of **biomechanical lameness,** we perceive adjustments that the horse makes in an attempt to accommodate a physical restriction or exaggeration in movement.*

*• In the case of **neurologic lameness,** we perceive adjustments that the horse makes in an attempt to maintain balance.*

Improved pattern recognition enables:

- Owners to recognize lameness in their horses more quickly.
- Veterinarians to assist in owner recognition of lameness more quickly.
- Veterinarians to more accurately classify and characterize lameness during clinical examination, thereby streamlining the diagnostic process.

Let's imagine for a moment that a horse's lame limb would visibly change color for us, making it much easier to distinguish it from the other limbs. Most of us have had the ability to discriminate between colors since childhood, so this scenario would make determining the horse's affected limb a breeze.

Now let's imagine that the affected half of the limb (upper or lower) would glow brightly for us. In addition to seeing the affected limb, we would instantly know which aspect of the limb harbored the problem.

Finally, imagine that the specific source of the horse's lameness (such as a certain joint) would flicker for us. In this instance, we could determine the source of the problem simply by looking at the horse. Visual lameness assessment of the horse would accordingly seem much less intimidating to the average observer.

Obviously, we don't have the luxury of using colors and flickering lights to help us determine the anatomic source of a horse's lameness. There are other visual markers, however, that can be just as obvious and just as definitive. It is the goal of this book to make the reader aware of these markers as well as their physiologic implications. The fundamental methodology of proper lameness assessment is highlighted in Figure 4.1 and detailed in section VI (see p. 157) of this book.

4.1 Basic Method of Visual Assessment

A. Initially, you scrutinize the adjustments the horse makes in its median anatomy to avoid or accommodate the problem. The visible expression of these adjustments helps you to identify which region(s) or limb(s) might be afflicted. In this instance, the right forelimb is lame.

B. Next, you determine the nature of the horse's lameness. This can help you to discriminate which aspect of the region or limb is likely harboring the problem. In this example, the horse exhibits a *combination* lameness that suggests that the problem likely resides within the mid-aspect of right forelimb.

C. Finally, you can use the horse's display of characteristic or unique gait deficits to establish a list of potential causes for the lameness. Based on the appearance of this horse's combination lameness, for instance, you might suspect a problem along the right fore fetlock region.

5
Obscure (Hidden) Lameness

Obscure lameness manifests when the horse is unable or unwilling to make the physical adjustments that we as observers require to assess the problem. It is appreciably easier to see lameness when the right side of the horse moves decidedly differently than the left. In the case of **unilateral lameness,** the horse may favor one side by transferring body weight to the other side (fig. 5.1 and see **VL 5a**). This manifests as asymmetrical movement and is readily detected by our eyes.

5.1 Unilateral Transfer of Weight

Lame horses will often transfer weight off the uncomfortable (lame) side and onto the comfortable (sound) side.

VL 5a
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Bilateral Lameness

It may be more difficult to perceive **bilateral lameness,** especially when both sides are comparably (equally) affected. The relative lack of asymmetry displayed by the bilaterally lame horse can make accurate visual assessment somewhat challenging, especially if the visible disparity between sides is less than 25%.[6] It is for this reason that many horses with bilateral gait deficits of similar appearance and degree are deemed to be sound.

In the case of bilateral lameness, the horse may not be permitted to adjust its movement to avoid one problem without exacerbating a separate problem in the **contralateral limb** (fig. 5.2 and see **VL 5b**).

5.2 Bilateral Suppression of Lameness

A horse may not want to favor one limb if it exacerbates a problem (such as pain) in the contralateral limb. In this case, the horse may be unable to underload a sore right front limb without overloading a sore left front limb.

VL 5b

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As you remember, the horse alters movement to avoid pain, accommodate a biomechanical restriction, and/or maintain balance. But what if something prohibits the horse from making the desired physical adjustments? In this case, our visual perception of the lameness may become more obscure even though the source of the problem persists. For example, if both of your knees were equally painful you might walk funny but not necessarily "limp." This is because your bilateral discomfort poses a dilemma: to which leg can you transfer weight without exacerbating your pain? Your inability to visibly limp in this instance doesn't mean that your knees don't hurt—it just becomes more difficult for others to discern that you have a problem.

Multifactorial Lameness

We may also have difficulty perceiving asymmetric movement in horses exhibiting certain forms of **multifactorial lameness,** especially if pathology coexists in a **compensating limb.** For example, a trotting horse with severe foot pain in the right hind limb will often choose to transfer weight to the left front limb, which constitutes the other limb of the respective **diagonal pair.** As observers, we try to use this visible shift in weight to identify and characterize the horse's lameness. However, concurrent left front foot pain might preclude this horse from comfortably and effectively transferring weight off the right hind limb (fig. 5.3 and see **VL 5c**). This presents a quandary because the horse doesn't have a comfortable place to which to transfer the weight. The observer is also in a predicament because the lack of obvious body adjustment may give the false impression that the horse is relatively comfortable.

5.3 Multifactorial Suppression of Lameness

A horse may not want to favor one limb if it exacerbates a problem (such as pain) in a compensating limb. In this case, the horse may be unable to underload a sore right hind limb without overloading a sore left front limb.

VL 5c
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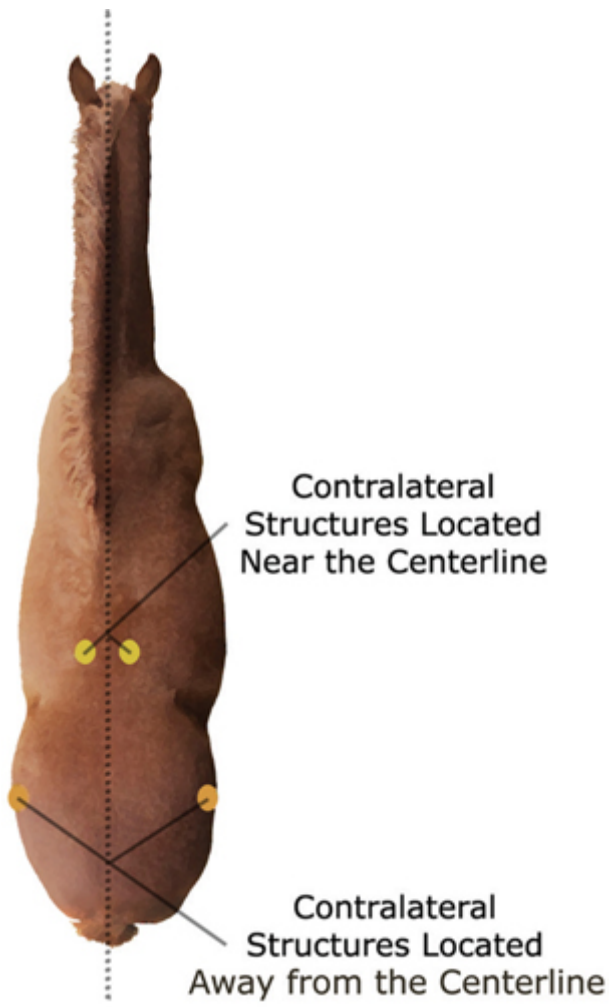
Axial Lameness

Axial structures are those situated along or near the horse's **midline** or **centerline.** They include the head, neck, chest, back, and rump. Due to their proximity to the horse's centerline, asymmetric movement between right and left axial anatomy may be difficult to discern for the observer. This is because disparities in movement become harder to detect as we assess analogous structures that reside closer to one another (fig. 5.4 and see **VL 5d**). Dissimilarities are much easier to see when they are associated with structures farther away from the midline, toward the **periphery** of the horse.

5.4 Axial Suppression of Lameness



The farther away a structure is from the centerline of the horse's body, the easier it is to discern a difference in movement between it and its contralateral counterpart. By contrast, it is more difficult to detect asymmetric movement between contralateral structures located closer to the horse's midline.

VL 5d
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In general, the closer that **contralateral counterparts** are from one another, the more difficult it is to discern asymmetric movement between the two. Consider the manner in which a seesaw displays movement, for example. It is relatively easy to visually appreciate that the ends of the board are moving dissimilarly from one another: one rises as the other lowers. Of course, reciprocal movement is also occurring along portions of the board located near its pivot point at the center of the seesaw. Yet, motion along this area is considerably more challenging to perceive with our eyes.

To further convolute our frustration, horses often respond to back and neck pain by *evading* movement along the affected area(s) rather than altering it. And as you might imagine, it is nearly impossible for most horses to brace one side of the neck or back without significantly limiting movement of the other side. The understated

discrepancy in movement between corresponding axial structures coupled with their mitigated activity (as the horse attempts to avoid pain) can make proper assessment of the median anatomy very challenging for the observer. Even the most seasoned equine performance professionals will usually choose to pursue hands-on palpation, physical manipulation, and diagnostic imaging to supplement their visual impressions and build confidence in their assessment.

> *We should always remember that our lame horses are "talking" to us. They're using a form of "sign language" that is expressed through altered movement. Similar to the way people use gestures to illuminate conversation, horses use them to illustrate what they are experiencing as they move. As effective observers, it is imperative that we learn to listen with our eyes instead of our ears.*

Horses suppressing axial pain that exists independent of any limb deficits will often exhibit general stiffness, resistance and/or poor behavior rather than overt lameness (**VL 5e**). In many instances, a problem is "felt" by the rider rather than seen by a spectator. On the other hand, the size and configuration of the horse's axial anatomy does lend itself to fairly clear expression of inappropriate position, orientation, and carriage. These visible aberrations are characterized in chapter 23 (p. 161).

VL 5e
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Moreover, we have ways of exposing most forms of "hidden" lameness to facilitate our assessment. The principle function of the lameness examination, for instance, is to create a setting intended to increase the horse's asymmetric or irregular movement, thereby allowing the observer to more easily detect the presence of one or more problems. Environmental manipulation strategies are discussed in chapter 26 (p. 183).

6

The Issue of Visual Subjectivity Among Observers

The issue of *visual subjectivity* has become a hot topic in the field of equine-performance medicine. Despite our attempts to "standardize" lameness assessment (via the use of various grading systems), research suggests that veterinarians do not always agree on the location or severity of lameness based on visual impression alone.[7,8] This is primarily why advanced diagnostic imaging techniques and motion-sensing devices have become more popular in recent years.

None of us will observe lameness in exactly the same way, just as none of us views artwork identically. Even so, a wealth of information is both visibly expressed by the lame horse and available to the acute observer. Forfeiting this vital information solely based on our inability to agree with one another is a mistake, in my opinion.

Despite having different strategies for building a jigsaw puzzle, for example, multiple individuals within a group still have a good chance of successfully completing the project. Each may have their own set of self-imposed rules and strategies, which may or may not parallel those of other individuals. For instance, some might start by separating the edges from the inside pieces, finding it is easier to work from the outside inward. Grouping pieces of similar design and color may also help to facilitate completion of certain portions. Others will make use of the picture on the box as a guide. Many of us sort pieces by appearance, whereas another group might use the shape of the pieces to determine their location. Although the knowledge of general puzzle-building concepts would be helpful to all of the individuals, all will find the specific method that works best for them. And in the end, everyone completes the same puzzle successfully.

It is important to realize that multiple observers formulating a multitude of visual impressions still have the ability to reach common conclusions when it comes to the location, degree, and nature of a horse's lameness. Our visual perceptions, if carefully codified, can only facilitate and accelerate the process of accurate diagnosis.

In summary, don't worry if you don't see what others see. It is not important that we all agree on what we perceive, but rather that we reach similar conclusions. Use your own "built-in" detection hardware to find the alterations in movement that will steer you in the right direction. The important thing is that we glean as much as possible from the visual exam before moving forward.

SECTION III

Forms of Lameness

There are many terms used to describe and characterize lameness in the horse (fig. III.1). Gaining familiarity with both the literal and conceptual meaning of each (as it pertains to one's visual interpretation) is vital to becoming a competent observer. The outward expression of various forms of lameness will be discussed in section VI (p. 157).

III.1 Common Forms of Lameness

Limb Lameness	Associated with one or more of the horse's limbs. *Example: Foot abscessation usually causes severe limb lameness.*
Axial Lameness	Affects the median anatomy of the horse, which comprises the head, neck, chest, back, pelvis, and tail. Concurrent limb lameness may or may not be present. *Example: Horses with lower back pain will often display axial lameness when trotting in a tight circle.*
Primary Lameness	Occurs as a result of injury, trauma, or an event outside of the horse's body or control. *Example: A chip fracture in the horse's knee would result in primary forelimb lameness.*
Secondary Lameness	Occurs as a result of an issue or lameness that coexists elsewhere in the horse's body. *Example: A horse suffering from a chip fracture in the knee might develop secondary suspensory desmitis in the other forelimb due to excessive overloading of the latter.*
Simple Lameness	Only one limb or region of the horse is affected. *Example: The recent loss of a shoe might produce a simple lameness associated only with the affected limb.*
Complicated Lameness	Multiple limbs or areas are affected. Complicated lameness can be interpreted as a conglomerate of simple components, since each is assessed separately during the evaluation process. Each complicated lameness comprises at least one primary component; other components might be secondary or tertiary. *Example: Laminitis often results in complicated lameness involving both front or all four limbs.*
Multifactorial Lameness	Refers to complicated lameness with two or more primary components, each of which will require exclusive attention during the diagnostic and treatment processes. *Example: A horse with lower hock pain and forelimb coffin joint disease might be suffering from multifactorial lameness in all four limbs.*
Unilateral Lameness	Affects the right or left side of the horse, but not both. *Example: A loss of a single shoe often produces a simple, unilateral lameness.*
Bilateral Lameness	Affects both sides of the horse. *Example: Navicular syndrome is often considered a bilateral disease, affecting both front feet.*
Quadrilateral Lameness	Affects both sides and both ends (i.e. all four limbs) of the horse. *Example: Grass founder can make all four feet sore, producing quadrilateral lameness.*
Pain-Mediated Lameness	Lameness resulting from something that produces pain (i.e. something that "hurts"). *Example: Foot abscessation results in pain-mediated lameness.*
Biomechanical Lameness	Lameness resulting from something that physically prohibits or restricts normal movement, in the presence or absence of pain. *Example: Fibrotic myopathy of the horse's hamstring musculature can generate biomechanical lameness in the affected limb.*
Neurologic Lameness	Lameness resulting from neuropathy and usually due to a lack of motor innervation and/or reduced proprioception. *Example: Horses diagnosed with wobbler's syndrome often exhibit neurologic lameness.*
Weight-bearing Lameness	Gait abnormalities will be most obvious during the **stance** phase of the stride, when the foot is in contact with the ground surface. *Example: Due to severe pain, foot abscessation often results in obvious weight-bearing lameness in the affected limb.*
Non Weight-bearing Lameness	Gait abnormalities are most evident during the flight phase of the stride, when the foot is airborne. *Example: Fibrotic myopathy of the hamstring musculature often produces a biomechanical, non weight-bearing asymmetry by prohibiting full extension of the affected hind limb.*
Combination Lameness	Lameness displaying both weight-bearing and non weight-bearing characteristics. *Example: A recent chip fracture in the horse's knee often produces simple, unilateral, combination lameness of the affected limb*
Associated Lameness	A secondary lameness that occurs in the same locality and as a direct consequence of a primary issue. *Example: Altered loading of the lower limb due to fetlock joint pain could precipitate associated bruising of the foot.*
Compensatory Lameness	A secondary issue that occurs in a different locale or limb and as an indirect consequence of a primary lameness. *Example: Moderate hock pain might produce compensatory forelimb lameness as a result of chronic overloading and excessive challenge to the suspensory ligaments.*
Referred Lameness	An artificial precipitation of gait deficit(s) occurring in one limb as a product of gait deficit(s) in one or more of the other limbs. Referred gait deficits are not genuine and disappear pursuant to successful treatment of the primary lameness. *Example: A horse might exhibit a primary left hind weight-bearing lameness with a referred left front weight-bearing component.*
Intermittent Lameness	A lameness that appears to come and go from one day to the next. Issues that occasionally provoke biomechanical interference can generate intermittent lameness, as can sporatic bouts of muscle cramping or inflammation. *Example: Equine Hyperkalemic Periodic Paralysis (HYPP), a muscular disease caused by an inherited genetic mutation, can produce symptoms of intermittent lameness in Quarter Horses and related breeds.*
Shifting Lameness	A lameness that appears to move from one limb or region of the horse to another, depending on the day or time of evaluation. *Example: Some horses with bilateral forelimb navicular pain will exhibit shifting-limb lameness; one minute they'll be worse in the left front and the next minute they'll be worse in the right front.*
Metabolic Lameness	Lameness that occurs as a result of something not directly related to the horse's musculoskeletal or nervous system. Colic, pleuropneumonia, and aortic-iliac ("saddle") thrombosis are metabolic ailments that can affect a horse's stride and/or make the horse reluctant to move. *Example: My mare exhibited severe metabolic lameness during her recent bout of pneumonia; she refused to walk down the barn aisle.*
Pathognomonic Lameness	Produces one or more gait deficits unique to a specific pathologic condition. Since pathognomonic deficits are definitive, their detection enables the observer to make a cursory diagnosis of the horse's problem. *Example: "Goose-stepping" of the hind limb is considered to be pathognomonic for fibrotic semitendinosis myopathy.*

7
Primary versus Secondary Lameness

Lameness comes in two basic forms: *primary* and *secondary.* Primary asymmetry occurs as a consequence of an event that originates independent of other preexisting lameness. Trauma, breed, age, and poor conformation could all play a role in the development of primary lameness. Identifying the primary gait deficit(s) is the chief goal of the veterinary examiner, because this is where treatment will eventually be directed.

Secondary lameness, on the other hand, manifests as a consequence of one or more preexisting gait deficits elsewhere in the horse. It can be genuine (as in cases of *associated* and *compensatory* issues) or artificial (as in the case of *referred* asymmetry).

The relationship between primary and secondary lameness is unidirectional (fig. 7.1). Secondary lameness would not exist without the presence of a primary underlying problem. This is an important concept when considering the fact that permanent resolution of secondary lameness would, at least in part, demand mitigation of its primary counterpart. As long as a primary problem exists, the potential for secondary pathology is not far behind.

7.1 Primary and Secondary Lameness

The relationship between primary and secondary lameness is unidirectional.

On the other hand, secondary components may or may not coexist with primary lameness. A single primary lameness with no secondary elements would be classified as *simple.* Simple lameness is the most basic form since examination, diagnosis, and treatment are all directed toward a single anatomic region of the horse.

In cases of *complicated* lameness, however, more than one anatomic region is involved. Differentiating regions that require preferential attention from those that don't is one of the primary objectives of the adept observer. All primary issues will require accurate diagnosis and treatment in order to reestablish the horse's performance. Depending on the duration and nature of secondary lameness, however, exclusive treatment may or may not be necessary. In many instances, secondary issues will spontaneously resolve once the primary issue has been successfully addressed. The smarter approach, therefore, is to identify and treat primary problems *first.*

Consider the analogy of your car's front-end alignment and how it affects tire wear. Poor alignment would be considered a primary issue. Accelerated tire wear would be expected to occur secondary to poor alignment. The expensive and time-consuming application of new tires might improve your car's performance in the short term, but the persistence of poor alignment will repeatedly result in premature tire depletion. The appropriate course of action is unidirectional: fix the car's alignment first, then evaluate the status of the tires to determine if and when replacement is necessary. Successful management of a horse's soundness over the long term requires a similar approach.

Those of us that regularly and carefully observe our horses in motion will see fewer complicated cases because we are more likely to detect gait abnormalities soon after their onset and before additional primary or secondary issues have time to develop. Unless it's due to a common traumatic event, it is relatively rare for two separate, unrelated problems to occur simultaneously. In this way, regular observation actually simplifies the process of evaluation by making the incidence of multifactorial and secondary lameness less probable.

Even so, the majority of lameness cases are complicated. In order to orchestrate our diagnostic and treatment plans appropriately, we must first recognize and designate each component of the horse's lameness as primary or secondary. Although this task may seem daunting to the casual observer, there are some basic guidelines that will help. Knowledge of the typical ways that secondary lameness manifests at the trot can aid in accurate classification.

Consider the following points:

- All lame horses have at least one primary component that is contributing to their altered movement. That said, most lame horses have only *one* primary component. Multifactorial lameness is a relatively uncommon form of complicated lameness.
- There is little correlation between the degree of lameness and its primary/secondary denotation. A severe lameness does not certify a "primary" classification, since secondary lameness is often more visibly obvious

than its underlying primary inciter.

- Forelimb lameness is more likely to be secondary to hind limb lameness than vice versa. Since the forelimbs normally experience the majority (approximately two-thirds) of the horse's weight, very slight hind limb lameness may be enough to significantly influence forelimb load and action. By contrast, marked forelimb lameness is typically required to generate corresponding repercussions behind.
- When forelimb and hind limb asymmetries coexist:
    - If the hind limb component is worse, it is more likely to be primary.
    - If the forelimb component is considerably worse (by more than one grade), it is more likely to be primary.
    - If the severity of the two is comparable (within one grade of each other), the hind limb component is more likely to be primary.
- In my opinion, back and neck issues are more likely to be secondary to limb lameness than vice versa. In fact, secondary sacroiliac (SI) and back pain are anticipated consequences of chronic and/or moderate hind limb lameness in the quadruped.

Common Forms of Secondary Lameness

Compensatory lameness occurs as a result of excessive stress experienced by tissues in one part of the body in response to a primary issue elsewhere in the body. Accordingly, compensatory problems don't typically occupy the same locality as their primary source. In most cases, they occur in a limb experiencing abnormal weight-bearing forces and/or an abnormal **flight path** as a result of a primary issue in a separate limb. Compensatory issues can develop from chronic overloading as the horse attempts to underload the primarily affected limb or from soft-tissue inflammation in cases where the horse is trying to adjust the timing and length of the stride to maintain synchronous movement with another limb.

Compensatory lameness might be observed in the following cases, as examples:

- A horse that developed **laminitis** in one forelimb as a result of chronic severe lameness in the other (contralateral) forelimb.
- A horse that developed proximal **suspensory desmitis** in a forelimb as a consequence of chronic overloading in an effort to underload the contralateral hind limb (of the same diagonal pair).

As adept observers, we should consider the following characteristics that are typically representative of compensatory lameness:

- Compensatory lameness in the forelimb generally occurs secondary to primary lameness in the contralateral forelimb or hind limb. For example, primary issues in the right hind (RH) and/or right front (RF) limbs could precipitate compensatory lameness in the left forelimb (LF).
- Compensatory lameness in the hind limb usually occurs secondary to a primary weight-bearing lameness in the contralateral (other) hind limb. For instance, we would expect secondary symptoms associated with severe weight-bearing lameness of the RH limb to manifest most readily in the LH limb. Unless chronic and severe, it would be unlikely for a forelimb lameness to precipitate compensatory lameness in a hind limb.

Associated lameness, though secondary, does not generate complicated asymmetry and, therefore, does not need to be designated until the veterinary diagnostic phase of examination. Causes of associated gait deficits generally reside in the same locality as the primary source of lameness. Their presence further alters the horse's gait and may affect both the degree and nature of abnormal movement within a specific anatomic region. Variations in the amount of local inflammation and/or modifications in motion or weight bearing can precipitate associated problems within the affected limb.

Examples of associated lameness might include the following:

- **Greater trochanteric bursitis** (also known as "whorlbone") is commonly associated with **distal tarsitis** (or lower hock pain). The greater trochanteric **bursa** is a small synovial sac that lubricates the middle gluteal muscle tendon as it courses over the greater trochanter of the femur just outside of the hip joint. Inflammation within the greater trochanteric bursa often occurs as a result of chronic excessive pelvic limb **adduction** (i.e. pulling the limb underneath the center of the body) during movement (**VL 7a**). This motion results in increased strain of the middle gluteal muscle and its associated tendon. Excessive limb adduction is, in turn, a gait characteristic classically associated with distal tarsitis. Therefore, greater trochanteric bursitis is a common consequence of chronic hock pain.

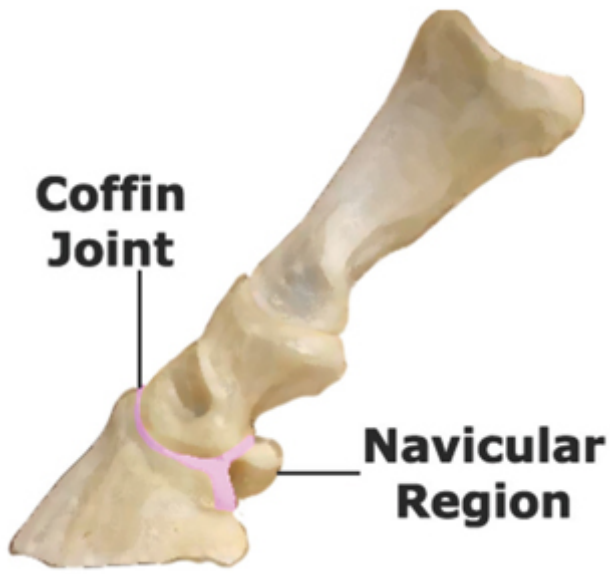
VL 7a
Scan/Click to view video.
www.getsound.com/tutorials/7a

- Lameness within the carpus (or "knee") will often induce inflammation of the brachiocephalics muscle, which forms an attachment between the horse's neck and upper limb (humerus). In an attempt to avoid or reduce carpal flexion, some animals will overuse the brachiocephalicus muscle in order to achieve ample forelimb **protraction** during movement (**VL 7b**). This action, in turn, can result in associated inflammation (termed **myositis**) and pain.

VL 7b
Scan/Click to view video.
www.getsound.com/tutorials/7b

- In some cases of chronic (long-term) navicular inflammation, horses will develop associated inflammation of the coffin joint, a portion of which resides just in front of (or **dorsal** to) the navicular bone (fig. 7.2). In this instance, many professionals implicate the close proximity of the two structures as the reason for the associated lameness: inflammation in the navicular region may "diffuse" into the nearby coffin joint.

7.2 Relative Locations of the Navicular Bone and Coffin Joint



Inflammation originating in the navicular region can indirectly "diffuse" into the coffin joint (highlighted in pink) and vice versa.

Sources of associated and compensatory lameness are genuine in that they precipitate their own gait deficits. Both may persist even after the primary source(s) of lameness are successfully treated.

Referred lameness, on the other hand, is not authentic; it is merely a visible extension of a problem existing somewhere else in the horse. This form of secondary lameness can be expressed in a variety of ways, depending on the location and nature of the primary issue. Areas of the body displaying referred asymmetry do not require direct diagnostic or therapeutic attention since corresponding gait deficits will disappear upon resolution of the primary inciter. Expected manifestations of referred lameness are discussed in chapter 10 (p. 46).

The adept observer not only acknowledges the presence of both primary and secondary components of lameness, but accurately distinguishes between the two. Always considering the potential for physiologic relationships between two or more coexisting gait deficits better prepares us for this task.

8
Painful versus Non-Painful Lameness

To most of us, a lame horse is a horse in pain. While this is true in the majority of cases, gait abnormalities can also be generated by issues that *don't* hurt. The adept observer has the ability to recognize both forms.

As you might imagine, **pain-mediated lameness** is easier to diagnose for the average veterinarian. Hands-on manipulation of the body and limbs (such as **palpation** and flexion testing) can be used to increase discomfort associated with specific anatomic regions, thereby helping the practitioners aim their diagnostic efforts. Local anesthesia **(perineural** and **synovial** blocks) can be used to confirm or deny suspicions with regard to potential sources of pain. The animal's response to empirical treatment in the form of local anti-inflammatory or **arthrotherapy** can also help to implicate various structures as the point of origin.

Non pain-mediated issues, by contrast, cannot be accentuated through manipulation nor be "blocked out" during lameness examination. They are often invisible upon diagnostic imaging and refractory to medical therapy. Many non-painful issues, therefore, can only be diagnosed via their display of characteristic gait deficits. This is where meticulous visual analysis becomes a critical part of the workup.

Non-painful issues comprise those that are biomechanical (usually restrictive) and neurologic in origin. Biomechanical lameness usually results from abnormal interaction between soft tissue and bone. Since **tendons, ligaments,** and **muscles** attach to bone (directly and indirectly, respectively), they are most often implicated as sources for biomechanical lameness. Intermittent upward fixation of the patella (IUPF) and fibrotic myopathy of the hamstring musculature are two well-described biomechanical problems that occur in horses. Each are further characterized in chapters 16 and 19 (respectively) because, like most biomechanical issues, their corresponding gait deficits are distinctive (pp. 91 and 127).

Horses can also exhibit non-painful lameness in response to neurologic disease. Neurologic lameness often arises as a consequence of compromised motor innervation (in which nerves are supplying inadequate input to the muscles that move the body and limbs) and/or decreased proprioception (in which reduced sensory output from the limbs affects spatial awareness). Neurologic lameness can be weight-bearing and/or non weight-bearing in nature (see chapter 9—p. 43), depending on the nerves and structures affected. **Circumduction** is a gait deficit most evident at the walk and often attributed to neurologic disease (**VL 8a**).

VL 8a
Scan/Click to view video.
www.getsound.com/tutorials/8a

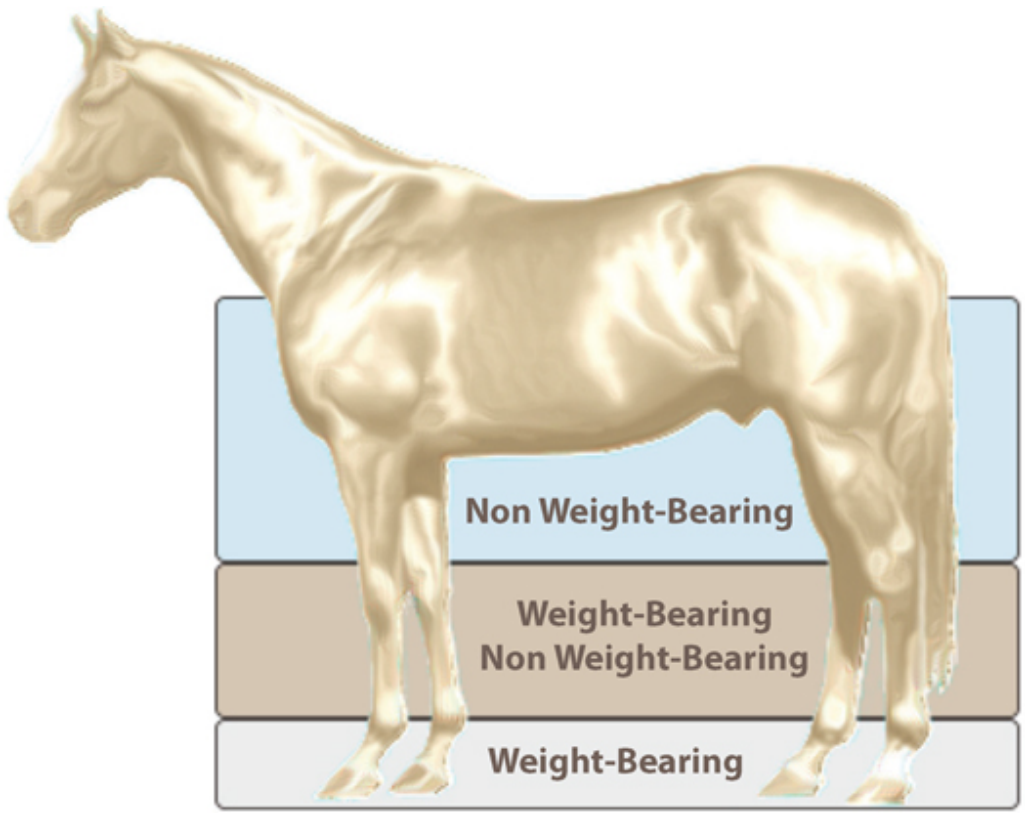
Non-painful issues usually produce non weight-bearing lameness. This is easily demonstrated via the application of a splinted brace to one of your knees. The splint, when properly positioned, should not be uncomfortable nor prevent you from bearing a normal amount of weight on the limb during full extension. Yet it will effectively prohibit flexion of your limb, thereby resulting in a visibly obvious gait deficit as you try to ambulate.

9

Weight-Bearing versus Non Weight-Bearing Lameness

Characterizing the nature of the horse's lameness is one of the key objectives of effective visual examination. Achieving this task in conjunction with identifying the lame limb(s) comprises the foundation of any satisfying assessment. This is because there tends to be a healthy correlation between the nature of a horse's lameness and the general location of its source (fig. 9.1). For instance, we can usually rule out a foot problem in a horse exhibiting purely non weight-bearing lameness in a forelimb. On the other hand, if the same horse subsequently develops severe unilateral weight-bearing lameness a few days after being reshod, there's a good chance that the issue can be successfully addressed with the help of the farrier. This region-specific information becomes invaluable as we navigate through the examination process.

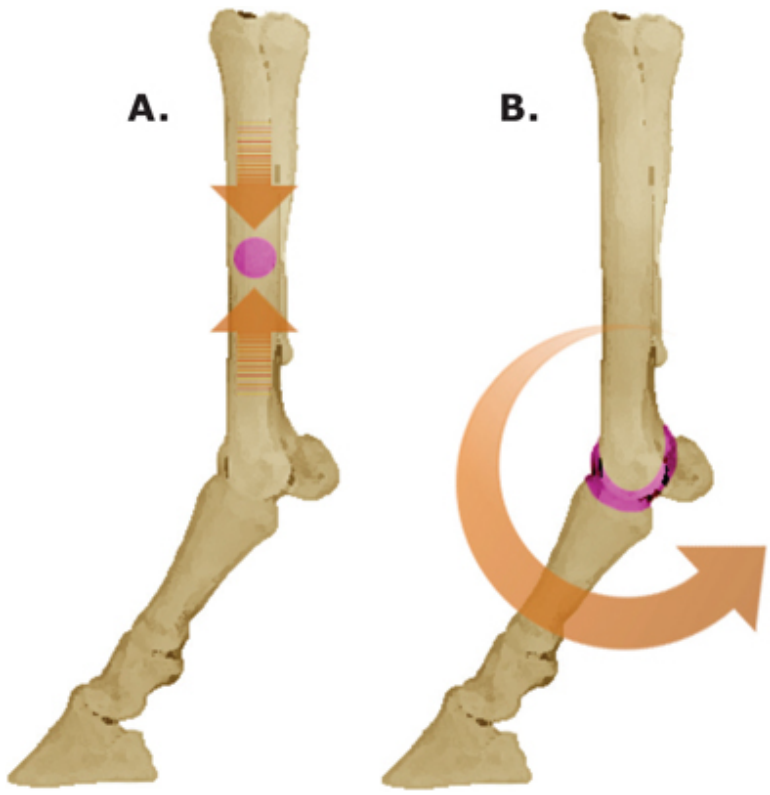
9.1 Relationship Between Limb Region and Nature of Corresponding Lameness



Problems originating below the level of the fetlock joint(s) usually produce weight-bearing lameness. Pathology involving structures within the horse's upper limb often generate non weight-bearing lameness. Issues affecting the horse's mid-limb commonly manifest as combination lameness, comprising both weight-bearing and non weight-bearing components.

As you'll learn in chapter 15 (p. 85), there is also a close relationship between the nature of a horse's lameness and the physical design of the structure that is causing it: structures that undergo load-bearing stress (i.e. bear weight) have the capacity to produce weight-bearing lameness, whereas structures that change their shape during movement tend to generate non weight-bearing lameness (fig. 9.2).

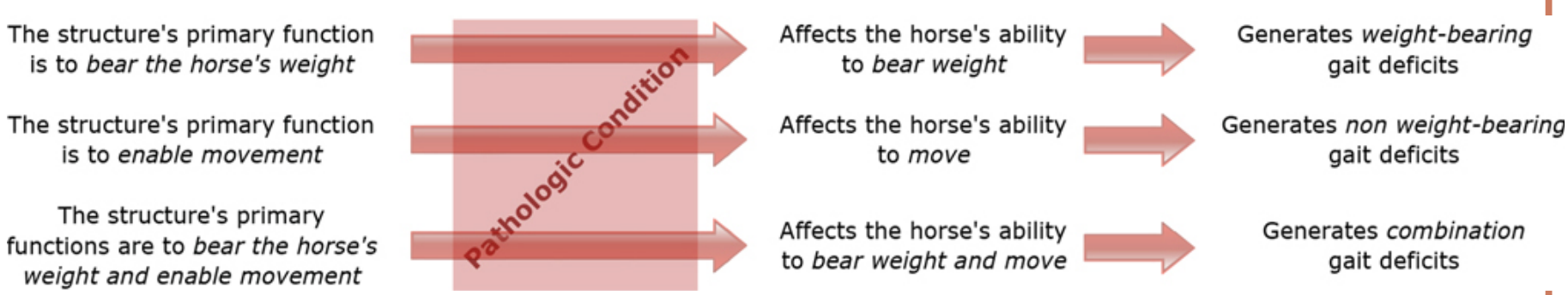
9.2 Relationship Between Anatomic Role and Nature of Corresponding Lameness



A. Structures that "feel" the load of the horse's weight have the potential to generate weight-bearing lameness.
B. Structures that change shape as the horse moves have the potential to generate non weight-bearing lameness.

Knowledge of this interrelationship enables the experienced observer to more easily decipher the true cause of a problem amongst a myriad of possibilities—all based solely on the way the horse moves. This can be achieved by using the nature of the horse's lameness to reveal the primary function(s) of its source (fig. 9.3 and **VL 9a**). For example, the differences in the physical roles of the cannon bone and fetlock joint with regard to load bearing and movement will be reflected in the weight-bearing versus non weight-bearing characteristics of the horse's gait, respectively. Appropriately, issues affecting these structures can often be discriminated without the assistance of local anesthesia (blocks) or diagnostic images.

Figure 9.3 Disclosure of Anatomic Function Based on the Nature of Lameness



VL 9a
Scan/Click to view video.
www.getsound.com/tutorials/9a

As you can see, determining the nature of a horse's lameness is a critical part of our inspection methodology in view of the valuable diagnostic clues that this intelligence provides. Fortunately, this exercise is relatively simple for the informed observer. Several visual markers that are unique to both weight-bearing and non weight-bearing issues enable one to judge the nature of most gait deficits with confidence. The majority of these indicators, which will be highlighted in chapter 24 (p. 170), are relatively obvious once we know which aspects of the horse's gait demand special attention.

10
Authentic versus Artificial Lameness

From a visual standpoint, all gait deficits are "real" in that they alter movement and produce lameness. Some deficits, however, exist for the sole purpose of helping the horse to adjust for a shift in body weight or balance that occurred as a result of a problem somewhere else. Without constant incitement from the primary issue, this secondary "adjustment" would instantly resolve. Since it is nothing more than a visible product of another lameness, we denote this type of gait deficit as *artificial* or *referred.*

Referred lameness is secondary by definition. Even though it isn't considered to be "real" and doesn't factor into the treatment strategy, its existence dramatically facilitates our ability to accurately assess the poorly performing horse. Like all secondary issues, referred gait deficits provide valuable insight into the nature of the primary problem.

Referred deficits, although seemingly obscure, are fairly easy to predict in the lame horse. Proper identification and classification of the primary component usually exposes the basis behind any referred elements. And, as previously mentioned, acute characterization of a referred component can correspondingly lead us to likely primary instigators.

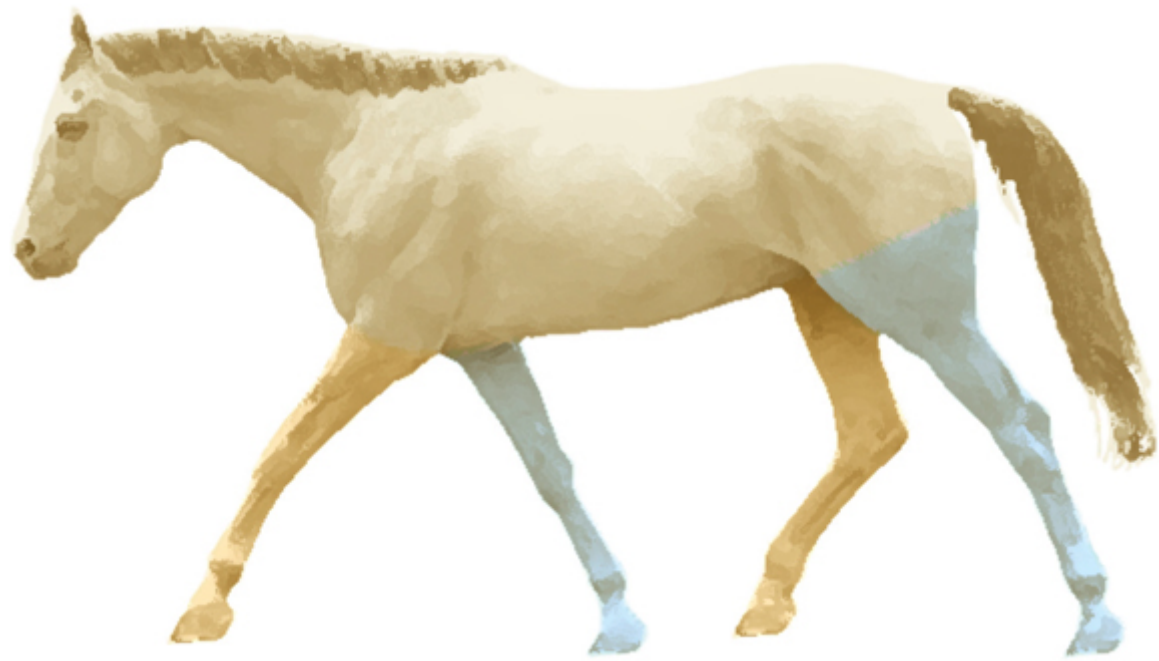
It is important to note that primary deficits will typically generate referred deficits of comparable nature. In the event that referred lameness manifests, the observer will usually find that:

- A primary *weight-bearing* lameness in the forelimb generates a referred *weight-bearing* lameness in the **contralateral** hind limb.
- A primary *non weight-bearing* lameness in the forelimb generates a referred *non weight-bearing* lameness in the contralateral hind limb.
- A primary *combination* lameness in the forelimb generates a referred *combination* lameness in the contralateral hind limb.
- A primary *weight-bearing* lameness in the hind limb generates a referred *weight-bearing* lameness in the **ipsilateral** forelimb.
- A primary *non weight-bearing* lameness in the hind limb generates a referred *non weight-bearing* lameness in the contralateral forelimb.
- A primary *combination* lameness in the hind limb generates a referred *combination* lameness in the ipsilateral forelimb.

The Concept of Diagonal Synchrony

The walk and trot each comprise a two-beat stride pattern in which the horse's weight is distributed evenly between diagonal pairs of limbs. The left hind and right front limbs comprise one diagonal pair, whereas the right hind and left front limb constitute the other (fig. 10.1). The horse maintains similar movement (i.e. synchrony) between the two limbs comprising each diagonal pair at these gaits. In other words, the diagonal pair of limbs move at the same time and in the same way (**VL 10a**). The left pelvic (LH) and right thoracic (RF) limbs maintain concurrent weight-bearing and non weight-bearing (flight) phases of the stride. The right pelvic (RH) and left thoracic (LF) limbs do the same. This form of coordinated movement is known as *diagonal synchrony.*

10.1 Diagonal Pairs of Limbs



The left hind and right front limbs comprise one diagonal pair *(blue)*. The right hind and left front limbs constitute the other diagonal pair *(orange)*.

VL 10a
Scan/Click to view video.
www.getsound.com/tutorials/10a

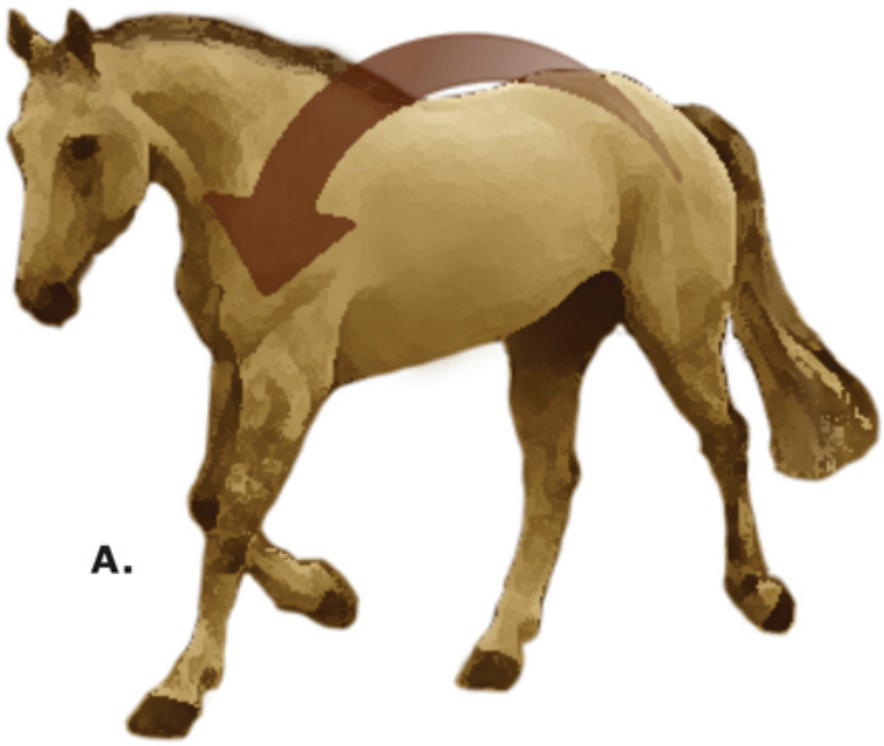
The horse will attempt to maintain diagonal synchrony whether it is sound or lame. Stride-to-stride gait compensation is necessary at the walk and trot to support the horse's weight and maintain balance. This is an important consideration during lameness assessment, because what affects the movement of one limb will also affect the movement of the contralateral limb on the other end of the horse (**VL 10b**). For example, application of a toe extension to the left hind foot as a means of delaying limb **breakover** and lengthening stride will generate a similar gait adjustment of the right forelimb. And as you might expect, the limbs constituting the other diagonal pair will adapt in reciprocal fashion so that the horse can remain stabilized during movement.

VL 10b
Scan/Click to view video.
www.getsound.com/tutorials/10b

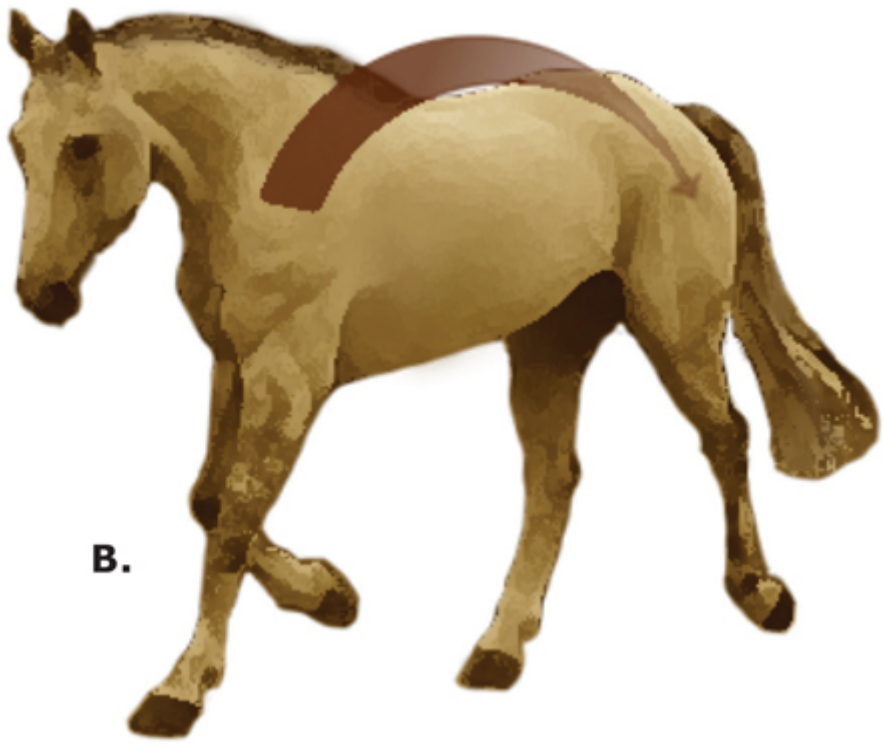
During lameness evaluation, the practitioner must determine whether the origin of each gait deficit is primary in nature or a result of the horse's attempt to maintain synchrony with its diagonal counterpart. In the latter case, lameness would be considered to be artificial or *referred.*

Referred lameness manifests dissimilarly between the front and hind limbs. This is due to a number of factors, most of which incriminate the horse's general physique. Since horses are inherently *front-end heavy,* it is much easier for them to transfer weight in a hind-to-fore direction (fig. 10.2 A). Hind limb asymmetry, even when very mild, can dramatically influence how the horse loads the front end (**VL 10c**). In many instances, the degree of secondary/referred forelimb lameness exceeds that of its primary hind limb complement.

10.2 Dissimilar Transfer of Weight Between the Front and Hind End



A. Since horses naturally bear more load on their front end, it is relatively easy for them to transfer weight from the hind end in a forward direction (toward the front end).



B. Dissimilarly, horses are not very proficient at transferring weight in a backward direction (from the front end toward the back end).

VL 10c
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www.getsound.com/tutorials/10c

By contrast, horses are only marginally effective at transferring weight from the front to the back end (fig. 10.2 B). For this reason, forelimb weight-bearing lameness only generates visible hind limb asymmetry when it is pronounced (**VL 10d**). Moreover, the components of the lameness that get transferred tend to be those associated with stride length rather than load burden. Since the driving motive is usually to maintain balance (rather than shift weight), fore-to-hind referrals are almost always expressed in the contralateral hind limb. This is useful knowledge to the observer, who can surmise that any demonstrative weight-bearing lameness in the hind limb is probably authentic, since it is rare for referred deficits to manifest in this way.

VL 10d
Scan/Click to view video.
www.getsound.com/tutorials/10d

It is important to keep in mind that the severity of the referred component may exceed that of its parent source. In fact, recognition of secondary and/or artificial lameness is often what prompts the horse owner to solicit veterinary intervention in the first place. Examiners who visually separate front and back halves of the animal, evaluate each half individually and actively seek potential relationships between coexisting deficits, will both clarify their assessment and enhance the quality of their interpretation.

11
Axial versus Peripheral Lameness

We generally think of lameness affecting the horse's limbs, but irregular movement can also be demonstrated between different sides of the horse's **axial skeleton,** such as the neck and back. Axial lameness almost always occurs in conjunction with limb lameness because the head, neck, and trunk serve as powerful tools for transferring weight and maintaining balance (**VL 11a**). As a matter of fact, the adept observer will usually appraise movement of the horse's **median anatomy** at the outset of assessment (chapter 20), since it can provide valuable clues with regard to the region(s) or limb(s) that should be evaluated next.

VL 11a
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www.getsound.com/tutorials/11a

Appropriately, many practitioners will presume that axial asymmetry is either artificial (referred) or compensatory if it coexists with **peripheral lameness.** In these instances, the median anatomy will assuredly display unnatural movement to adjust for gait deficits in one or more limbs, thereby making it extremely challenging for the observer to validate the primary contribution of any axial component.

Interestingly, horses with authentic axial lameness are more likely to *avoid* movement altogether as opposed to moving irregularly. As previously discussed (in chapter 5—p. 26), the relative proximity between contralateral axial structures in combination with the horse's emphatic desire to safeguard painful areas can make effective assessment of the median anatomy (by itself) very perplexing.

For these reasons, it is important that we do everything at our disposal to simplify the process: observing the horse on a regular basis (from one day to the next), asking the horse to move in concentrically smaller circles and appraising the effect of a rider's weight are all common techniques intended to clarify our visual assessment of head, neck, and back motion.

The Head and Neck

The horse's head and neck are quite heavy, accounting for almost 10% of the entire body weight. The head is located a considerable distance from the trunk on account of the appreciable length of the neck. As such, it serves as an extremely effective means for transferring weight, particularly that associated with the front end. It is also a very large and visibly prominent structure that can be easily tracked with our eye. Based on these characteristics, the horse's head can essentially be considered a **peripheral** structure that aptly awards the observer with an explicit marker for assessing lameness. In combination with the withers, the head and neck are evaluated foremost during visual lameness assessment (see chapter 23 and **VL 11b**).

VL 11b

Scan/Click to view video.

www.getsound.com/tutorials/11b

Notwithstanding, the horse's head also embodies the aspect of anatomy most often utilized to emit emotion, such as anxiety or resistance. Abnormal movement related to these forms of expression can be very difficult to interpret, and should, therefore, be considered in conjunction with how the rest of the horse's body is moving.

12
Consistent versus Intermittent Lameness

Most forms of lameness are apparent from one day to the next. Consistent lameness usually commands our attention until we accurately identify its cause. Inconsistent or intermittent lameness, on the other hand, is less captivating because we're not always convinced that there's actually a performance problem with the horse.

All cases of intermittent lameness entail some form of instigation or prompting. In other words, there is something that *triggers* the observed gait abnormality(ies). It could be **extrinsic** (such as a rock lodged in the foot, cold weather, very deep footing, a large bandage) or it could be **intrinsic,** resulting from pathology within the animal itself. The key to accurately decoding inconsistent lameness lies in our ability to identify its trigger(s).

As attentive equestrians, it is our duty to make sure that we don't overlook any intrinsic excuses for intermittent lameness.

Tripping

Most horses have tripped at one time or another. Some might stumble on a regular basis while others falter sporadically. Most riders implicate extrinsic causes, such as holes in the ground, dramatic inclines, or deep footing (**VL 12a**). Others believe that their horses are inherently "lazy" and unwilling to pick their feet up enough to clear the terrain. Albeit there are many extraneous causes for tripping, we should always consider the possibility that the horse is faltering as a result of an unsoundness, especially if it occurs with some regularity and/or if certain limbs are exclusively affected. Indeed, there are multiple forms of lameness that feature tripping as a symptom (fig. 12.1). It is important to remember that the inciter may be ever present, even though the horse only trips on occasion.

VL 12a
Scan/Click to view video.
www.getsound.com/tutorials/12a

12.1 Intrinsic Causes of Tripping in the Horse

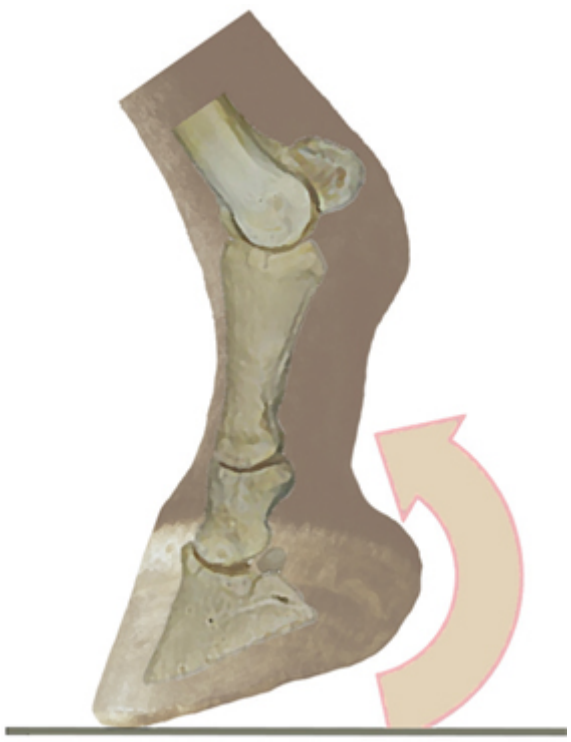
Instigator: *Premature breakover of the limb*—the horse picks the limb up before the amount of leverage required to break over naturally is realized.

Possible Underlying Circumstance(s)	Comments
Excessive toe length of the foot.	The horse's toe(s) may be so long that he/she is unable to acquire enough mechanical leverage to break the foot over naturally. In this scenario, the horse simply "runs out of leg" and has to pick the foot up before the natural breakover process can be initiated. A person can relate to this dilemma by attempting to walk or run while wearing swim fins on his/her feet. The amount of leverage afforded by the fins is too much for the length of the human leg to accommodate. Hence the person is forced to pick his/her feet up prematurely, thereby increasing the risk for tripping.
Excessive tension of the deep digital flexor tendon (DDFT).	The process of foot breakover is initiated by tension along the deep digital flexor muscle and the distal accessory ligament (DAL or inferior "check" ligament), both of which act through the deep digital flexor tendon (DDFT) and suspensory ligaments of the navicular bone (chapter 20, p. 136). Excessive tension and/or pain associated with one or both of these structures can prompt the horse to pick the foot up prematurely, thereby increasing the risk for tripping.
Excessive tension of the distal accessory (or check) ligament (DAL).	
Pain associated with the deep digital flexor tendon (DDFT).	
Pain associated with the distal accessory (or check) ligament (DAL).	
Pain associated with structures that the DDFT influences, such as the navicular and coffin bones.	The events that precipitate breakover ultimately involve anatomic structures that reside within the foot. The horse might choose to pick up the foot prematurely in an attempt to avoid pain if it's generated when increased tension and/or pressure is applied to one or more of these structures.
Instigator: *Decreased proprioception*—a lack awareness pertaining to the spatial orientation and/or movement of one or more limbs.	
Possible Underlying Circumstance(s)	**Comments**
Decreased sensory nerve input that occurs in some cases of neurologic disease.	As you might imagine, a horse that cannot accurately perceive the location or movement of one or more limbs is destined to trip on occasion.
Instigator: *Unwillingness to achieve maximum stride height*—the horse voluntarily avoids normal flexion of the limb.	
Possible Underlying Circumstance(s)	**Comments**
Joint pain, especially when associated with the carpus, elbow, shoulder, stifle, and/or fetlocks.	Some joints undergo considerable flexion during protraction of the limb. Since joint pain is usually exacerbated upon increased flexion, most horses will resist bending the respective limb(s) as much as possible when one or more of these joints is affected. This, in turn, makes them much less likely to clear the ground surface and more likely to trip.
Muscle pain, especially when associated with the *brachiocephalicus, biceps brachii, quadriceps femoris,* and *extensor* musculature.	Certain muscles function to raise and/or protract the horse's limb, either through direct or indirect means. Most animals will choose to disengage these muscles when they're sore, thus inhibiting their ability to clear the ground surface and/or complete a full stride with the affected limb(s).
Instigator: *Inability to achieve maximum stride height*—the horse is not able to achieve normal flexion of the limb.	

Possible Underlying Circumstance(s)	Comments
Muscle fibrosis (scarring), especially when associated with the *brachioce-phalicus, biceps brachii, quadriceps femoris,* and *extensor* musculature.	When scar tissue replaces normal muscle fibers, some functionality of the structure is relinquished. Horses will be more likely to trip if certain muscles cannot be adequately employed to raise or protract the horse's limb.
Inadequate muscle strength, especially when associated with the *brachiocephalicus, biceps brachii, quadriceps femoris,* and *extensor* musculature.	Horses will sometimes encounter a loss of muscle strength and function when they are debilitated due to metabolic issues such as chronic infection, pneumonia, myopathy, and malnutrition. These horses may be forced to exert increased effort in order to avoid stumbling.
Biomechanical interference involving muscles, ligaments, and/or tendons.	Physical interference associated with structures that comprise the horse's stay mechanism(s) can sometimes precipitate stumbling. This is more prevalent in the hind limb (see chapter 18, p. 114).
Referred gait abnormalities.	An obligation to maintain diagonal synchrony and/or accommodate a dramatic shift in weight can cause the horse's secondary limb to trip on occasion.

The act of faltering usually occurs due to an event that transpires at the time of foot breakover (fig. 12.2) and/or during limb flight. Horses that experience excessive tension or pain during maximum limb extension (just before breakover) and/or pain-mediated or biomechanical restriction during flexion (limb protraction) will be predisposed to stumbling. Both of these issues are accentuated by circumstances that delay breakover of the foot and/or increase the length of the stride (an excessively long toe, for example).

12.2 Foot Breakover



During ambulation, the horse's hoof works like a lever with the toe acting as the pivot point. The term *breakover* refers to the action of the hoof as it pivots over the toe to lift and move the limb forward.

Muscle-Related Lameness

Muscles function to mobilize the bones to which they're attached through the action of **tendons.** The kinetic interaction between the bones that comprise the skeleton enables horses to move. The respective dynamics of these bones is also responsible for generating the horse's observed gait. Appropriately, something that affects muscle function will also affect movement of the corresponding bone(s), which, in turn, will influence the horse's gait. And as we know, any alteration of the horse's gait would be deemed lameness by definition.

Muscles require the basics to perform their intended function: adequate nerve input, blood flow, oxygen, vitamins, minerals, electrolytes, for example. Deficiencies or imbalances in these essential elements can negatively affect metabolism, which, in turn, disrupts the muscle's physical application. By the same token, conditions that jeopardize the muscle's ability to assimilate these constituents would also result in tissue damage and compromised performance. The term **myopathy** is used to describe any form of muscle disease, many of which are highlighted in figure 12.3.

12.3 List of Equine Skeletal Myopathies

Myopathies *Not* Induced by Exercise	
Genetic myopathy	Glycogen branching enzyme deficiency (Quarter Horse-related breeds)
Immune-mediated myopathy	Hemorrhagic purpura; immune-mediated polymyositis (Quarter Horses primarily)
Infectious myositis	*Anaplasma phagocytophilum* (anaplasmosis); *clostridial spp.* myositis (bacteria); Equine Infectious Anemia (virus); influenza myositis (virus); local abscessation: *Staphylococcus aureus, Streptococcus equi, and Corynebacterium pseudotuberculosis* (bacteria); *Sarcocystis myositis* (protozoa); *Streptococcus equi* myositis (bacteria)
Nutritional myodegeneration	Vitamin E/selenium deficiency
Toxic myopathy	Ionophore toxicity; organophosphate toxicity
Pasture/plant-related myopathy	*Acer spp.* trees (containing hypoglycin A); blister beetles (cantharidin poisoning); *Eupatorium rugosum* (white snakeroot); *Isocoma wrightii* (rayless goldenrod); *Senna occidentalis*
Polysaccharide storage myopathy	Type I; Type II
Spasmodic myopathy/cramping	Shivers; *Otobius megnini* (tick) infestation of ear
Toxic myopathy	Ionophore toxicity
Traumatic/anesthetic myopathy	Compressive myopathy (compartment syndrome); fibrotic myopathy; malignant hyperthermia (Quarter Horses)
Myopathies Induced by Exercise	
Dietary imbalances	Electrolyte disturbance; hypocalcemia
Exertional rhabdomyolysis	Chronic rhabdomyolysis; recurrent rhabdomyolysis; sporadic rhabdomyolysis
Genetic myopathy	Mitochondral myopathy
Myotonic disorders	Myotonia congenita; myotonia dystrophica; Hyperkalemic Periodic Paralysis (HyPP)
Metabolic myopathy	Pituitary pars intermedia dysfunction myopathy
Polysaccharide storage myopathy	Type I; Type II
Traumatic myopathy	Malignant hyperthermia (Quarter Horses)

Horses with certain forms of muscular disease can experience intermittent lameness. A sudden inflammatory response within muscle tissue (known as *acute myositis*) serves as the trigger in many cases of muscle-related lameness, which has the potential to manifest and progress very quickly. Since the inflammatory response is intermittent, the resulting lameness will also be intermittent. Still, the display of altered movement is usually repeated throughout each myositic episode. In other words, myopathies can manifest as *inconsistent* lameness with a *consistent* expression of gait deficits. This detail can be helpful when trying to differentiate muscle-related problems from those originating within the nervous system.

Myopathic lameness that is instigated by inflammation can present in myriad ways because different muscles can be affected to varying degrees. Accordingly, the horse's display of gait abnormalities is often somewhat obscure. Rarely do multiple cases of myositis present with identical symptoms; most animals merely appear to be "stiff." Horses confronted with a severe myositic episode often resist activity altogether. Of course, other afflictions not excluding colic, pleuropneumonia, neurologic disease, bone **fracture,** laminitis, foot abscessation, tetanus, toxemia, and shock, should always be a part of the **differential list** in the event that a horse is not willing to move.

Muscle-related lameness can also occur secondary to other forms of pathology, particularly those that interfere with normal **innervation,** blood supply, mineral availability, electrolyte balance, and/or metabolism. In these cases, the instigator may be coming from an alternate source, such as the nervous or cardiovascular system. Neurologic, metabolic, and genetic problems that negatively affect muscle size, strength, and/or function, for instance, have the potential to generate related gait deficits. The appearance and consistency of these deficits will be commensurate with the severity and regularity of the underlying problem, respectively.

Fortunately for us, not all muscle-related problems assume a cryptic appearance; some forms of myopathy are profoundly explicit in their demonstration of gait abnormalities. In these cases, related deficits stem from chronic or permanent muscle pathology as opposed to intermittent, dynamic cues that may or may not present themselves from one day to the next. **Fibrotic myopathy** represents a great example of a disorder that displays reliable and definitive lameness. Visible evidence of this condition emerges as scar (or **fibrotic**) tissue develops within damaged muscle. The permanency of scar tissue and its inherent inability to stretch or contract impairs muscle movement in a predictable and consistent manner, resulting in a non-painful biomechanical lameness that often displays distinctive gait abnormalities (see chapter 19 and **VL 12b**).

VL 12b

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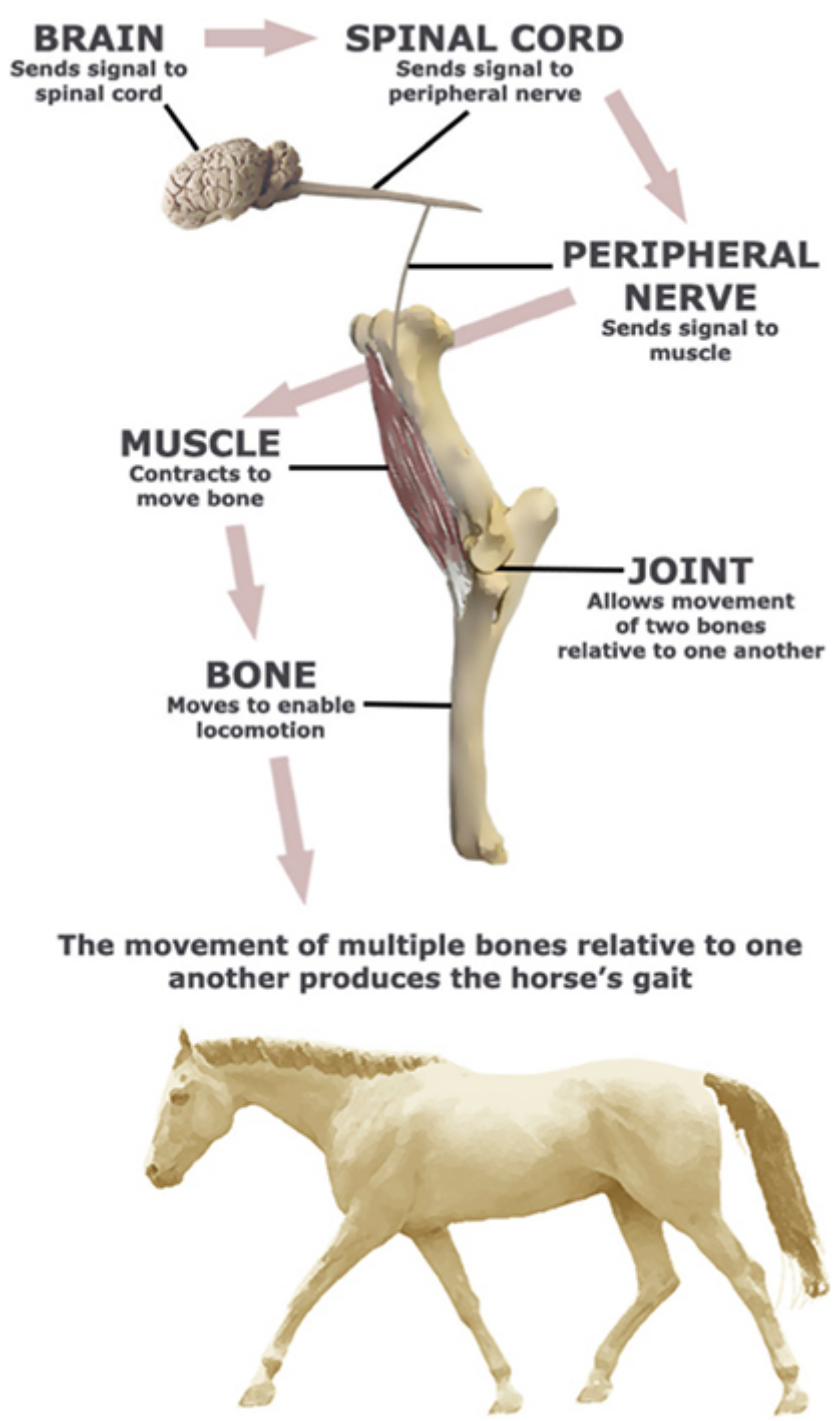
Neurologic Lameness

Like muscles, nerves are affiliated with every part of the horse that moves. Accordingly, neurologic lameness can manifest in an infinite number of ways and affect any or all regions of the horse. The appearance and degree of altered movement arising from neurologic disorders can change depending on the occasion, place, or circumstance, and is directly related to the relative location and extent of nerve impairment (or **neuropathy**) at any point in time.

Neurologic lameness can feature both **central** and **peripheral** symptoms. Central problems are usually exposed in horses that demonstrate general misdirection and/or imbalance, although issues affecting the spinal cord (part of the central nervous system) can produce gait deficits exclusively associated with the limbs (i.e. retain a peripheral expression). Pathologic conditions directly affecting the horse's peripheral nervous system generally affect one or more limbs, but rarely all four.

Peripheral neurologic and muscle-related lameness may be very difficult to visually differentiate from one another due to the physiologic connection between **motor nerves** and muscle tissue: motor nerves rely on muscles to convert electrical signals into movement and muscles rely on motor nerves for activation (fig. 12.4). Ultimately, it is the atypical function of muscle that generates the observed gait deficits in both neurologic and muscle-related forms of lameness. As adept observers, it is our duty to attempt to discern whether the source of the problem is more likely to reside within the nervous or **musculoskeletal system.**

12.4 The Mechanics of Neurologic Lameness



It is always helpful to start by considering the horse's probable motive for altering its gait; this information can assist in our interpretation of obscure lameness(es). In the case of muscle-related problems, the horse is usually trying to avoid excessive pain (as in inflammatory myositis) or accommodate a biomechanical restriction

in movement (as in fibrotic myopathy). Maintaining balance, on the other hand, is the primary objective of most horses suffering from neurologic disease.

Although both neurologic and muscle-related deficits can manifest on an inconsistent basis, we should note that neurologic deficits tend to be less predictable and less repetitive than those expressed within a single bout of myositis. In other words, *inconsistent* lameness with *inconsistent* expression of gait deficits may imply a neurologic source. This presentation combined with the display of visual markers that are often linked to atypical nerve function will prompt us to consider neurologic pathology as a possible cause for lameness (fig. 12.5). We can build additional confidence into our assessment by:

1. Identifying *multiple* visual markers for neurologic lameness (rather than just one) within a single animal *and*…

2. …being unable to attribute the presence of these markers to a single musculoskeletal source.

12.5 Visual Markers for Neurologic Disease in the Horse

Abnormal head or neck movement	Head or neck movement that is adverse to the movement of the rest of the body and/or limbs.
Circumduction	Affected limbs swing outwardly away from the body before moving back toward the midline to contact the ground surface.
Difficulty accelerating or decelerating	These activities require increased muscle strength and coordination.
Difficulty backing-up	The horse drags one or more limbs while backing or resists backing altogether.
Difficulty turning sharply	The horse pivots on one or more feet while turning rather than picking it up to move.
Dysmetria	The flight phase of the stride is excessively high (hypermetria) or low (hypometria). Hyperflexion and/or spasticity of one or more limbs may be observed.
Excessive toe-dragging	Excessive wear along the toe and/or dorsal wall of the foot might indicate chronic, excessive toe-dragging.
Excessive tripping	The horse inconsistently trips in multiple limbs.
Inability to navigate declines	The horse is notably clumsy when going downhill, especially with the head held in an elevated position.
Inability to navigate obstacles	A horse running into or tripping over obstacles may be displaying a lack of proprioception (or spatial awareness) with respect to one or more limbs.
Inability to track in a straight line	The horse appears to drift or turn while attempting to walk straight.
Landing excessively hard on feet	One or more limbs might achieve full extension prior to the foot contacting the ground surface at the walk and/or trot, resulting in an exaggerated impact (**VL 12c**).
Leaning to one side	Inappropriate leaning might suggest central (brain- or vestibular-related) problems.
Limb collapse or buckling	Muscle weakness or paralysis (as a result of neuropathy) may disable one or more components of the horse's **stay-apparatus.**
Hesitance in the stride	An obvious delay in limb protraction occurs as a result of biomechanical interference and/or neurologic disorders.
Variable foot placement	The horse may consistently place one or more feet on the ground in a contradictory or uncertain manner.
Variable gait deficit	The horse's gait abnormalities transform from one minute or one day to the next.

VL 12c
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Since most visual indicators of neuropathy can also be individually-attributed to certain musculoskeletal ailments, we will be more likely to verify their origin within the horse's nervous system if we observe a mixture of symptoms that all lead us to a similar conclusion.

Shifting Lameness

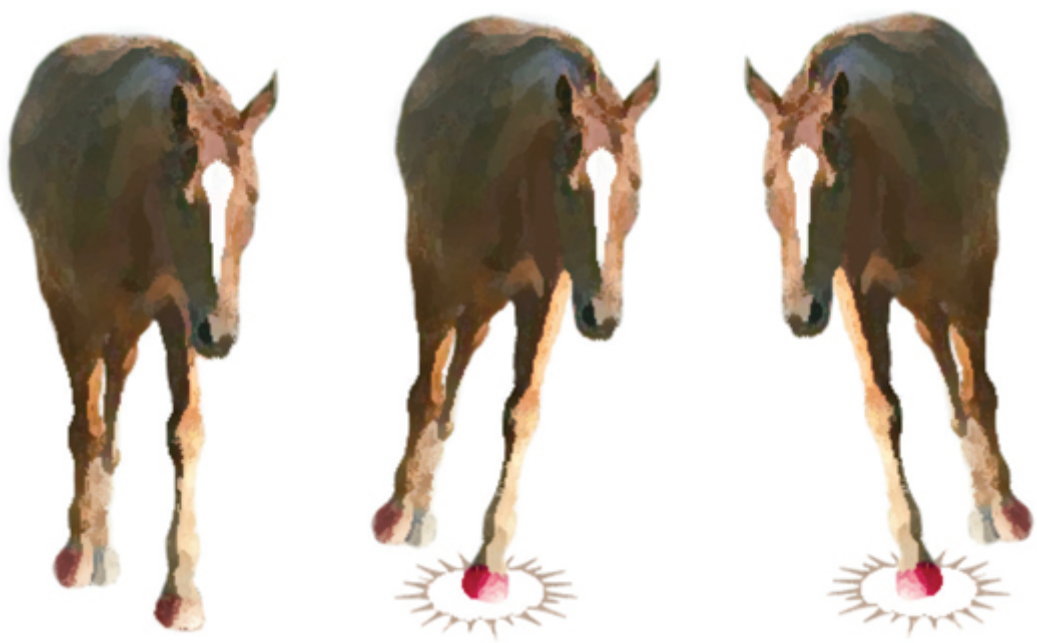
Depending on the moment in time, altered movement will manifest in different regions and/or limbs in horses exhibiting shifting lameness. A horse might appear to be lame in the right front limb one day, for example, and lame in the left front limb the next. In these instances, it is important to remember that, as observers, we are not actually seeing the horse's problem(s) but rather how the horse's body *reacts* to the problem(s). Thus the erratic appearance of shifting lameness is more a reflection of the animal's inconsistent response to pathology and not necessarily a difference or change in pathology.

> *Shifting lameness usually represents multiple forms of expression of the same problem as opposed to the expression of multiple problems.*

Neurologic lameness, which tends to display inconsistent gait deficits on an inconsistent basis, exemplifies this concept very well. It is not uncommon for horses suffering from certain forms of neuropathy (particularly those of infectious origin, such as Equine Protozoal Myeloencephalitis or EPM) to display a variety of gait deficits over a course of time. This occurs on account of the fact that different nerves may be affected to different degrees at different points in time. Still, the pathologic source of the corresponding lameness usually remains constant.

Lamenesses involving multiple limbs (i.e. complicated forms), such as those displayed by horses with bilateral, referred, and/or multifactorial issues may appear to "shift" over time. In the majority of these cases, the inconsistent appearance is triggered by environmental changes (i.e. extrinsic factors) rather than by (intrinsic) pathologic transformation. For example, horses experiencing pain as a result of navicular inflammation (in the feet) will often display bilateral weight-bearing lameness in the forelimbs. In many instances, the degree of lameness is comparable (relatively the same) between the two limbs, making it difficult to perceive as the horse moves in a straight line (see *Bilateral Suppression of Lameness* in chapter 5—p. 27). However, an increase in weight-bearing load experienced by the limb situated along the inside of a turn (or circle) will often precipitate increased pain associated with the respective foot, thereby exposing a corresponding lameness on that side of the horse (fig. 12.6). It is not uncommon for horses with navicular pain, therefore, to display forelimb lameness that can shift from one side to the other depending on the direction of travel (**VL 12d**). Appropriately, we should always consider the possibility of bilateral pathology in horses that exhibit shifting lameness between limbs on the same end of the horse (front or back), particularly if the nature of lameness (weight-bearing, non weight-bearing, etc.) is similar between the two.

12.6 The Inconsistent (Shifting) Appearance of Navicular Pain in the Horse



A. Horses suffering from bilateral forelimb navicular pain of similar degree may not exhibit asymmetric movement while trotting in a straight line.
B. Increased weight-bearing load experienced by the left fore-limb as a result of turning to the left may accentuate navicular pain in the respective (LF) foot, revealing left front weight-bearing lameness.
C. Increased weight-bearing load experienced by the right fore-limb as a result of turning to the right may accentuate navicular pain in the respective (RF) foot, revealing right front weight-bearing lameness.

VL 12d
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Environmental changes can also expose shifting lameness in horses with multifactorial ailments, particularly if each problem responds differently to certain extrinsic factors. Consider a horse with coexisting issues in the left hind suspensory ligament and right front foot, for instance. Since these limbs comprise the same diagonal pair, it may be difficult for the horse to favor one of the limbs without simultaneously increasing challenge to the other (see *Multifactorial Suppression of Lameness* in chapter 5—p. 28). Thus in most instances this horse might actually appear to be sound. If longed on a soft surface, however, we would expect right front foot pain to abate while discomfort associated with the left hind suspensory ligament would likely increase. Accordingly, the horse might appear to develop left hind limb lameness in this setting. Subsequently, longeing the same horse on asphalt might produce the opposite effect: increased tenderness associated with the right front foot and a concomitant reduction of pain associated with the suspensory ligament. In the latter context we might anticipate the appearance of right front limb lameness to prevail.

Generally speaking, gait deficits that shift between different ends of the horse (front to back and vice versa) and feature dissimilar nature (e.g. one is weight-bearing and the other is non weight-bearing) usually have multifactorial sources. By contrast, we should consider the possibility that two lamenesses are related (one is referred by the other) when they each reside on opposite ends of the horse but are of similar nature (e.g. they're both weight-bearing or they're both non weight-bearing).

The concurrent presence of more than one intermittent issue can make accurate visual assessment of the horse particularly challenging, because the instigator(s) are not always easy to identify and may not be readily exposed through environmental manipulation(s). In some instances, the trigger(s) for inconsistent lameness may have only occasional success with respect to generating visible gait deficits. In consequence, accurately assessing a horse that suffers from multiple (coexisting) intermittent issues demands a careful and methodical approach. Consider a horse with sporadic upward fixation of the patella, for example, that may or may not display associated lameness on days that follow a period of stall rest or that are exceptionally cold. The same horse might concurrently harbor a forelimb problem that is temporarily mitigated during warmup, only to worsen with increased exercise (and fatigue). The complexity of our evaluation builds commensurately with the addition of each intermittent form of asymmetry, especially if gait deficits regularly shift from one place to another. Notwithstanding, we still have the ability to decode this elaborate brand of lameness: success lies in our capacity to individually assess each gait abnormality as it is revealed. As you can surmise, solving the puzzle is much simpler once we gather all of the necessary pieces. As most of us have experienced, veterinarians devote an extraordinary amount of time to collecting as much of the horse's performance history as possible with the goal of establishing which instigators (if any) might be influencing the exposure and appearance of the observed lameness.

Rein Lameness

All types of lameness materialize as the horse makes the physical adjustments necessary to accommodate pain, biomechanical restriction, or lack of balance. In the freely moving (i.e. untacked) animal, the motive(s) for making these adjustments are of intrinsic origin; the source(s) exist within the animal itself. Horses under saddle, however, are faced with the added responsibility of accommodating the *rider's* source(s) of pain, biomechanical restriction(s), or faulty balance along with their own. Visible regulation demonstrated by horses that are assimilating abnormal or asymmetric impetus from a rider often generates rein (or "bridle") lameness. This form of lameness is merely a product of extrinsic influence(s) and is, therefore, considered to be artificial or referred. The motive(s) behind the horse's altered movement is the same, it's just that the source is different. Like any referred lameness, associated gait deficits will disappear with removal of the inciter (which, in this case, is the rider).

Riders who endure afflictions that result in asymmetric pain, weight, movement, proprioception, or strength have the potential to transmit those asymmetries to the horse, who is then obligated to counteract the abnormal input via abnormal (or altered) movement. Aberrant stimuli of human (rider) origin might include the following (among others):

- Asymmetric distribution of weight (in the saddle)
- Asymmetric movement of weight (in the saddle)
- Asymmetric saddle configuration
- Asymmetric saddle placement
- Asymmetric saddle movement
- Asymmetric stimulation from the rider's leg
- Asymmetric stimulation along the horse's neck (reins)
- Asymmetric stimulation of the horse's mouth (bit)

Resulting gait deficits demonstrated by the horse can assume weight-bearing, non weight-bearing, or combination features. Changing or removing the rider in these instances can be extremely revealing with respect to the horse's actual level of soundness.

We should note that in the author's opinion, riders are blamed all too frequently for provoking artificial lameness in their horses. Experience has taught us that rein lameness is fairly uncommon, since gait deficits similar to those observed under saddle can oftentimes be detected in the "naked" animal, albeit they may be less conspicuous. In fact, a rider's influence often gives prominence to certain forms of pathology that might otherwise go undetected. Many veterinarians, as an example, prefer to evaluate back problems while the horse is moving under saddle and supporting the weight of a rider. Stifle issues may become more evident during periods of heavy **collection,** which is an activity best prompted by a rider. Diagnosis of temporomandibular joint disease (aka TMJ) almost always entails the help of a rider who, through rein tension, can provoke distinguishing symptoms. In all of these examples, the pathology was preexisting, it simply came to light with the inadvertent assistance of a rider.

Behavioral Lameness

Bad behavior is not a common cause for poor performance but is frequently implicated as a *symptom* of the cause. We usually observe inappropriate conduct in horses that do not have an effective way of avoiding or accommodating their problem(s) through standard means, such as transferring weight from one limb to another. Issues affecting the horse's median anatomy (head, neck, withers, back, sacroiliac joint, etc.), for instance, cannot always be effectively managed through altered movement of the limbs. Consequently, many horses with axial afflictions are defiant but appear to move symmetrically.

For the purpose of being fair to our horses, we should always presume that poor behavior is a result of an unsoundness rather than an inherent problem in the brain, especially if resistance is expressed exclusively during movement or work. The **intermittency** associated with most hostile reactions supports the presence of an underlying instigator (or trigger), the identification of which is critical to arriving at the correct diagnosis.

There are many motivators for bad behavior in the horse, not excluding hormonal fluctuations, diet, genetics, poor training, upper respiratory allergy, upper airway foreign body, tooth abscess, guttural pouch infection, stylohyoid osteitis, petrous temporal osteitis, photic head shaking, and ear infection. For the purpose of this book, we're going to highlight issues that are expressly triggered in the *moving* horse, thereby precipitating visible signs of resistance during exercise. They are associated with the mouth, head, neck, thoracolumbar spine, pelvis, sacrum, and limbs (fig. 12.7). In almost all cases, it is the horse's inherent inability to escape the problem that leads to cantankerous behavior.

12.7 Common Causes of Poor Behavior in the Symmetrically Moving Horse

Oral Cavity/Mouth		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Premolar dental issues	Excessive bit pressure applied to cracked, abscessed, or diseased teeth.	The horse may keep the mouth open, move the jaws from side-to-side, or incessantly stick the tongue out. Some horses will twist or raise the head upon increased pressure from the bit.
Presence of wolf teeth (maxillary first premolars) Mandibular bar osteitis Buccal/lingual mucosal injuries Tongue injuries	Excessive bit pressure or inappropriate bit selection.	
Head		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Photic head shaking	Sunlight.	The horse may incessantly shake the head or develop an involuntary "twitch" or "snap" to the head.
Poor eyesight	Changes in the appearance of the environment and/or replacement of obstacles.	The horse may be excessively "spooky" and/or resist turning in one direction.
Temporomandibular joint disease (TMJ)	Rein tension applied in a specific way and in a specific direction.	Some animals keep the mouth open, move the jaws from side-to-side or incessantly stick the tongue out. Many horses resist turning in one direction (usually toward the affected side).
Osteitis of the occipitus (poll) or dorsal aspect of C1	Pressure applied along the back of the head (i.e. over the poll) from the bridle.	General resistance to tension on the reins and/or oral pressure from the bit. Some horses will rear if related pain is excessive.
Allergic sinusitis	Pressure applied to facial nerves as they exit the infra-orbital foramina along either side of the horse's face.	Many horses incessantly shake the head or develop an involuntary "twitch" or "snap" to the head.
Partial pharyngeal collapse	Flexing at the poll.	Flexion at the horse's poll further compresses the pharynx, increasing turbulence and in some cases significantly obsructing upper airflow. Horses consequently resist flexion at the poll and may rear if they are unable to inspire sufficiently.
Neck		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Cervical vertebral injury or fracture	Bending of the neck in a lateral and/or dorsoventral fashion, such as when turning or assuming a collected frame.	Obvious pain and resistance is expressed while asking the horse to bend through the neck. Affected horses often carry their necks abnormally low but at the same time may be unwilling to drop their heads to the ground. In some cases forelimb lameness and/or generalized ataxia is observed.

Cervical arthrosis (joint disease)		The horse exhibits general stiffness in the neck. In cases of caudal cervical arthrosis (affecting the C5-6 and/or C6-7 articulations), non weight-bearing lameness associated with the ipsilateral forelimb may also be evident.
Cervical vertebral subluxation (dislocation)		Horses will often carry the neck in the extended position (with the head sticking straight out) in addition to demonstrating obvious neck stiffness.
Nuchal desmopathy	Flexing at the poll and/or lowering of the head, such as when assuming a collected frame.	Horses will continually resist rein tension, be unwilling to flex at the poll and avoid lowering the head toward the ground surface. Occasionally affected horses will shake the head and even rear.
Diskospondylitis (intervertebral disc disease)	Dorsoflexion of the neck, which may accentuate disc impingement of the spinal cord.	General neck stiffness, occasionally forelimb non weight-bearing lameness, and in severe cases ataxia.
Jugular vein thrombophlebitis	Bending of the neck in a lateral and/or dorsoventral fashion, such as when turning or assuming a collected frame.	General neck stiffness and occasionally non weight-bearing lameness in the ipsilateral forelimb(s).
Back (Thoracic and Lumbar Regions)		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Vertebral fracture	Direct pressure on affected area(s) and/or excessive dorsoventral and/or lateral bending of the back.	Decreased flexibility and reduced rotation of the back, causing the horse to appear excessively stiff or rigid along affected areas. Plaiting of the forelimbs may be observed. Direct pressure along pathologic regions can trigger pronounced contempt, often inducing rearing, bucking, or refusal to move.
Fracture of the spinous processes		
Fistulous withers	Forward movement and/or cranial pressure applied to the withers by the saddle.	Very short, stiff, and choppy forelimb stride. Many horses will resist movement altogether.
Impingement of the spinous processes (aka “kissing spine syndrome”)	Direct pressure on affected area(s) and/or excessive **ventral** flexion of the back. Often accentuated by rider weight, ill-fitting tack, and lordosis, especially along the T10-L6 segments of the horse’s spine.	Horses generally demonstrate a short/choppy gait quadrilaterally, decreased back flexibility/rotation (increased stiffness), and reduced hind limb propulsion. Horses with severe pathology may “bow-up,” buck, kick out, or refuse to move.
Arthritis of the intervertebral articulation(s)	Excessive dorsoventral and/or lateral bending of the back.	
Rib fracture	Saddle and/or leg pressure at or near the fracture site(s).	
Supraspinous ligament desmopathy	Direct pressure on affected area(s) and/or excessive dorsoventral movement of the back. Often accentuated by rider weight and ill-fitting tack, especially along the T15-L3 segments of the horse’s spine.	
Muscle-related issues	Direct pressure on affected area(s) and/or excessive dorsoventral and/or lateral bending of the back. Often accentuated by rider weight and ill-fitting tack. The nature of instigation depends on the muscle(s) that are affected in addition to the type, location, and degree of pathology affecting the muscle(s).	
Sternal fracture/osteitis	Excessive pressure along the sternum (from the girth).	Very short, stiff, and choppy forelimb stride. Many horses will resist movement altogether. Some horses will object to tightening of the girth; others may rear as it is cinched up.
Subacutaneous thoracic granuloma (girth gall)	Excessive pressure over the granuloma site and/or pinching of adjacent skin (from the girth).	Unilateral or bilateral shortening of fore-limb stride is observed in most cases.
Pelvis and Sacrum		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Pelvic fracture: non-articular (not involving the hip joint)	Applying weight along the horse’s back; asking the horse to walk or trot.	Initially the horse may exhibit severe combination or non weight-bearing lameness in one or both hind limbs, typically displaying a very short and stiff stride. “Hunching” of the lower back may also be observed. An obvious lack of action and propulsion is demonstrated by the hind limbs, which may plait (cross underneath) at the walk. Comfort and soundness usually improve quickly as the fracture fragments stabilize, often within 48–72 hours. Still, most horses will resist abduction of the affected limb(s) and avoid standing on one limb for an extended period of time.
Pelvic fracture: articular (involving the hip joint)		Horses suffering from acetabular fractures of the pelvis are extremely lame (grade 4+/5) and most often demonstrate combination deficits (**VL 12e**). Many are unwilling to bear weight on the affected limb and resist movement, particularly when turning in tight circles. In some cases the horse will “hop” along rather than employ the affected limb. If the horse does use the affected limb, he/she will often orient the body so as to move along three tracks, positioning the comfortable hind

		limb between the two forelimbs. Marked objection to abduction of the limb is demonstrated.
Pubic bone fracture		Fracture of the pubic bone (along the underside of the pelvis) often produces unilateral lameness, although general hind end stiffness is evident. The horse may hold the tail toward the affected side due to spasm of the associated musculature.
Sacral fracture	Asking the horse to walk, trot, and/or jump.	Initially the horse may exhibit severe combination or non weight-bearing lameness in one or both hind limbs, typically displaying a very short and stiff stride. "Hunching" of the lower back may also be observed. An obvious lack of action and propulsion is demonstrated by the hind limbs, which may plait (cross underneath) at the walk. Most horses will resist abduction of the affected limb(s) and avoid standing on one limb for an extended period of time. The tail may demonstrate less mobility or flaccid paralysis.
Sacroiliac (SI) joint disease		Horses with SI joint pain typically exhibit general hind limb stiffness and a shortening of the stride (particularly the forward or cranial component). Associated lameness is most often non weight-bearing in nature, affecting one or both hind limbs. Many horses are reluctant to jump straight (aiming off to one side) or refuse to jump at all. Decreased propulsion and/or plaiting (rope-walking) of the hind limbs may be apparent.
Dorsal sacroiliac (SI) desmitis		Affected horses will exhibit general hind limb stiffness and in some cases bilateral non weight-bearing lameness. They will often resist jumping straight and/or struggle to make sharp turns. Decreased propulsion and/or plaiting (rope-walking) of the hind limbs may be apparent.
Aorto-iliaco-femoral artery (saddle) thrombosis	Asking the horse to walk, trot, or canter.	The formation of a blood clot that compromises blood flow to the hind limbs can be extremely painful, especially during the early stages. This problem is intermittent and usually precipitated by the onset of work (exercise). Moderate to severe unilateral or bilateral non weight-bearing hind limb lameness is observed.
Limbs		
Forms of Pathology	**Instigator(s)**	**Symptoms/Notes**
Delayed patellar release and/or intermittent upward patellar fixation	Canter departs, jumping, going downhill.	During periods of increased hind limb extension, the patella is more likely to "hang up" on the medial trochlear ridge of the femur. It may also be more difficult to disenegage in this setting. In many cases, an obvious "pop" or vibration associated with the affected limb(s) is observed at the moment of limb flexion from the extended position. In more severe cases, the horse may stumble behind, appearing to "fall through a trap door" with the hind limbs. As frustration builds, the horse may refuse to canter, pick up the incorrect canter lead, regularly swap leads, stop, rear, kick out, buck, or bolt.
Quadrilaterally sore feet	Asking the horse to walk, trot, or canter, especially on hard surfaces.	Visible gait abnormalities may be difficult to detect in horses that experience pain in all four feet due to the inherent inability to transfer weight off of one limb without exacerbating pain in another limb. Appropriately, these horses display a very short, choppy, and stiff way of moving, especially on hard surfaces. The horse with severe foot pain might rear, buck, or refuse to move at all.
Fibrotic myopathy of the hamstring musculature	Increased collection or activity behind, particularly during the canter **lead** on the affected side.	The inability of the horse to achieve full extension of affected hind limbs creates a physical dilemma when the rider asks for more engagement of the hind end, particularly if the affected muscles are affiliated with the lead limb at the canter. Many horses will refuse to canter, refuse to pick up the correct canter lead, regularly swap leads, stop, rear, buck, or bolt.

VL 12e
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With respect to any intermittent issue affecting the horse, we should attempt to a) determine if the problem is occurring with enough consistency to justify further investigation and b) use visible evidence to reveal potential trigger(s) for the problem. Accurate identification of any instigator(s) provides us with the means to establish the underlying cause for the observed gait deficits, whether they be musculoskeletal, neurological, or behavioral in nature.

13
Unique Lameness

Unique gait deficits are those that display consistent and reproducible patterns from one animal to the next. Some can be distinctive. For the examiner, visual access to unique patterns of movement is almost like cheating, because fairly bold but accurate deductions with regard to potential cause(s) of lameness can often be forged. In cases of **pathognomonic** deficits, the horse's diagnosis may be visibly expressed in its movement; the observer merely has to accurately interpret what is presented. Knowledge of distinctive gait anomalies, therefore, is extremely valuable to the equine professional. Since many unique deficits are not dictated by pain, their accurate depiction can be central to arriving at the correct diagnosis.

Indubitably, expressive gait abnormalities can provide varying degrees of specificity and precision when it comes to implicating potential source(s) of lameness. Some are very explicit; others are less revealing (fig. 13.1). And of course there are always exceptions. Still, the resolute observer will exploit every visual clue available to facilitate and enhance the analysis.

13.1 Classification of Unique Gait Deficits

	Description	Examples
Lameness traits that are *suggestive*	General features associated with a horse's performance that might be demonstrated on a consistent basis.	**A.** Intermittency is a less-specific trait considered to be unique to some forms of lameness. In general, deficits that occur intermittently (rather than consistently) during the course of evaluation are more likely to have a biomechanical (versus a pain-mediated) source. **B. Laterality** refers to the side or sides of the horse that are affected. Most forms of lameness affect only one side (i.e. are unilateral). Some problems, however, such as navicular disease and distal tarsitis, more often affect both sides of the horse (i.e. are bilateral). **C.** A horse might exhibit a reproducible response to deliberate modification of the environment. For example, a horse with foot pain will usually display greater asymmetry when trotted on a hard surface and/or with the affected limb on the inside of a circle. **D.** Horses might react differently to "warming up." A horse with a tendon injury, for instance, might exhibit increased asymmetry during the course of evaluation whereas a horse with joint pain might appear to improve.
Gait deficits that are *symptomatic*	Horses may display consistent gait aberrations that carry casual significance with regard to potential sources.	**A.** Horses with severe thoracic navicular pain and laminitis might display an obvious and dramatic shortening of the caudal (backward) aspect of both fore strides (bilaterally). **B.** Horses with deep digital flexor tendon injury might increase foot angle and decrease associated tension by sticking the respective toe down into the footing. **C.** Outward deviation and/or rotation of the hock (often accompanied by inward rotation of the foot) during the stance phase of the stride is frequently observed in cases of distal tarsitis and gastrocnemius myositis. **D.** An obvious and excessive "fetlock drop" might be observed in a horse with compromise of the suspensory apparatus and/or superficial digital flexor tendon.
Gait deficits that are *pathognomonic*	Horses may display distinctive gait deficits which, when accurately recognized, can lead the observer to accurate conclusions with regard to the source of the altered movement(s).	**A.** Upward fixation of the patella will prohibit flexion of the hind limb, thereby "locking" it in the extended position. **B.** Fibrotic myopathy of the hamstring musculature can precipitate a characteristic "goose step" of the respective hind limb. **C.** A horse that is able to flex the stifle while maintaining extension of the hock has ruptured the peroneus tertius tendon. **D.** Rupture of the deep digital flexor tendon will cause the respective toe to pitch upward during stance.

Most equine sports medicine veterinarians have compiled and refined a "mental list" of unique gait characteristics during the course of their careers. Details of explicit deficits are often verbally passed from one veterinary generation to the next. Unfortunately, the subjectivity associated with the visual interpretation of these deficits has resulted in a meager body of related literature. Howbeit a detailed list of distinguishing features with corresponding translations is highlighted in chapter 27 (p. 200).

Deliberate and progressive documentation, refinement, and allocation of the patent details of reproducible gait patterns is an essential part of advancing the visual recognition and characterization aspects of equine lameness management. Gait abnormalities considered to be visibly distinctive should be archived by the examiner and categorized for future employment.

SECTION IV

Pertinent Equine Anatomy

An animal's movement is dictated by how all of its body parts work together. Acquiring casual knowledge of equine anatomy will not only help us to understand why our horses move the way that they do, but also enable us to make sense of what we see as observers. Our visual interpretation of normal (sound) and abnormal (lame) gaits will accordingly be much easier.

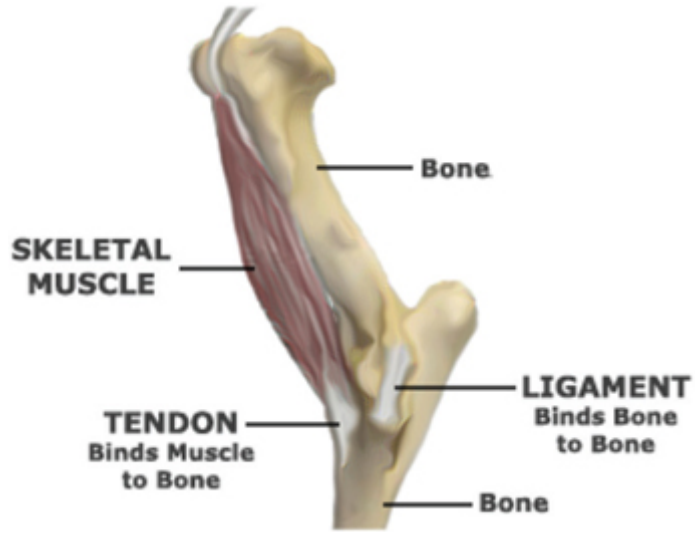
Since this manual is intended to be utilized by lay people as well as professionals, we will not explore all of the intricate details concerning musculoskeletal structure and function. We will, however, discuss the relationship between certain anatomic features and the horse's movement as it is perceived by the observer. Particular attention will be given to structural components that generate characteristic gait deficits when pathologic. Gaining familiarity with these aspects of the horse's anatomy will prove especially useful during our recognition and characterization of biomechanical lameness.

14
Terminology Relating to the Horse's Movement

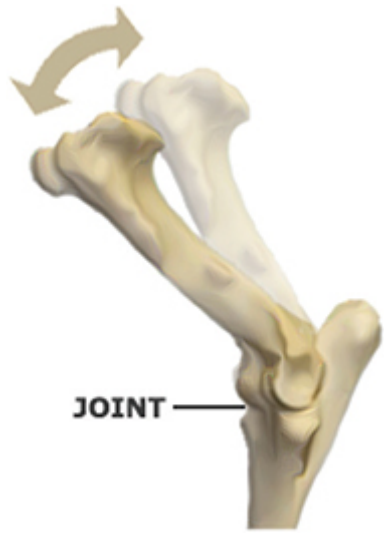
Equine professionals use a variety of terms to characterize motion associated with the horse's body and limbs. Developing a casual acquaintance with this language will both enhance your understanding of equine locomotion as well as heighten your perception of abnormal movement(s).

Structures directly affiliated with the horse's movement include bones, muscles, tendons, ligaments, and joints (figs. 14.1 A & B). These elements work in unison to produce the horse's way of moving (gait).

14.1 Anatomy That Moves the Horse



A. Bones, muscles, tendons, and ligaments.



B. Joints

The horse's skeleton is composed of *bones.* Bones not only form the structural framework for the body, they also move to enable the horse to travel along the ground surface. Horses utilize *muscles* to move the bones. For every skeletal gesture executed via the activation of a muscle, an alternate muscle provides antagonistic movement. Muscles employ the use of *tendons,* which act like ropes to operate various aspects of the horse's skeleton. Bones rely on articulations (or *joints*) to allow them to move relative to one another in a controlled and uniform manner. Joint movement is directionally specific thanks to the combination of their inherent design and the employment of *ligaments,* which function to stabilize the articulating end of each bone comprising it. In addition to permitting movement, joints effectively mitigate concussion by absorbing it into cartilage (which is compressible) and transmitting it to adjacent bones.

Joints are classified based on their relative mobility and/or structural composition (fig. 14.2). As observers, we can readily discern **diarthrodial** (or *synovial*) joint activity due to the extensive range of motion associated with these structures. Synovial joints are subcategorized with respect to their configuration (e.g. condylar, spheroidal) or the nature of motion that they favor (e.g. gliding, hinging, pivoting). In contrast to the fluid action associated with diarthrodial structures, **synarthrodial** motion is virtually unrecognizable and does not appreciably complement our assessment of the horse's soundness. Solitary **amphiarthrodial** joint movement may also be difficult to perceive, although the summation of slight movements contributed by numerous intervertebral cartilaginous articulations situated along the horse's spine provides us with the means to detect deflection (bending) along the neck, back, and tail with relative ease.

14.2 Basic Classification of Equine Joints

Joint Structure	Basic Description	Extent of Motion	Sub-Types	Sub-Type Description	Example(s)
Joint Type: Synarthrodial					
Fibrous	A very rigid ligamentous interface between bones that is essentially immobile (fixed).	Very little or no movement	Sutures	Form delineations between two bony plates that eventually grow together. Minimal motion.	The attachment of various bone plates that comprise the horse's skull.
			Syndesmotic	Bones directly facing each other and stabilized by short ligamentous attachment. Very little motion.	The attachment of a splint bone to the cannon bone.
			Gomphotic	The fibrous interface between a tooth root and bone. Minimal motion.	The attachment of a tooth to the surrounding bone socket.
Joint Type: Amphiarthrodial					
Cartilaginous	Bones interact through cartilage, which allows limited activity.	Limited movement	Synchondrotic	A cartilagenous juncture between two separate structures. Limited motion.	The articulation between the hyoid apparatus and the skull. The articulation between the first pair of ribs and the sternum.
			Symphotic	A cartilagenous juncture between right and left aspects of a single structure. Very low-motion.	The junction between the pubic bones of the horse's pelvis.
			Fibrocartilagenous	A cartilagenous cushion between two bones. Some motion.	Intervertebral discs along the spinal column.
Joint Type: Diarthrodial					
Synovial	Freely moveable. Composed of a synovial capsule, which encompasses the entire joint, a synovial membrane (the inner layer of the capsule), which secretes synovial fluid (to lubricate the cartilage), and cartilage, which covers the ends of the articulating bones.	Full movement	Hinge	Bones move (flex and extend) relative to a single plane or axis.	The elbow joint in the forelimb. The stifle joint in the hind limb.
			Pivot	Allows one bone to rotate relative to another along a transverse plane.	The atlanto-axial joint between the first and second cervical vertebrae.
			Condylar (ellipsoidal)	Allows for flexion, extension, and circular motion (adduction, abduction, and circumduction).	The radiocarpal joint in the forelimb.
			Gliding	Allows for one bone to glide over another along a transverse plane.	The intervertebral facet joints. The lower hock joints.
			Spheroidal (ball-and-socket)	Allows for movement on multiple planes (flexion, extension, adduction, abduction, internal rotation,	The shoulder joint in the forelimb. The hip joint in the hind limb.

				and external rotation).	

When the bones, muscles, tendons, ligaments, and joints are feeling good and working as intended, the horse moves normally. The result is a sound and symmetric gait. When things don't function properly (due to pain, biomechanical limitation, and/or compromised neurologic input) the horse's gait is altered and visible lameness emerges.

The horse's body moves relative to three fundamental planes (figs. 14.3 A–C). Most of us envision the horse's limbs moving along a sagittal plane, tracking from front to back in relatively straight lines that parallel the horse's long axis. While this may be true in a few cases, limb motion ordinarily deviates from this plane by either tracking to the inside (medial) or to the outside (lateral) of it. Depending on the nature and degree of limb deviation, the appearance of the stride may represent a variation of normal or an abnormal defect. If movement is directly related to the horse's conformation and not resulting from an attempt to avoid pain or accommodate another form of pathology then it might be considered to be "within normal reference limits" for the individual.

14.3 Basic Anatomic Planes of Movement

A. The **dorsal plane** passes through a body part parallel to its dorsal surface. This is analogous to the *frontal* or *coronal* plane in humans.

B. The **median plane** divides the horse's body into right and left halves. A **sagittal plane** refers to any plane that is parallel to the median plane but not directly along the horse's centerline.

C. The **transverse plane** passes through a body part perpendicular to its long axis. This is sometimes labeled the *axial* plane in humans.

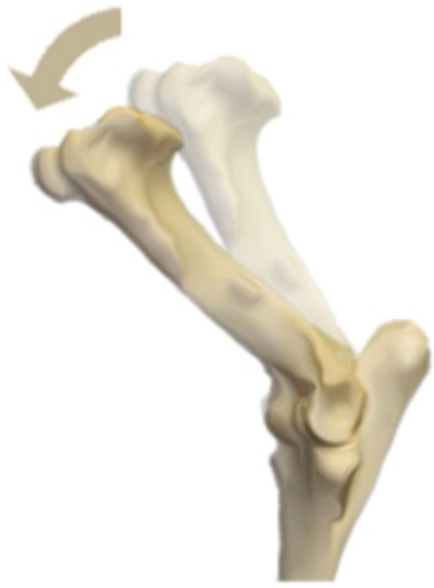
We should also consider the relative position and movement of the horse's body parts with respect to *time* in addition to space, as altered cadence of the stride will generate visible gait deficits, even in the absence of conspicuous deviations in carriage or flight path of the body and limbs, respectively. It is often helpful, therefore, for the observer to use an internal mental "chronometer" that is set to the tempo of the horse's stride during assessment (**VL 14a**).

VL 14a
Scan/Click to view video.
www.getsound.com/tutorials/14a

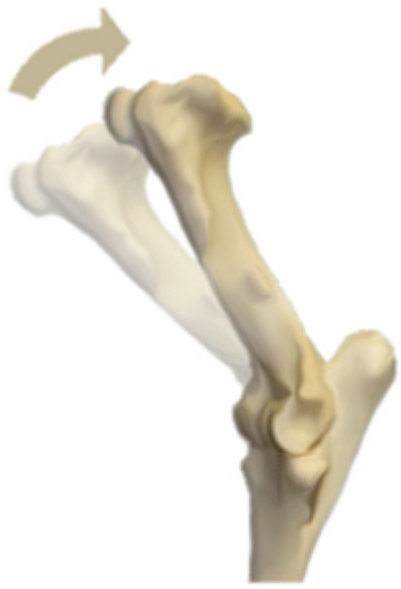
Movement of the Limb and Foot

Flexion and *extension* are joint movements that occur along the sagittal plane and designate an increase or decrease in the angle between two articulating bones, respectively. During flexion, the central portions of the respective bones move closer together (fig. 14.4 A). This activity requires **concentric action** (i.e. shortening) of the muscles that contract to flex the joint. At the same time, **eccentric action** (i.e. relaxation and lengthening) of antagonistic muscles (that normally contract to *extend* the joint) must occur. The opposite occurs during joint extension, during which the bones on either side of the joint move farther away from one other (fig. 14.4 B).

14.4 Flexion versus Extension



A. During *joint flexion,* the angle between the two participating bones is *decreased.*



B. During *joint extension,* the angle between the two participating bones is *increased.*

Elevation refers to movement in an upward direction (such as occurs when the horse picks up the head). *Depression* defines the opposing motion (such as when the horse lowers the head). These terms can be used to describe movement of the horse's axial anatomy (head, neck, back, pelvis, etc.) and/or certain aspects of the limbs.

Abduction is a term used to describe (outward) pendular movement of a limb away from the horse's centerline (fig. 14.5 A). This designation refers to activity along the body's transverse plane and does not pertain to travel along the sagittal (front-to-back) plane. **Adduction** represents inward pendular movement of the limb(s), directly toward the horse's medial plane (fig. 14.5 B). The horse's shoulder and hip joints, which maintain *spheroidal* (i.e. "ball-and-socket") properties, serve as points of rotation for this nature of motion in the front and hind limbs, respectively.

14.5 Abduction versus Adduction of the Limb

A. Abduction of the right hind limb.

B. Adduction of the right hind limb.

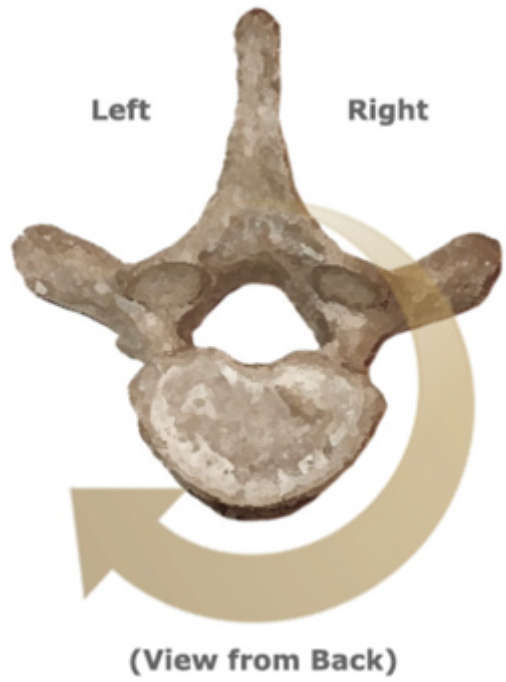
Rotation is used to describe pivoting of a limb along its long axis and occurs relative to the horse's sagittal and transverse planes simultaneously. **Medial** rotation (aka internal rotation) depicts circular progress toward the horse's medial plane, during which the foot assumes a toed-in position (fig. 14.6 A). **Lateral** rotation (aka external rotation) represents the antagonistic action, during which the toe turns away from the horse's centerline (fig. 14.6 B). As in the cases of abduction and adduction, limb rotation is generated within the shoulder (front) and hip (hind) joints.

Rotation also occurs along the horse's vertebral column, which passively spirals (i.e. twists along a transverse plane) as the horse bends the spine from side to side (in a dorsal plane).[10] The direction of axial rotation is designated by the side to which the *bottom* (or ventral aspect) of the vertebral body migrates along a transverse plane (fig. 14.6 C).

14.6 Rotation of the Horse's Limbs and Trunk

A. Medial (or internal) rotation of the limb leads to *toeing-in.*

B. Lateral (or external) rotation of the limb promotes *toeing-out.*



C. Axial rotation occurs in a transverse plane and is described with respect to the directional path of the *bottom* of the vertebral body relative to the horse's pelvis (rather than the top of the structure, as the lay person may be accustomed). In this example, therefore, the horse's spine is rotating to the *left.*

Pronation and *supination* are also used to describe rotational movement although these terms specifically refer to action of the horse's foot (as opposed to the limb). This form of motion occurs relative to the sagittal and dorsal planes of the horse and is primarily engendered within the horse's fetlock joint. Pronation refers to a "rolling in" of the foot, during which the inside aspect drops relative to the outside (fig. 14.7 A). Excessive pronation of the horse's foot upon impact with the ground surface results in a medial-first landing, which can have deleterious repercussions within the inside of the foot and/or lower articulations of the limb. Contrastingly, horses that supinate upon landing will be more likely to instigate lateral pathology—along the outside of the foot and limb (fig. 14.7 B).

14.7 Pronation versus Supination of the Foot

A. During *pronation,* the inside of the foot drops relative to the outside. This results in excessive compression along the inside of the horse's foot and lower limb.

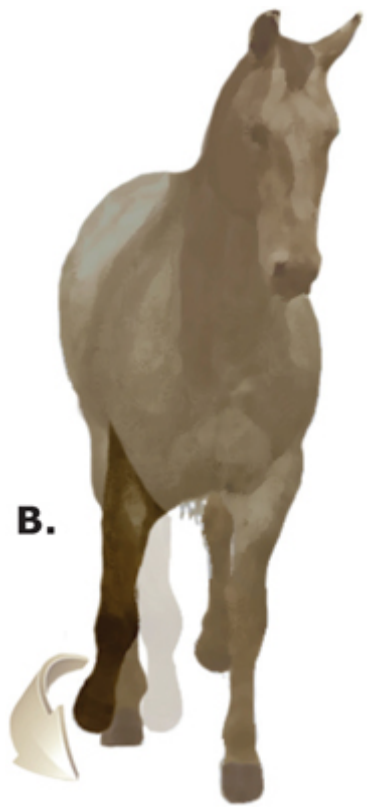
B. During *supination,* the outside of the foot drops relative to the inside. This results in excessive compression along the outside of the horse's foot and lower limb.

Winging-in (aka "dishing") is a denotation used to illustrate adductive movement (or inward "swinging") of the horse's foot during protraction (i.e. the flight phase of the stride) and illustrates movement in both transverse and sagittal planes (fig. 14.8 A and **VL 14b**). The foot will follow a flight path that forms an internal arc relative to the horse's long axis prior to assuming a relatively normal position upon contact with the ground surface. The author's experience suggests that this stride deviation, which can promote excessive wearing along the inside (medial aspect) of the foot or shoe, is displayed more often than straight-tracking or winging-out by the majority of horse breeds.

Winging-out (also known as "paddling") represents concurrent abduction and protraction (or outward "swinging") of the foot, which pursues an external arc relative to the horse's long axis during flight (fig. 14.8 B and **VL 14c**). It often hastens erosion of the outside (lateral aspect) of the horse's foot or shoe.

14.8 Winging-In versus Winging-Out of the Limbs

A. Winging-in or "dishing": The flight path of the foot forms an internal arc relative to the horse's long axis. This stride pattern may be naturally associated with a toe-out (aka "splayed foot") conformation.



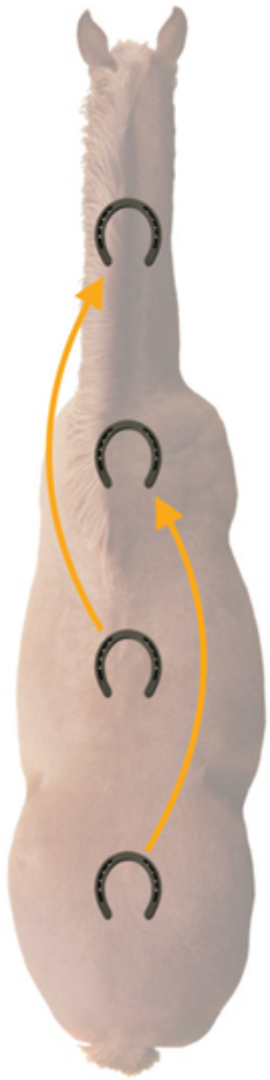
B. Winging-out or "paddling": The flight-path of the foot forms an external arc relative to the horse's long axis. This stride pattern is often observed in horses with a toe-in (aka "pigeon-toed") conformation.

VL 14b
Scan/Click to view video.
www.getsound.com/tutorials/14b

VL 14c
Scan/Click to view video.
www.getsound.com/tutorials/14c

Plaiting is a term used to describe a pattern of foot motion that mirrors "braiding" and delineates the eventual placement of the foot as opposed to the flight path of the limb (fig. 14.9). Horses displaying this gait abnormality will place one foot directly in front of (or in severe cases, even lateral to) the contralateral foot upon impact. Appropriately, the horse often looks as if it is walking along a rope (**VL 14d**). This action often results in limb **interference** (especially of the hind limbs) and is predominantly observed in horses with a base-narrow, toe-out conformation.

14.9 Plaiting (or Braiding) of the Stride



When plaiting (or "rope-walking"), the horse places one foot directly in front (and sometimes even to the outside) of the contralateral (opposing) foot at the walk and/or trot.

VL 14d
Scan/Click to view video.
www.getsound.com/tutorials/14d

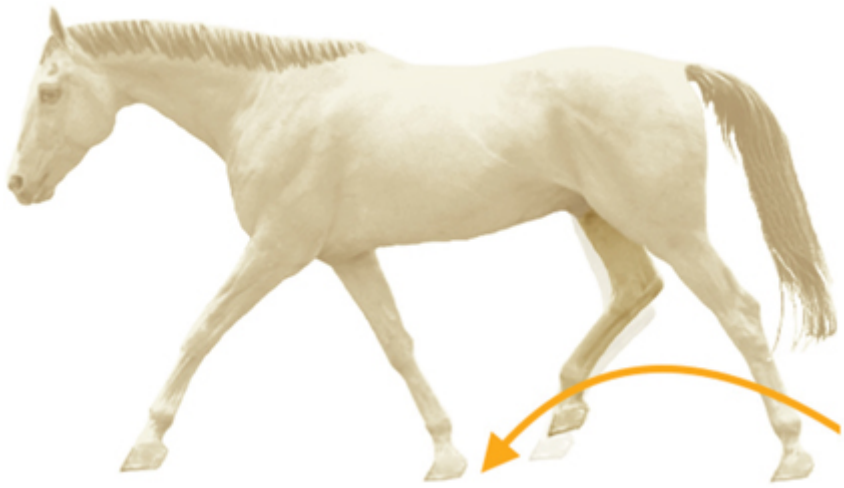
Circumduction describes c*onical* limb movement that is conducted by the shoulder joint (in the front) and/or hip joint (in the back) during the forward phase of the stride. This motion incorporates aberrant abduction, adduction, flexion, and extension of affected limbs, which swing to the outside of the body (away from the median plane) before veering back toward the centerline prior to striking the ground surface, VL 8a, p. 42). This action is similar to winging-out or paddling, except for the following subtle differences:

- The path of the foot more closely approximates that of a semicircle, often wandering far from the horse's centerline.
- In many cases, **caudal** movement of the foot (back toward the horse's hind end) can be perceived prior to its impact with the ground surface.
- In many cases foot impact with the ground surface will be exaggerated.
- Simultaneous external rotation of the limb (resulting in an outwardly facing foot) may be noted.

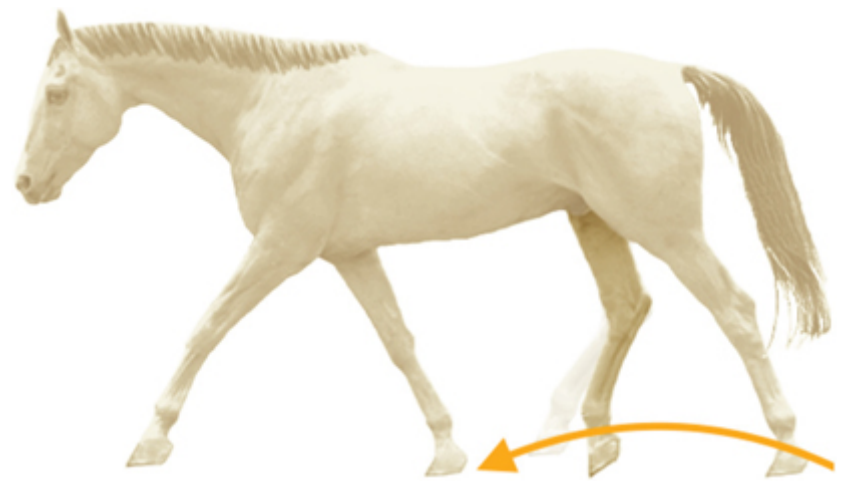
Circumduction is commonly attributed to neurologic disease and is frequently regarded as a marker for compromised *proprioception* (i.e. a lack of spatial awareness).

Dysmetria refers to abnormal stride height along the sagittal plane. Horses displaying a ***hyper*metric** stride will pick their feet up excessively high during protraction (fig. 14.10 A and VL 14e). This action is observed in several forms of lameness, such as string-halt (see chapter 27, p. 197). Horses can also demonstrate ***hypo*metricity** of stride, in which the foot is not elevated to a normal or expected height (fig. 14.10 B and VL 14f). This gait deficit is often detected in horses suffering from high-motion joint disease.

14.10 Dysmetria of Stride



A. The horse elevates the limb(s) excessively when exhibiting a **hypermetric** stride.



B. Horses demonstrating a **hypometric** stride do not achieve full or expected height during protraction of the limb.

VL 14e
Scan/Click to view video.
www.getsound.com/tutorials/14e

VL 14f
Scan/Click to view video.
www.getsound.com/tutorials/14f

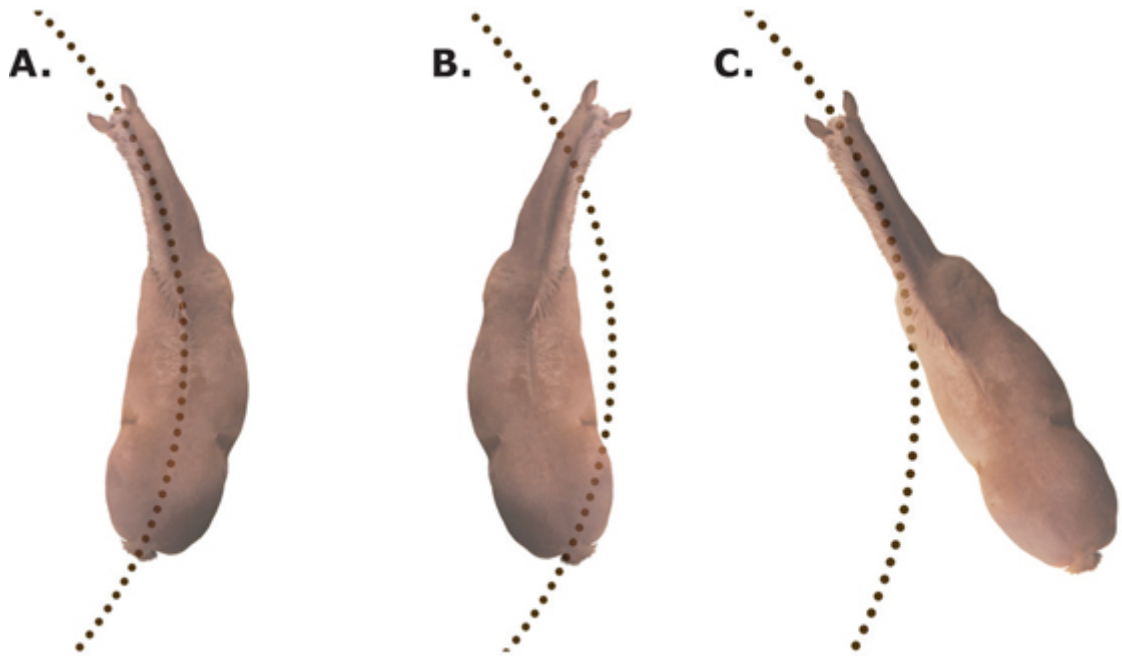
Forging or **overreaching** can occur in some cases, particularly if the stride length of the hind limb is abnormally long. These terms designate a scenario in which the toe of the horse's hind foot inadvertently contacts the heel of the ipsilateral front foot (on the same side). Forging can occur if the hind stride is excessively long, if impact of the hind foot occurs too early, and/or if breakover of the front foot is delayed. **Cross-firing** refers to physical interference of a hind foot with the back of the contralateral forefoot (on the opposite side of the horse).

Interference (or "brushing") of the limbs is a lateral gait deficit, and describes a scenario in which one foot contacts the inside of the opposing limb during flight. This is most commonly observed in horses that wing-in or plait due to the close proximity of contralateral limbs during these activities.

Movement of the Axial Anatomy

During work, horses will naturally bend their spine along the length of their neck and back. *Lateral bending* (from side to side) enables horses to circumnavigate (i.e. execute turns and travel in circles) in balanced and coordinated fashion (fig. 14.11 A). This action almost always occurs in conjunction with *vertebral rotation* on account of the structural design of the articular facet joints.[10] As will be discussed in chapter 22 (p. 145), horses that experience axial pain will often resist normal bending or choose to arc their body in inappropriate ways with respect to the median plane. *Counterbending,* for instance, is commonly demonstrated by horses that aspire to protect a specific region and/or circumvent pain along one side of their body (fig. 14.11 B). This action does not necessarily denote an exorbitant amount of bending, but rather suggests that the horse is curving the body contrary to its directional path of movement. **Drifting** often signifies the horse's desire to *avoid* moving affected areas of the axial skeleton as opposed to moving them abnormally or even at all (fig. 14.11 C and **VL 14g**).

14.11 Lateral Flexion of the Axial Skeleton



A. Normal bending: Normally, horses will bend their bodies to correspond with the directional path of movement.
B. Counterbending: While striving to protect certain regions of the body, some horses will bend in a way that is contradictory to the directional path of movement.
C. Drifting: Many horses manage axial pain by deflecting *any* movement of their median anatomy. These animals often navigate turns in similar fashion to that of a ship on the water.

VL 14g
Scan/Click to view video.
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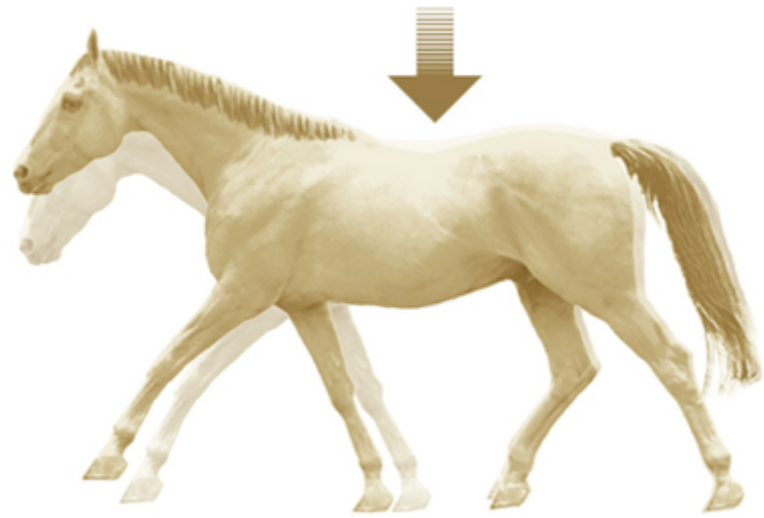
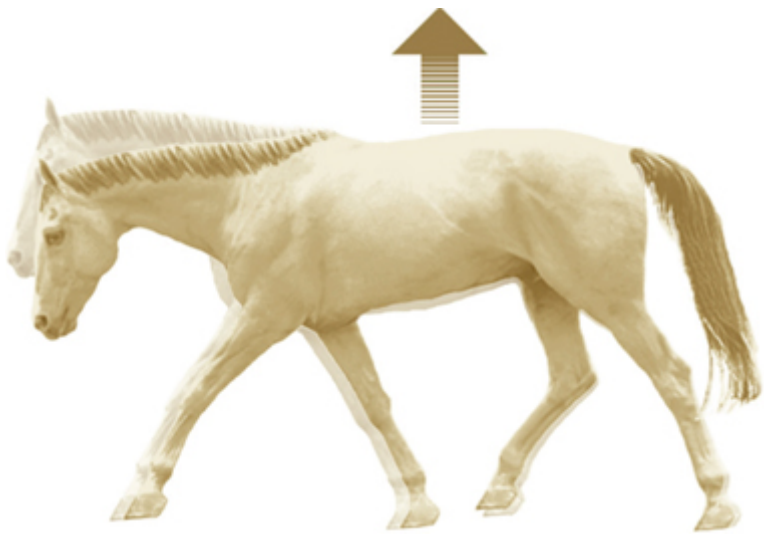
Depending on the nature of activity, horses may also demonstrate visible movement of the neck and back relative to the dorsal plane. **Dorsiflexion** of the spine denotes axial flexion (or **contraction**) along the dorsal surface (or top) of the horse's neck or back (fig. 14.12 A). Animals that visibly "hollow" their backs may be expressing disproportionate axial dorsiflexion (**VL 14h**). Engagement of the opposing musculature evokes flexion (contraction) along the underside of the horse's neck and belly, an action known as axial **ventroflexion** (fig. 14.12 B). Horses that "become round" during work may be demonstrating an appropriate amount of ventroflexion with respect to their medial anatomy. Dorsiflexion and ventroflexion are analogous to the terms **lordosis** and **kyphosis** (respectively), the latter of which denotes *static deviation* or abnormal conformation of the horse's back relative to the dorsal plane; they do not refer to movement. Lordosis is the formal term for "sway back" whereas kyphosis describes a "roached-back" condition.

14.12 Dorsiflexion and Ventroflexion of the Spine



A. During **dorsiflexion** of the spine, the horse's back becomes "hollowed" and the belly protrudes.



B. During **ventroflexion** of the spine, the horse's back becomes "roached" and the belly is drawn up (or "hunched").

VL 14h
Scan/Click to view video.
www.getsound.com/tutorials/14h

Collection is a term often used to describe the horse's movement, but actually refers to a front-to-hind shift in weight as opposed to a visible action. That said, this activity can usually be perceived by the trained eye. By carrying more weight on the hind limbs, the horse effectively compresses its axial anatomy, equipping it with enough potential energy to augment propulsion of the hind limbs. As the horse gathers power through this veritable form of "spring-loading," the observer may notice the body shortening and ventroflexing in a uniform manner. An increase in the duration of stance (i.e. the time that the feet are in contact with the ground surface) and a decrease in forward speed and stride length will also be apparent. The phrase "heavy on the forehand" describes the opposite action, during which the horse plainly avoids engagement of the hindquarters by "diving onto" and/or "pulling along with" the forelimbs. In this case, the observer may perceive an obvious back-to-front shift in weight that is conveyed through excessive depression of the head and neck during fore stance (**VL 14i**). Indubitably, many horses overload and overuse the front end in response to pathology within the **posterior** (back) half of the body.

VL 14i

Scan/Click to view video.

www.getsound.com/tutorials/14i

15

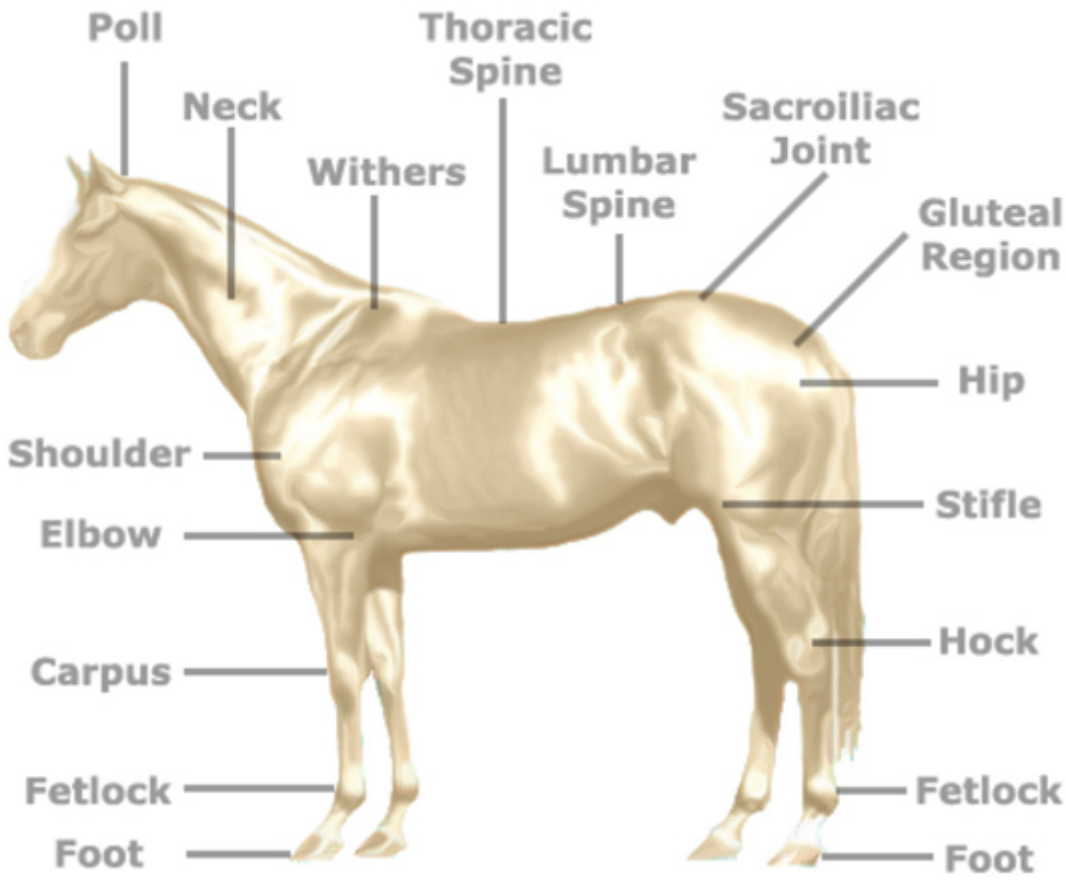
The Relationship Between Anatomy and Expression of Gait

The location and function of various anatomical structures are revealed through the horse's way of moving. Establishing a clear understanding of the reciprocity between the inner workings and the display of movement is vital to accurately decoding what we see as observers.

Influence of Regional Anatomy on Gait Expression

To start, let's review some very basic equine anatomy (fig. 15.1).

15.1 Basic Anatomy of the Horse



Next, let's mentally separate the horse's limbs into three anatomic regions (fig. 15.2).

15.2 Regional Anatomy of the Equine Limbs

A. The lower limb: Everything below the level of the fetlock joint constitutes the horse's lower (or distal) limb.

B. The mid limb: The mid limb comprises the anatomic structures located between the fetlock and carpus in the front limb and the fetlock and tarsus in the back limb.

C. The upper limb: Everything above the carpus and tarsus would be considered part of the horse's upper (or **proximal**) front and hind limb, respectively.

As discussed in chapter 9, there is a strong correlation between the nature of a horse's lameness and the approximate location of its source (see fig. 9.1, p. 43). In general, pathology affecting the horse's lower limb most often generates lameness of weight-bearing nature. Non weight-bearing lameness, on the other hand, is usually exhibited by horses with problems up high in the limb. Accordingly, disease of the horse's mid limb customarily results in combination deficits.

Influence of Anatomic Function on Gait Expression

As effective observers it is not only important that we know the location of key anatomical structures but also their design and function. This knowledge will help us predict the nature of any related gait deficit(s) displayed by the lame horse. In like manner, accurately discerning the nature of a horse's lameness provides us with an appreciation for the physical qualities of its cause.

> *The ease with which we visually discern the nature of a horse's lameness depends on the physical role of the structure(s) affected and the degree to which they are affected.*

The horse's musculoskeletal anatomy has two primary functions:

1. To provide skeletal support during load bearing (carry the horse's weight).

2. To permit and execute skeletal movements during locomotion (move the horse's body and limbs).

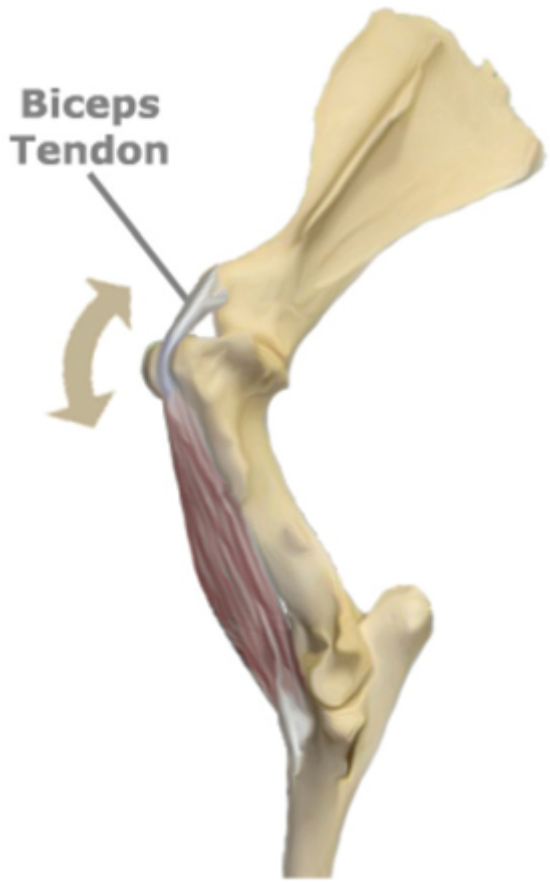
Each aspect of the horse's physical structure will satisfy these roles to varying degrees, and their relative contributions will dictate the nature of any correlating gait characteristics. It is extremely helpful to the observer, therefore, to think of the horse's anatomy with respect to the following points:

- *To what degree does the structure bear weight during the stance phase of the stride?* Structures that bear the most weight are generally situated within the central aspect of the limb, along or adjacent to its center of gravity. By contrast, anatomy residing along the outer aspect (such as the extreme front or back) of the limb tends to be less involved in load support.
- *To what degree does the structure move or change shape during the flight phase of the stride?* Structures that are distorted during limb protraction typically include joints, tendons, muscles, and ligaments. Most other structures (such as long bones) don't bend or deform appreciably.

Now, let's think about these qualities with respect to the nature of a horse's lameness. If a structure experiences weight-bearing load when the horse's limb is on the ground, then pathology associated with the structure will produce a weight-bearing gait deficit. The foot, for instance, will undoubtedly feel the horse's weight during the stance phase of the stride (i.e. when it is in contact with the ground surface). Yet it does not appreciably change shape during the flight phase of the stride. Accordingly, foot pain almost always results in weight-bearing lameness.

If a structure moves or distorts significantly as the horse advances the limb forward, then any associated pathology will accordingly generate non weight-bearing deficits. The bicipital tendon, for example, articulates with the underlying bicipital bursa as it slides over the front of the horse's shoulder during limb protraction (fig. 15.3). It is not directly involved in supporting the horse's weight, however. Pain associated with this area, known as **bicipital tenobursitis,** appropriately manifests as non weight-bearing lameness.

15.3 Non Weight-Bearing Operation of the Horse's Bicipital Tendon

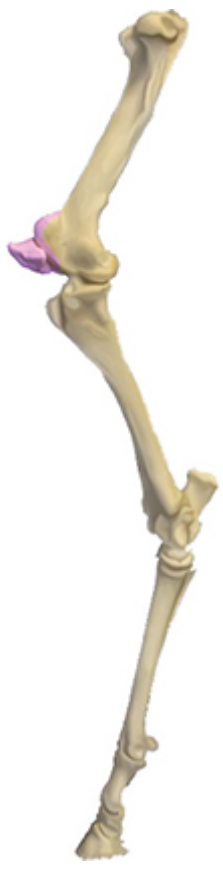


The horse's biceps brachii tendon travels a considerable distance along the surface of the greater tubercle (on the front of the humerus) as the horse protracts the front leg. This tendon does not encounter appreciable weight-bearing load during the stance phase of the stride, however. Accordingly, bicipital tendon injury produces non weight-bearing forelimb lameness.

Some structures provide support to the limb during the stance phase of the stride but also contort during limb protraction. Pathology associated with these structures inevitably generates combination lameness, which comprises both weight-bearing and non weight-bearing components. Depending on the relative contributions to load support and movement, corresponding lameness will display proportionate weight-bearing and non weight-bearing characteristics, respectively. Figure 15.4 illustrates the affiliation between the nature of the lameness manifesting as a consequence of arthritis associated with three joints of the equine hind limb.

15.4 Sample Correlation Between the Nature of Lameness and Joint Anatomy

Femoropatellar Joint

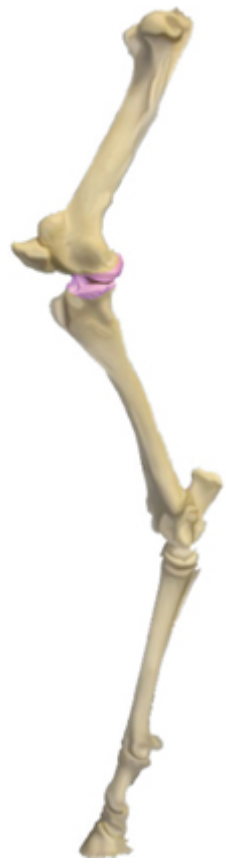


Joint Characteristics: High-motion, nominally weight-bearing.

Joint Description: This joint, between the femur (thigh bone) and "knee cap" in the hind limb (stifle area) is highly articulating (i.e. very high-motion). It is not, however, directly involved in supporting the horse's weight. The joint resides along the front of the stifle and primarily comes into play during the non weight-bearing phase of the stride.

Predicted Nature of Associated Lameness: Consequently lameness associated with this joint tends to be *non weight-bearing* in nature.

Femorotibial Joint

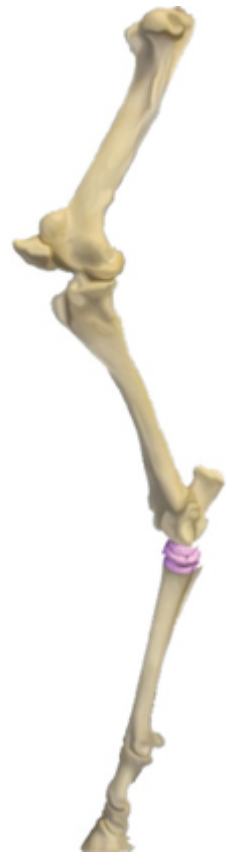


Joint Characteristics: High-motion, decidedly weight-bearing.

Joint Description: This joint, consisting of medial (inside) and lateral (outside) pouches, forms the articulation between the femur (thigh bone) and tibia (leg bone). It is also classified as high-motion. Unlike the femoropatellar joint, however, the femorotibial joint is also directly involved in supporting the horse's weight (i.e. is highly load-bearing).

Predicted Nature of Associated Lameness: Accordingly, problems associated with this joint manifest as a *combination* lameness.

Distal Hock Joints



Joint Characteristics: Low-motion, decidedly weight-bearing.

Joint Description: Of the four joints that make up the horse's hock, the lower two joints are relatively flat and separated by cuboidal bones. These joints, known as the distal intertarsal and tarsometatarsal joints, exhibit minimal movement during work and are therefore classified as very low-motion. They do, however, provide **columnar** support to the horse's limb and are directly involved in supporting the horse's weight.

Predicted Nature of Associated Lameness: Pain associated with these joints, denoted as "distal tarsitis," typically generates *weight-bearing* lameness.

The affiliation between the functional role of affected anatomy and the nature of the lameness that it generates enables the adept observer to accurately predict the physical duties of the anatomic source without direct knowledge of its designation or location. We know, for example, that combination lameness is produced by one or more structures that move appreciably during limb protraction in addition to bearing a significant amount of weight. For this reason, it may be helpful to construe the horse's anatomy in terms of "proportions or percentages" of how the two functional roles are shared. Anatomic structures that provide a significant amount (e.g. 80%) of load support but move very little (e.g. 20%) during limb protraction will generate a combination lameness that has a dominant weight-bearing component. Analogously, we would expect to observe combination lameness with equal weight-bearing and non weight-bearing components originating from a pathologic structure that bears substantial weight (50%) in addition to moving appreciably (50%).

Using this method, we can appraise the relative functionality of the anatomic source by accurately assessing the nature of the horse's lameness (fig. 15.5). We can also predict the nature of lameness expected to result from affliction of most major structures in the horse based purely on their functional roles.

15.5 The Correlation Between Anatomic Function and the Nature of Associated Lameness

Anatomic Structure	Relative Proportion of Weight-Bearing During Limb Stance (as a %)	Relative Proportion of Distortion During Limb Protraction (as a %)	Expected Nature of Associated Lameness
Foot	100	0	Weight-bearing (WB)
Coffin Joint	80	20	Primarily weight-bearing
Pastern	80	20	Combination; prevalent WB component
Fetlock	50	50	Combination with equal WB and NWB components
Cannon Bone	100	0	Weight-bearing
Carpus	50	50	Combination with equal WB and NWB components
Lower Tarsus	90	10	Combination; prevalent WB component
Upper Tarsus	50	50	Combination with equal WB and NWB components
Radius	100	0	Weight-bearing
Tibia	100	0	Weight-bearing
Elbow	50	50	Combination with equal WB and NWB components
Stifle: Medial Femorotibial Joint	60	40	Combination; slightly prevalent WB component
Stifle: Lateral Femorotibial Joint	40	60	Combination; slightly prevalent NWB component
Stifle: Femoropatellar Joint	0	100	Non weight-bearing (NWB)
Humerus	100	0	Weight-bearing
Femur	100	0	Weight-bearing
Shoulder	50	50	Combination with equal WB and NWB components
Hip	50	50	Combination with equal WB and NWB components
Supracarpal Bursa	0	100	Non weight-bearing
Bicipital Bursa	0	100	Non weight-bearing
Neck	0	100	Non weight-bearing
Distal Patellar Apparatus	0	100	Non weight-bearing

Extensor Apparatus	10	90	Combination; prevalent NWB component
Flexor Apparatus	70	30	Combination; slightly prevalent WB component
Suspensory Apparatus	60	40	Combination; slightly prevalent WB component
Hamstring Musculature	10	90	Combination; prevalent NWB component

It is important to note that as the severity of combination lameness increases, the depiction of both its weight-bearing and non weight-bearing constituents will also increase. As a potential consequence, features of the horse's gait that were previously indistinct or imperceptible may become more pronounced, thereby yielding an altered impression with respect to its overall appearance. The observer may then be at risk of misjudging the true nature of the horse's lameness. For example, the non weight-bearing component typically associated with distal tarsitis (lower hock pain) is often considered to be insignificant because the combination lameness associated with this affliction is predominantly weight-bearing in nature. As observers, we're accordingly not accustomed to seeing a conspicuous abnormality associated with the "push-off" aspect of the horse's stride in this instance. Yet evidence of a non weight-bearing deficit may emerge as the problem becomes more advanced and corresponding pain intensifies (**VL 15a**). The visual manifestation of disparate gait characteristics may thus persuade the observer to suspect a source other than hock pain.

VL 15a

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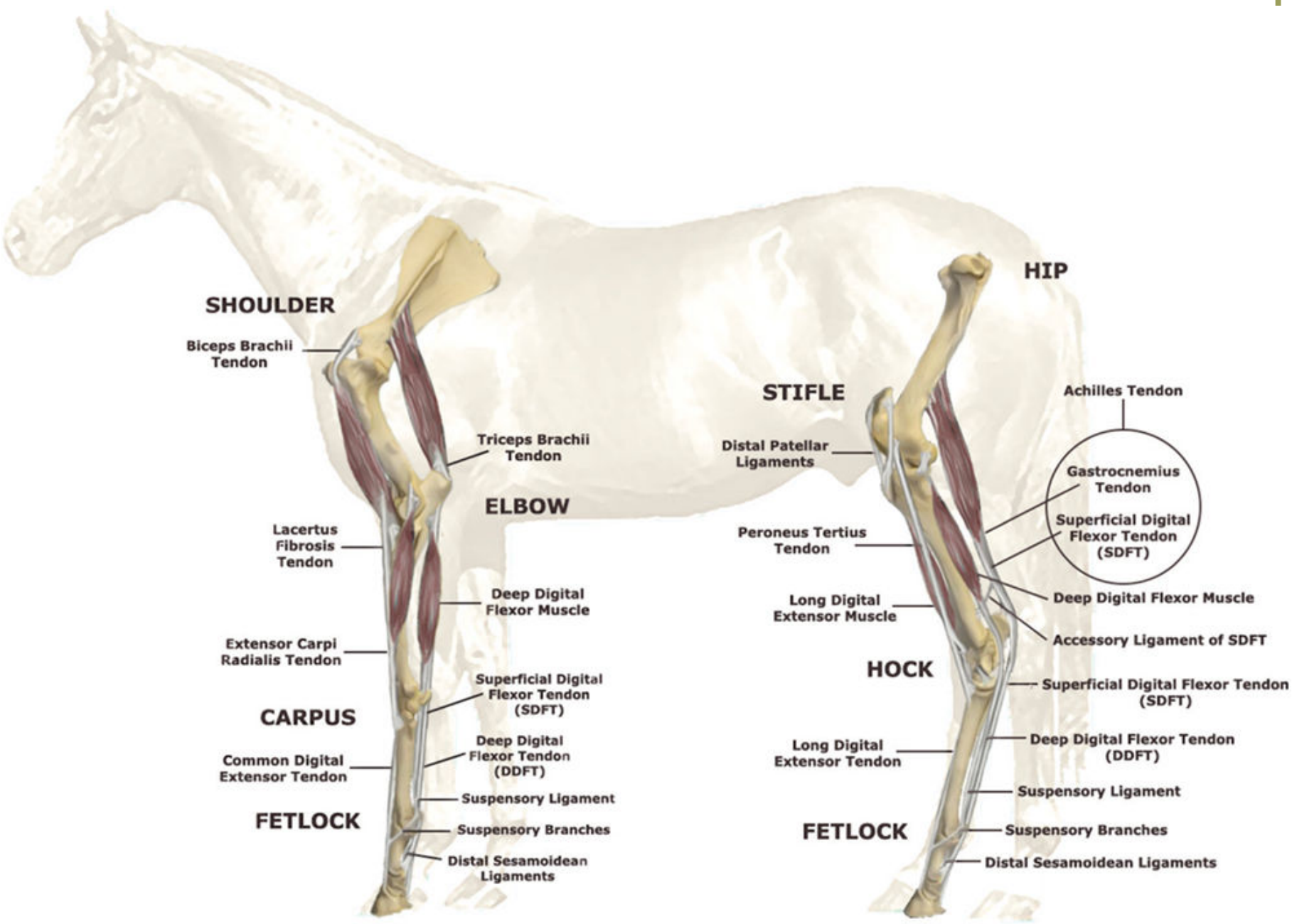
16
The Stay-Apparatus

The *stay-apparatus* (also known as the *reciprocal apparatus*) comprises a number of muscles, tendons, and ligaments that work collectively to stabilize the joints of the horse's limb(s) while in the weight-bearing position (fig. 16.1). It can be viewed as a mechanical pulley-lever system that functions to maintain limb extension at the expense of minimal muscular activity. This mechanism not only permits the horse to sleep while standing, but also serves to improve the action and efficiency of the limbs during movement. Effort of the intrinsic limb muscles is reduced during engagement of the stay-apparatus, which allows the horse to move proficiently and with normal stride characteristics (timing, length, and height). Since tendons and ligaments do not "tire" with exercise, their employment enhances limb stamina as well as action.

16.1 The Stay-Apparatus of the Horse



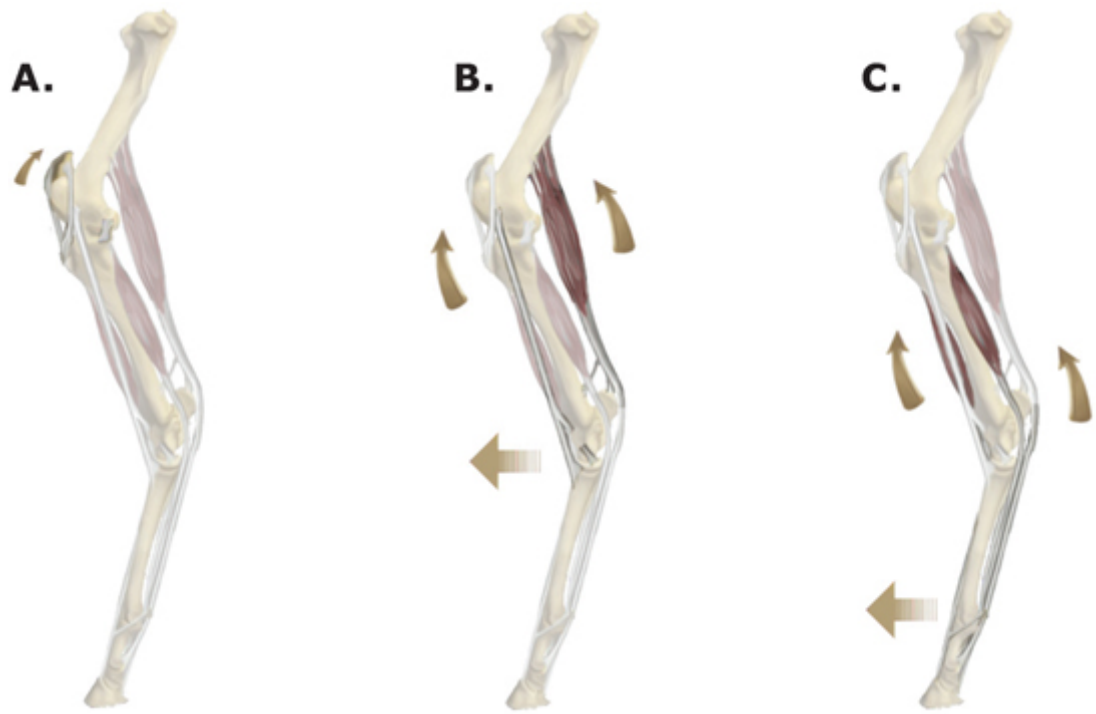
A. The stay-apparatus of the forelimb.
B. The stay-apparatus of the hind limb.

Inflammation, interference, or breakdown of any of the structures that constitute the stay-mechanism can deleteriously affect the horse's performance. Ensuing lameness usually renders distinctive gait abnormalities. Accordingly, the effective observer should acquire basic knowledge regarding the design and function of both the fore and hind components of this implement. The hind stay-apparatus tends to generate more frequent and conspicuous gait abnormalities and is, therefore, discussed foremost.

The Stay-Apparatus of the Hind Limb

The hind stay-apparatus comprises three essential elements, the principle of which relies on structures surrounding the horse's stifle joint (fig. 16.2).

16.2 Elements of the Hind Stay-Apparatus



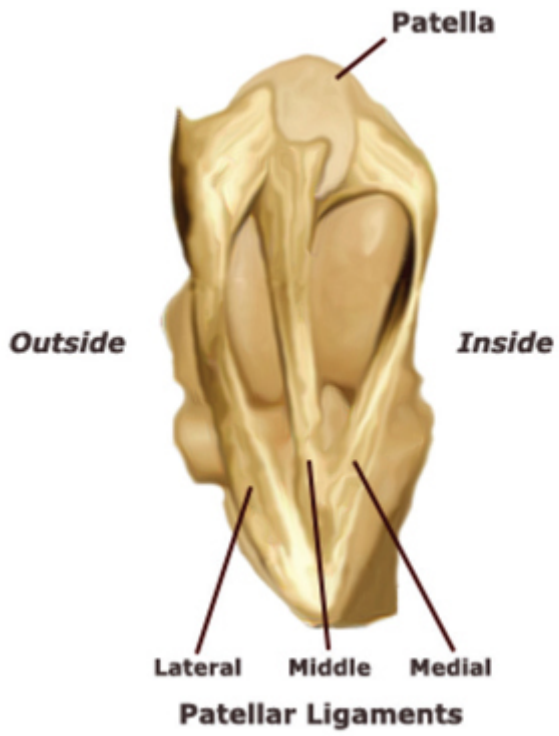
A. The first component involves the stifle-locking mechanism that comprises the patella, the distal patellar ligaments, and the collateral ligaments of the femorotibial joint. This element, when engaged, enables the horse to rest its hind body weight on the "locked" stifle joint.
B. The second element, the *reciprocal mechanism,* ensures that the stifle and hock joints work in unison with one another. It relies on the synchronous action of the *peroneus tertius* tendon along the front of the limb and the *Achilles* tendon along the back of the limb. This coordinated system allows the horse to navigate the pelvic limb in a smooth and coordinated manner. The first and second elements of the horse's stay-apparatus operate conjunctively.
C. The third element involves other muscle, ligament, and tendon structures within the leg. These units conduct the effects of the preceding elements to the horse's lower hind limb.

Although the anatomy and function of the hind limb stay-apparatus is physiologically normal, its inadvertent engagement can sometimes interfere with the horse's ability to flex the hind limb from the extended position during movement. Many equestrians attribute the associated gait deficit to a "loose," "slipping," "locking," or "catching" stifle, since the principle components of the stay-apparatus that precipitate the interference reside in and around this joint.

The Locking Patella

The patella (knee cap), the bottom end of the femur (medial trochlear ridge), and three distal patellar ligaments (ligaments that attach the knee cap to the tibia below the stifle) form the "command post" of the stay-apparatus in the horse's hind limb (fig. 16.3).

16.3 Pertinent Anatomy of the Horse's Patellar Region

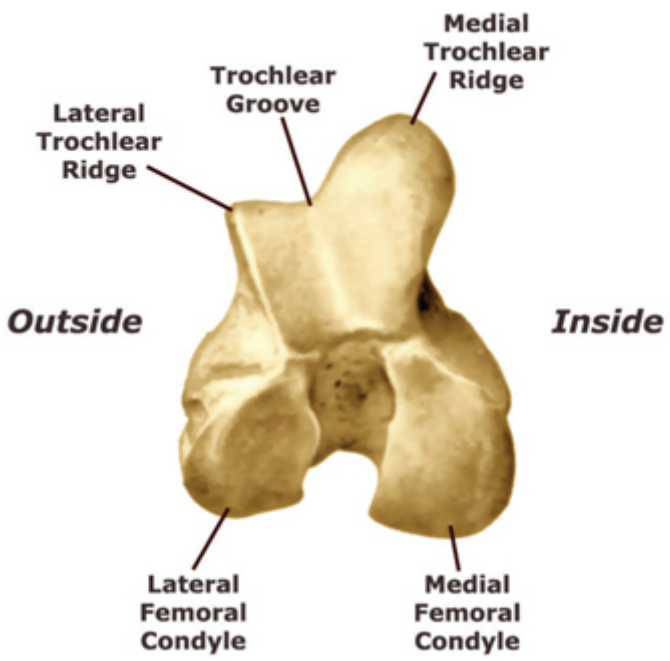


A view of the right stifle from the front.

A trochlear groove (or channel) bordered by two trochlear ridges along the front side of the horse's lower femur provides a tract through which the patella can slide during protraction (or forward movement) of the hind limb. The patella is stabilized within the groove via the action of applied tension along either end. The *quadriceps* and *biceps femoris* musculature attach to the patella along its top side. These muscles function to extend the stifle joint and retract (pull) the patella upward. Three ligaments attach to the patella along its bottom side: one along the inside (the medial patellar ligament), one along the outside (the lateral patellar ligament), and one in between (the middle patellar ligament). The ligaments function to stabilize the patella's movement within the trochlear groove as well as enable fixed extension of the stifle joint.

The horse's trochlear ridges are dissimilar in appearance: the inside (or medial) trochlear ridge is much larger along its upper half (proximal aspect) as compared to its lateral counterpart (fig. 16.4). The inherently prominent medial trochlear ridge of the horse's femur forms a recess or "nook" along its junction with main body of the femur. The horse fixates or locks the stifle in extension by retracting the patella until the space between the inside and middle patellar ligaments slides into this nook. Once the patella is locked in place, the accessory components of the stay-apparatus function to secure the rest of the limb in the extended position.

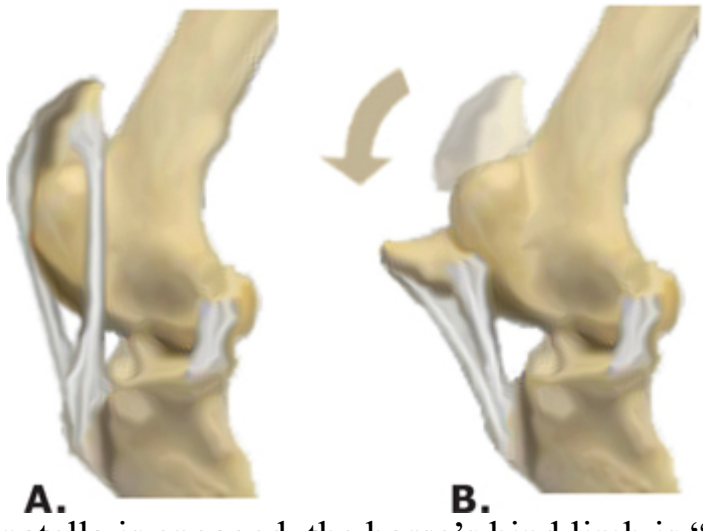
16.4 Pertinent Anatomy of the Horse's Lower Femur



A view of the right femur from the bottom.

The horse must unfix the patella from its location within the recess at the top of the medial trochlear ridge in order to liberate the stay-apparatus and flex the hind limb from the extended position. Unhitching of the apparatus may occur too late or not at all, depending on the situation. Figure 16.5 illustrates the dissimilar locations of the engaged (a) and disengaged (b) patella.

16.5 Engaged Versus Disengaged Patella



A. The *locked* patella: When the patella is engaged, the horse's hind limb is "locked" in the extended position.
B. The *unlocked* patella: The patella must be released from the engaged position to permit flexion of the hind limb.

Delayed patellar release or *proximal patellar hesitation* are terms frequently used to denote late disengagement of the hind stay-apparatus. This causes deferred flexion/protraction of the affected limb, which then doesn't have enough time to achieve full-stride length. The result is a shortened forward (or **cranial**) component to the stride and affiliated non weight-bearing lameness. In most cases, the stride also assumes a hypometric appearance, as the horse will often drag the affected toe(s) in the footing. Associated gait deficits may be understated or very obvious to the observer, depending on the relative degree of interference (**VL 16a**). The more delayed the patellar release, the shorter the stride, and more obvious the associated non weight-bearing gait deficit.

VL 16a
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In most cases of delayed patellar release, a *quiver* or *wiggle* associated with the point of the hock (*tuber calcaneus*) can be visibly discerned at the moment that the horse initiates flexion of the affected hind limb from the extended position (fig. 16.6 and **VL 16b**). This motion represents sudden and involuntary disengagement of the patella from its position atop the medial trochlear ridge of the femur, of which the effects are transmitted directly to the hock via the reciprocal apparatus (most notably through the *peroneus tertius* tendon). Manifestation of abrupt patellar release is easier to see at the level of the hock due to the inherent lack of muscles covering the area.

16.6 Visible Detection of Patellar Release

In cases of delayed patellar release, the point of the hock can often be seen *vibrating* or *wobbling* at the instant the horse picks up the affected hind limb to move it forward.

VL 16b
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In mild cases of patellar interference, only a slight hock wobble can be perceived at the walk and trot. During the canter, the off-lead (trailing) limb may be seen "breaking loose" as this limb leaves the ground surface to advance forward. A mild patellar hesitation may or may not significantly interfere with the horse's ability to perform its job.

As the severity of patellar interference increases, the abrupt shimmy visible at the point of the hock may be accompanied by a jerking motion of the limb as the foot leaves the ground. The latter often looks as though the horse is freeing the foot from thick mud, during which it suddenly and abruptly pops loose (**VL 16c**). In this case, the flight phase of the stride may assume a hypermetric appearance as the horse overexerts the extensor muscles in an attempt to liberate the foot from the "imaginary muck." Due to its outward appearance, this action is sometimes confused with a condition known as *stringhalt* (see chapter 27, p. 195). The deeper the muck, the more animated the limb's observed reaction upon flexion from the extended position. To the observer, the dramatic limb activity implies that the degree of patellar interference may be noteworthy.

VL 16c
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A horse will occasionally "buckle" or stumble behind if the patella is suddenly and unpredictably liberated, following a brief stint of detainment. Riders often feel as though the horse's hind end is "falling through a trap door" during these episodes, which can occur with varying frequency but most often in tight corners and during downward transitions (canter-to-trot and trot-to-walk). In most cases horses recover very quickly and are moving relatively normally within a few strides after the incident (see **VL 12a**, p. 52). Infrequently, this action may be accompanied by an audible "thud," which is generated by friction between the patella and medial trochlear ridge of the femur as the former pops free. This gait abnormality is pathognomonic for a condition known as **intermittent upward patellar fixation (IUPF).**

In exceptionally severe cases, the horse may not be able to disengage the apparatus at all, inciting a more dramatic form of upward patellar fixation. This is also very easy for the visual observer to identify, as the hind limb is locked in straight posture and the horse is unable to flex it during ambulation (**VL 16d**).

VL 16d
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The behavior of the stay-apparatus depends on a number of extrinsic factors such as limb position, level of fitness, and gait, in addition to circumstances brought about by outside events or other forms of pathology (fig. 16.7). Appropriately, delayed patellar release and intermittent upward patellar fixation are commonly implicated as *secondary* problems. Since operation of the stay-apparatus is influenced by fitness level as well as the degree of hind limb extension, primary causes of related interference should always be ruled out to ensure accurate identification of this affliction.

16.7 Extrinsic Influences on the Behavior of the Hind Stay-Apparatus

Extrinsic Factor	Effect on Function of the Stay-Apparatus	Examples
Level of Fitness	Lack of tone associated with the muscles, tendons, and ligaments intimately affiliated with the stay-apparatus can make inadvertent engagement more likely to occur and subsequent disengagement more difficult to achieve.	A "loose stifle" is more likely to develop in unfit animals and is often secondary to issues that result in chronic loss of use, muscle **atrophy** or neuropathy of the affected limb(s).
Conformation	Horses that feature a downhill topline, insufficient (excessively straight) hind-limb angle and/or **negative plantar angulation** are predisposed to accidental engagement of the hind stay-apparatus.	Young animals that grow more rapidly behind (and develop a downhill topline) may transiently exhibit "weak" or "loose" stifles.
Degree of Limb Extension	The patella is more likely to lock into position atop the medial trochlear ridge of the femur during periods of increased hind-limb extension and may be more difficult to disengage amid these moments.	This effect is clearly demonstrated by a horse that is walking downhill, an activity that requires increased hind-limb extension in order to support the horse's **anteriorly** displaced weight (**VL 16f**).
Gait	Movements that demand increased and/or prolonged extension of the hind limb will predispose the horse to delayed patellar release, especially if the rider is asking for concurrent collection of the horse.	The effect of limb position accounts for why stifle interference is more frequently encountered at the canter as opposed to the trot. Hindrance increases commensurately with the degree of collection.
Trauma	External insult or internal strain of the muscles and/or ligaments associated with the patellar mechanism can induce related biomechanical interference.	Horses that hyperextend the hind limb while sliding to a stop may stretch the distal patellar ligaments in the process, thereby making accidental engagement/fixation of the patella more likely to occur.
Other Pathology(ies)	Certain forms of inflammatory-mediated musculoskeletal disease can also provoke hindrance of the stay-apparatus, particularly if they lead to increased or prolonged extension of the hind limb.	**A.** A horse with severe weight-bearing lameness of both forelimbs may experience increased patellar interference while overextending the hind limbs in an attempt to underload the front end. **B.** A horse experiencing severe hock pain may choose to avoid flexion of the affected hind limb so as not to accentuate discomfort. This action may result in increased/prolonged extension of the respective limb, thereby precipitating secondary stifle interference.

It is also important to note that biomechanical interference of the horse's hind limb stay-apparatus is not dictated by pain or inflammation and, therefore, may not respond to physical limb manipulation (flexion testing), local anesthesia, nor anti-inflammatory therapy. Moreover, visible abnormalities will rarely be apparent upon diagnostic imaging of the affected limb, thereby requiring proper recognition of characteristic gait deficits for accurate diagnosis. Common presenting signs for biomechanical interference of the hind stay-apparatus are highlighted in figure 16.8.

16.8 Classic Symptoms Associated with Mild Interference of the Hind Stay-Apparatus

Clinical Expression	Comments
Non weight-bearing hind-limb lameness.	• This issue is biomechanical in origin.

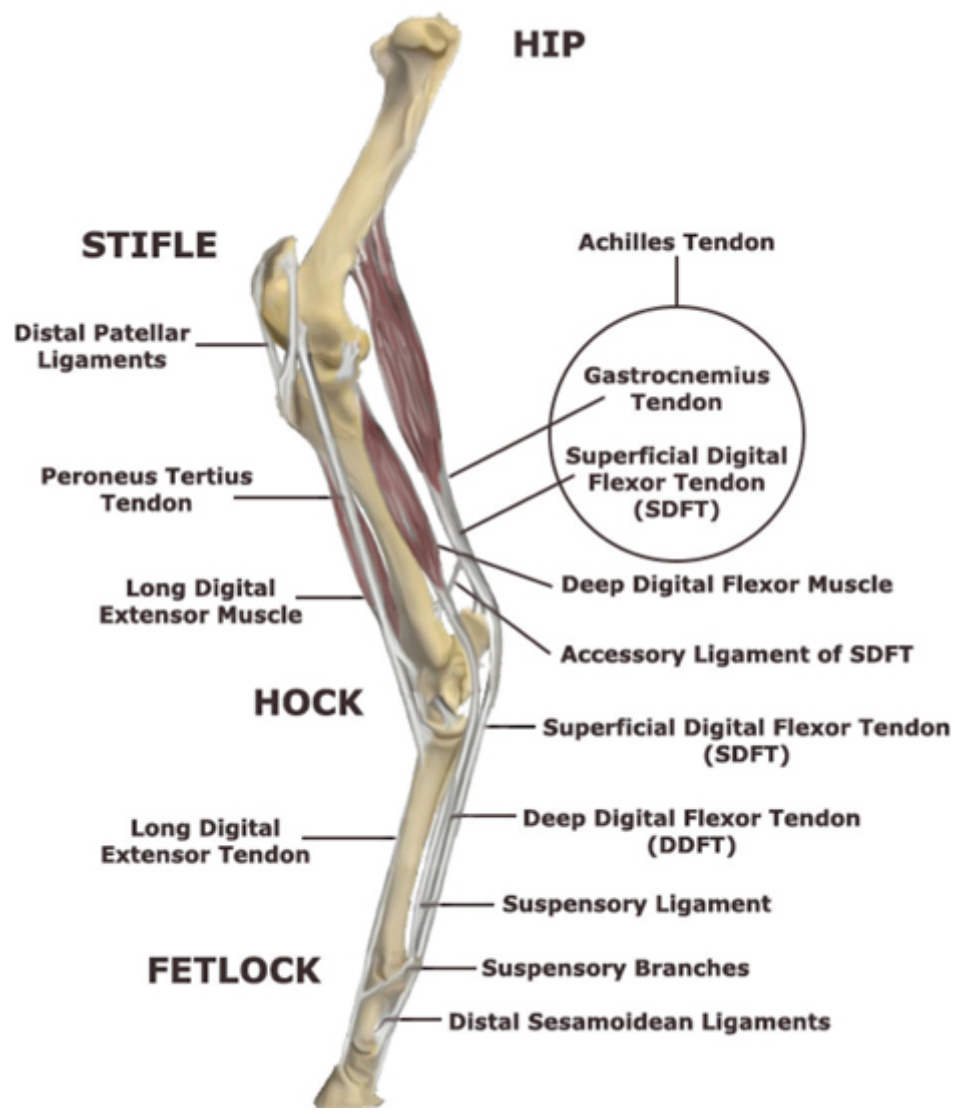
	• It can be distinguished from other pain-mediated issues that generate weight-bearing deficits (such as hock pain). • The horse may drag the toe(s) of the affected hind limb(s) during exercise. • Excessive toe-wear may be visible on the shoe and/or foot. • The affected limb(s) will display a low-arc pattern during the flight phase of the stride. • This is known as a hypometric gait, in which the horse may drag the hind toes. • The cranial (or flight) phase of the stride will usually be delayed. • It appears as though the horse is picking the foot up too late. • The cranial (or flight) phase of the stride will usually be shortened. • The limb doesn't have enough time to complete a full stride when the breakover is delayed. • During lateral work (such as a half-pass) the trailing limb may appear to "lag" behind the horse. • This looks as though the horse is dragging along a weight (e.g. brick) with the affected limb.
Resistance in the canter.	• Resistance may be accentuated with the affected limb on the inside of a circle. • The leading limb encounters a greater degree of extension (up and under the horse) and increased weight-bearing load when along the inside of a circle. • Resistance may be most noticeable during the upward transition between the trot and canter. • During a trot-to-canter transition, the horse is forced to extend the pelvic limb for a prolonged period of time. • Many horses will display poor behavior. • Head-tossing, tail-swishing, rearing, kicking out, bucking, or stopping is often encountered during canter departs or while in the canter. This may be due to the horse's anticipation of impending patellar interference. • The horse might prefer to trot rather than canter. • The canter is physiologically easier than the trot, because less active flexion of the limbs is required. However, the stay-apparatus is challenged to a great degree in the canter.
Problems with canter leads.	• Picking up the wrong canter lead, dropping out of the **correct lead** and swapping leads behind. • Increased challenge to the reciprocal apparatus of the lead limb at the canter will often result in inappropriate lead changes, especially when the affected limb is on the inside of a circle.
The canter is very "rough" or bouncy.	• This is most evident during downward transitions. • Consistent intereference of the stay-apparatus results in a disjointed way of going, as the horse can't match tempo between the forelimbs and hind limbs.
Swelling, heat, or pain may be associated with one or both stifle (femoropatellar) joints.	• This is suggestive of concurrent or secondary inflammation in the joint between the patella and femur. • Femoropatellar synovitis/arthritis may occur secondary to chronic moderate patellar interference.
Resistance and/or difficulty when backing up or walking down hills.	• When confronted with these scenarios, the horse is forced to extend the pelvic limb for a prolonged period of time. • Many horses will volunteer to traverse the decline rather than walk straight downhill. • The horse may avoid full extension of the hind limb in an attempt to avert engagement of the stay-apparatus. • Rather than fully extend the hind limb(s), the horse may "crouch" while walking. • The horse may be unable to flex the hind limb from the extended position within a reasonable amount of time. • Rather than flex the pelvic limb(s) normally, horses will often swing their limbs outwardly to clear the ground surface.
Lameness is most evident as the horse first leaves the stall.	• Many horses maintain upward fixation of the patella while standing in the stall, as this is natural and requires little effort. • Since the stay-apparatus has grown "accustomed" to being engaged while in the stall, it tends to re-engage more easily until the horse warms up. • The horse might prefer to trot rather than canter.
Lameness persists or worsens following periods of rest.	• Lack of regular exercise can result in a loss of tone associated with the muscles, ligaments, and tendons that comprise the stay-apparatus. • Increased laxity makes involuntary interference of the stay components more likely to occur.
Lameness persists in the face of anti-	

inflammatory therapy.	• This issue is biomechanical in origin. • Systemic (e.g. phenylbutazone, banamine) and intraarticular (e.g. corticosteroids) therapy are relatively ineffective at alleviating symptoms of patellar interference.

Compromise of the Hind Stay-Apparatus

As previously stated, the horse's hind stay-apparatus relies on a number of structures that work collectively to maintain simultaneous extension of the stifle and hock joints (fig. 16.9). Compromise of one or more of these structures can disable the mechanism, making fixed extension of the hind limb difficult and, in some cases, impossible. In order for the horse to move normally and maintain stamina, the stay mechanism should be engaged at some point during the stance phase of the stride to support the horse's weight. It must also appropriately disengage to enable natural flexion of the hind limb during protraction (the flight phase of the stride).

16.9 The Hind Component of the Stay-Apparatus



Collapse of the stay-apparatus is fairly easy to recognize from a visual standpoint (**VL 16e**). With careful inspection, we can often ascertain which component(s) of the apparatus have failed.

VL 16e
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www.getsound.com/tutorials/16e

VL 16f
Scan/Click to view video.
www.getsound.com/tutorials/16f

Engagement of the stay-apparatus depends on the horse's ability to lock the patella over the medial (inside) trochlear ridge of the femur. This, in turn, requires that the patella be intact (in one piece) and that the middle and medial distal patellar ligaments occupy the space along the outside and inside of the medial trochlear ridge, respectively (see fig. 16.3, p. 95). The inability of the patella to maintain this position disables the horse's hind stay-apparatus.

Patellar fracture. In some cases of patellar fracture, tension at the attachments of the middle and medial patellar ligaments causes distraction and separation of bone fragments, each of which moves from its fixed position atop of the medial trochlear ridge of the femur. This allows the horse's femur and tibia to move independently of one another, effectively disabling the stay-apparatus.

Patellar ligament rupture. Rupture of the medial and/or middle distal patellar ligaments removes the fixed attachment between the locked patella and tibia, thereby allowing the latter to move independently of the femur. Medial patellar desmotomy, a surgical procedure that mimics this injury, is sometimes implemented to treat severe cases of upward patellar fixation.

Luxation of the patella. While in the trochlear groove of the femur, the patella can normally be "guided up and onto" the top of the medial trochlear ridge of the femur to enable fixed extension of the limb. This action may not be attainable in the event that the patella has been dislocated (i.e. **luxated**) and no longer resides within the trochlear groove. The patella can luxate to the inside or outside of the trochlear groove, the latter being the more common scenario.

Compromise of one or more accessory structures associated with the stay-apparatus can also produce characteristic gait deficits. In the majority of cases, lameness results from biomechanical aberration rather than from discomfort.

Rupture of the peroneus tertius tendon. The *peroneus tertius* tendon is one of the primary structures along the front of the limb that physically links the stifle and hock joints. As such, it is a critical component of the horse's stay-apparatus. Rupture of this tendon occurs secondary to hyper- or overextension of the hock joint(s). As a consequence of this injury, the hock is no longer coupled to the stifle upon flexion of the limb. This produces a gait deficit that is classic in appearance: the hock joint does not flex in conjunction with the stifle as the hind limb moves forward and the distal aspect of the limb appears to hang limp (**VL 16g**). Most horses with *peroneus tertius* tendon rupture are willing to bear full weight on the limb since pain is not a typical characteristic of this affliction. Associated lameness, therefore, is predominantly non weight-bearing in nature.

VL 16g
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The peroneus tertius tendon originates in common with the long digital extensor (LDE) tendon from the extensor fossa of the femur (in the hind limb) and inserts on the dorsal surfaces of the proximal third metatarsal, calcaneal, and fourth tarsal bones. The tendon is a critical part of the reciprocal apparatus of the pelvic (hind) limb and is responsible for generating simultaneous stifle and hock joint flexion in the normal horse.

Rupture of the Achilles tendon. The Achilles tendon incorporates the tendons of the gastrocnemius and superficial digital flexor (SDF) muscles along the back of the horse's limb and is a major part of the secondary

element of the hind stay-apparatus. Compromise to the Achilles tendon or one of its constituents may occur during an episode of extreme exertion whilst the horse attempts to extend the hock. The associated gait deficits are definitive: during stance, the hock joint will assume an excessive (flexed) angle and appear to “drop” even though the rest of the limb maintains extension.

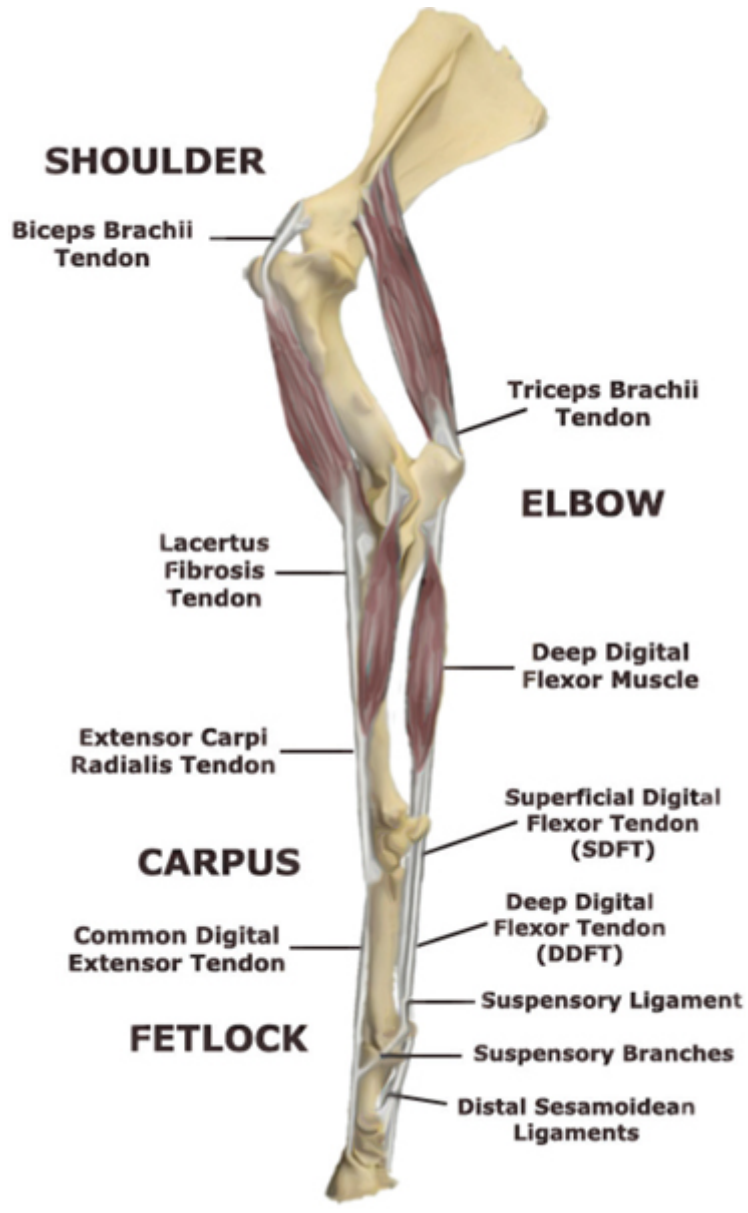
In the event of *gastrocnemius muscle rupture* alone (which is more common than SDF tendon rupture), the hock will drop until the SDF tendon is completely engaged. The horse can bear weight although will appear to *squat* under load bearing, especially if both limbs are affected.

The affected limb(s) is unable to support any weight in the case of complete Achilles tendon rupture, as the hock drops to or close to the ground surface. In all cases of Achilles tendon disruption, the horse is unable to fully straighten the affected hind limb(s) and, therefore, displays pronounced lameness with combination deficits.

The Stay-Apparatus of the Forelimb

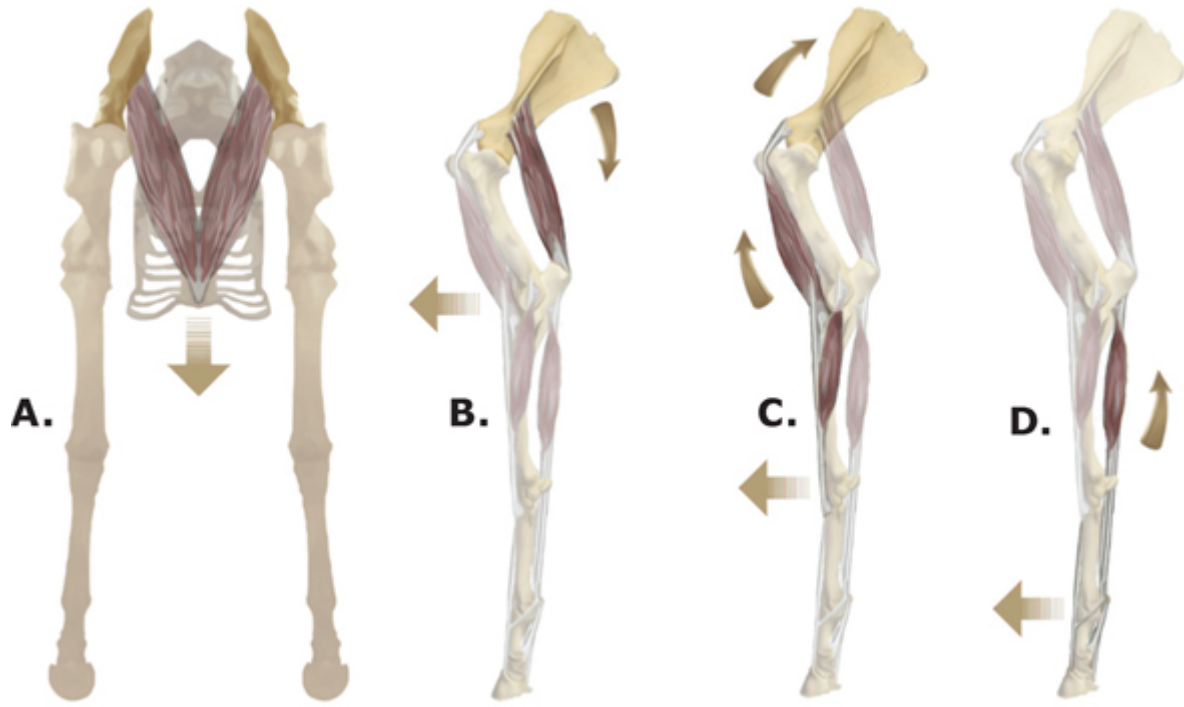
The design and operation of the fore stay-apparatus parallels that of the hind end, although the veritable "latching mechanism" is absent (fig. 16.10). Rather, this component relies on the action of muscles, tendons, and ligaments that are activated by the horse's weight. Accordingly, no inadvertent "catch" or biomechanical delay influences the horse's forelimb stride in the same way that it does that of the hind limb.

16.10 The Fore Component of the Stay-Apparatus



The structures that comprise the "command post" of the fore stay apparatus are primarily affiliated with the shoulder joint. These structures are employed as gravity pulls the head, neck, and chest toward the ground during the stance phase of the stride or while the horse is standing still. The downward gravitational force is opposed via application of the *serratus ventralis* musculature, which functions to secure the horse's forelimbs to the axial skeleton (primarily the rib cage). As the horse loads each forelimb, the respective shoulder blade (also known as the *scapula*) is pulled in a downward direction by tension from the *serratus ventralis,* which attaches along its inner surface. The paired muscles (one on either side of the chest) function as a type of "sling" to support the weight of the horse's front end (fig. 16.11 A). These muscles work in conjunction with the *triceps* muscles to engage the primary element of the stay mechanism. Resting tension associated with the triceps prevents flexion of the elbow joint and concurrent collapse of the upper limb (fig. 16.11 B).

16.11 Elements of the Fore Stay-Apparatus



A. Front view: The *serratus ventralis* musculature along either side of the rib cage functions to suspend the horse's axial anatomy (i.e. head, neck, and chest) between the shoulder blades to which they attach. These muscles are subjected to tension during stance (when the horse is bearing weight), resulting in downward movement of the respective shoulder blade(s).
B. Side view: Steady tension of the triceps musculature is required to prevent flexion of the elbow joint whilst the shoulder blade moves downward.
C. As the shoulder blade tips downward, the biceps musculature is stretched. A collagenous tendon within the biceps musculature prevents excessive elongation of this structure, thereby allowing the horse to rest on the forelimb without collapse of the shoulder, elbow, and carpal (knee) joints.
D. The effects of mid-limb extension are transmitted to the lower limb via the engagement of tertiary elements that include the flexor tendons and their accessory (check) ligaments.

Downward movement of the shoulder blade induces flexion of the shoulder joint, an action that is governed by the *biceps* musculature. A collagenous tendon that resides within the biceps muscle body (and extends its entire length) prohibits excessive elongation of this structure, which physically links the shoulder blade and mid forelimb. On the top side, the biceps utilizes a large (bi-lobed) tendon that attaches to the front/top border of the shoulder blade. Below, the muscle implements two tendons: a short tendon that inserts along the front of the upper forearm (radius) and a long tendon (known as the *lacertus fibrosis*) that joins the *extensor carpi radialis* tendon to insert along the front of the cannon bone. This complex comprises the secondary element of the fore stay-apparatus (fig. 16.11 C). Akin to that of the hindquarters, the horse's lower forelimb is stabilized by complementary employment of the extensor, flexor, and suspensory apparatuses (fig. 16.11 D).

The lacertus fibrosis tendon originates in common with the biceps brachii muscle and continues down the limb to blend with the tendon of the extensor carpi radialis muscle and forearm ***fascia,*** *eventually inserting along the proximodorsal (top/front) aspect of the cannon bone. The tendon is a critical part of the reciprocal apparatus of the thoracic (fore) limb and is responsible for maintaining mid-limb extension during stance.*

Compromise of the Fore Stay-Apparatus

Pathology that impedes the employment of one or more components of the fore reciprocal mechanism can impair its operation. The source may be musculoskeletal or neurologic in origin. In both instances, the horse will display an abnormal posture during the stance phase of the stride. A complete disruption of the apparatus may prohibit the horse from bearing any weight with the affected limb(s) (**VL 16h**). Consonant with several conditions affecting the hind component, there are some forelimb maladies associated with the stay-apparatus that generate distinctive gait abnormalities.

VL 16h
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Rupture of the serratus ventralis muscle(s). As previously discussed, the *serratus ventralis* musculature is responsible for suspending the horse's chest between the forelimbs. As such, it can be regarded as a "sling" or "hammock" that holds up the horse's front end. Contraction of both muscles in the standing horse will elevate the chest and extend the neck. Contraction of one muscle (by itself) will tip the trunk and move the neck toward the contralateral side of the horse. As you might expect, lack of serratus ventralis muscle tone/function will produce the opposite effect(s). When both (right and left) muscle bellies are compromised, the chest will sink relative to the forelimbs. Accordingly, the shoulder blades become more prominent and may even assume a position higher than that of the withers. In the case of unilateral affliction, the shoulder blade of the affected side will appear to rise and deviate outwardly relative to the horse's chest. In this instance, the horse's median anatomy tips toward the affected side.

Shoulder "slip." Loss of **collateral** support around the shoulder joint can precipitate a recognizable gait deficit in the affected limb: the shoulder area will bulge outwardly under weight-bearing load. At the same time, the horse's foot will turn inwardly (**VL 16i**). These gait abnormalities may be suggestive of *sweeney,* a condition affecting the muscles that lie along the outside of the shoulder blade. Shoulder slip is most easily observed when viewing the horse from the front. The conspicuity of the gait abnormality tends to be commensurate with the degree of weight-bearing load encountered by the affected limb.

VL 16i
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www.getsound.com/tutorials/16i

Conditions affecting the function of the triceps musculature. Some degree of muscle tone associated with the triceps is necessary to maintain extension of the elbow and engagement of the fore stay-apparatus during stance. Failure of this structure to maintain tension effectively disables the mechanism. The result may manifest as a *dropped elbow.* In this state, the horse will often stand with the elbow and carpus (knee) forward and partially flexed (see **VL 16g**, p. 100). This affliction can result from direct trauma to the muscle although is more commonly associated with *radial nerve paralysis.* Clinical presentation may vary, although difficulty advancing (protracting) and standing on the limb are typical manifestations. Accordingly, profound combination lameness is observed. Affected horses are predisposed to tripping up front. In many cases, the affected limb will appear to be longer than its contralateral counterpart. Other maladies (such as humeral or radial fracture) can produce similar clinical signs and should be systematically ruled out by the veterinarian.

Rupture of the biceps brachii tendon. The biceps musculature is under tension during stance and contracts to extend the shoulder joint and advance the limb forward during ambulation. Lameness associated with most cases of biceps *myositis, tendonitis,* or *tenobursitis* is non weight-bearing in nature: the horse labors during protraction of the affected limb (**VL 16j**). Only in severe cases of biceps compromise or rupture is weight-bearing lameness observed. Absence of tension afforded by the collagenous tendon within the biceps muscle results in hyperflexion of the shoulder joint and failure of the fore stay-apparatus.

VL 16j
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www.getsound.com/tutorials/16j

Pathologic conditions affecting the function of the tertiary components of the stay-apparatus are discussed in chapter 17, p. 106.

17
Anatomic Behavior of the Lower Limb

The horse's lower limb anatomy (what resides below the level of the carpus in the forelimb and the tarsus in the hind limb) has three primary responsibilities that are fulfilled via the engagement of tendons, ligaments, and bones:

1. *To stabilize/suspend the lower-leg bones.* This role is co-managed via engagement of the flexor (tendon), extensor (tendon), and suspensory (ligament) apparatuses. An inability to properly support the horse's lower limb leads to visible abnormalities expressed during the stance (weight-bearing) phase of the stride.

2. *To move the lower leg bones.* Flexor tendons are employed to flex the lower limb during limb advancement, whereas extensor tendons are engaged prior to and throughout the stance phase of the stride. Both flexor and extensor tendons serve as "mechanical extensions" to their corresponding parent muscles located higher up in the limb.

3. *To initiate and manage breakover of the foot during ambulation.* This responsibility is assumed by concurrent activity of the flexor and suspensory apparatuses. Abnormal foot breakover (with respect to both pattern and timing) can produce visible deficits in the horse's gait.

Although tendons and ligaments have a similar physiologic composition, they function in very different ways. A tendon is a band of fibrous connective tissue that binds muscle to bone or another movable structure (such as an eyelid). Tendons provide muscles with the direct means to move tissues to which they are attached and, therefore, are necessary for normal locomotion. A ligament is a band of fibrous connective tissue that binds bone to bone or tendon to bone. Ligaments function to stabilize structures relative to one another, such as two bones on either side of a joint. Although ligaments are not voluntarily employed by the horse to move bones, they do possess viscoelastic properties that allow them to gather, store, and return energy to the structures to which they are attached. This action both facilitates movement and improves musculoskeletal stamina. The horse's flexor, check, and suspensory apparatuses comprise the major components for the lower portion of the front and hind stay mechanisms.

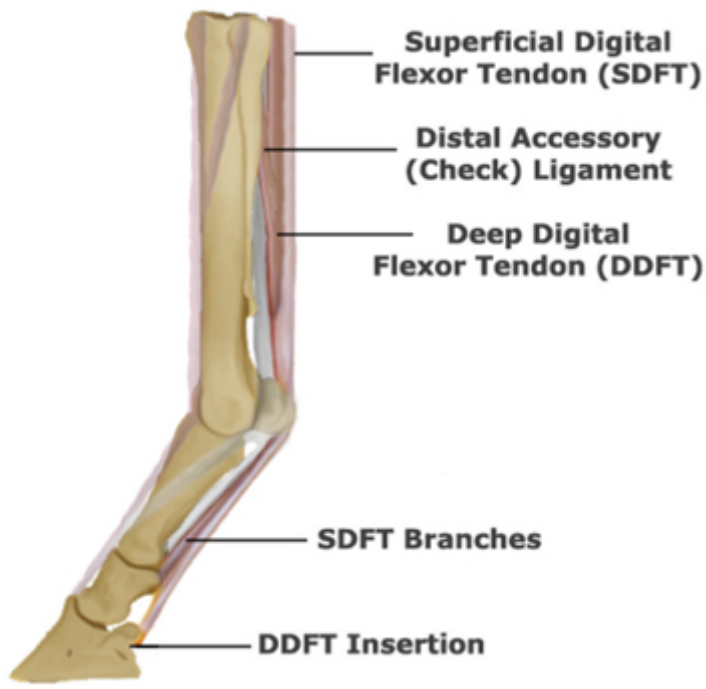
> *Tendon and ligament injuries usually generate combination gait deficits, because these structures are employed during limb stance (for support) in addition to physically deforming during limb protraction. A* ***sprain*** *refers to a tendon injury, whereas a* ***strain*** *refers to a ligament injury.*

Tendons and ligaments are most susceptible to injury along the site of their bony attachment(s), known as the **fibro-osseous junction,** where they are inherently the weakest. Damage to the **periosteum** overlaying the bone is typically the most painful element of a tendon sprain or ligament strain. Other sources of pain (in order of average contribution) include ligament bodies, tendon bodies, fascia (that encompasses muscle bellies), and muscle tissue. As you might imagine, tendon and ligament injuries are most painful when they are under *tension* (as opposed to compression).

The Flexor Apparatus

The foundation of the horse's flexor apparatus comprises the superficial digital flexor (SDF) muscle and tendon, the deep digital flexor (DDF) muscle and tendon, and a variety of accessory structures (such as synovial **sheaths, retinacula,** and **annular ligaments**) that enable and facilitate their employment (fig. 17.1). SDF and DDF tension is regulated by the proximal accessory (or check) and distal accessory (or check) ligaments, respectively, which indirectly affix their corresponding tendon structures to bone. The check ligaments serve to discourage overextension (and, therefore, injury) to the flexor tendons during periods of intense load bearing. As we'll learn in the following section, the check ligaments also contribute to the tertiary components of the fore and rear stay-apparatuses.

17.1 Basic Elements of the Lower Flexor Apparatus



The horse's flexor apparatus performs two primary roles:

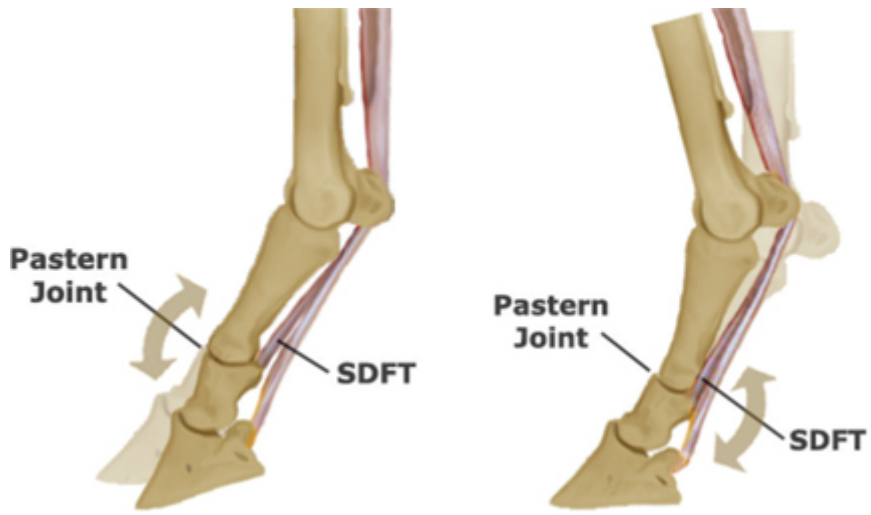
- *To flex the limb during protraction.* This allows the foot to clear the ground surface and enables the horse to advance the limb forward during movement.
- *To prevent overextension of the lower limb during stance.* Without counteractive tension afforded by the flexor apparatus, operation of the extensor tendons would prevail, promoting **hyperextension** of the limb's lower joints.

As we know, tendons are principally intended to move things, so affiliated injuries typically result in painful movement. Tendons are also engaged when the limb is under weight-bearing load, however, and help ligaments to support the horse's lower limb during the stance phase of the stride. Accordingly, tendon pain also comes into play when the limb is bearing weight. Thus a horse with a sore superficial or deep digital flexor tendon will display combination gait deficits.

Compromised tendon integrity or **adhesions** between tendons and surrounding tissues can alter the ability of the parent muscle to perform its function(s) even in the absence of pain. Such forms of tendon pathology can generate biomechanical lameness. If tendons cannot move or slide normally through tissues, for instance, they cannot transmit the effects of the parent muscle to the respective bone(s) to produce the desired movement and/or sufficiently administer opposing action to the extensor tendons.

The superficial digital flexor tendon (SDFT) enforces the action of its parent structure (the superficial digital flexor muscle) by flexing the horse's pastern (or proximal **interphalangeal**) joint. It inserts on the second (or middle) phalanx (P2) and functions to move this bone relative to the first (or proximal) phalanx (P1) residing just above (fig. 17.2 A). Chronic excessive SDFT tension (such as may occur in cases of adhesions, **flexural deformity,** or contraction) can lead to increased pastern angle (which becomes more upright) and reduced fetlock "drop" during stance.

17.2. Basic Effects of the Superficial Digital Flexor Tendon (SDFT)

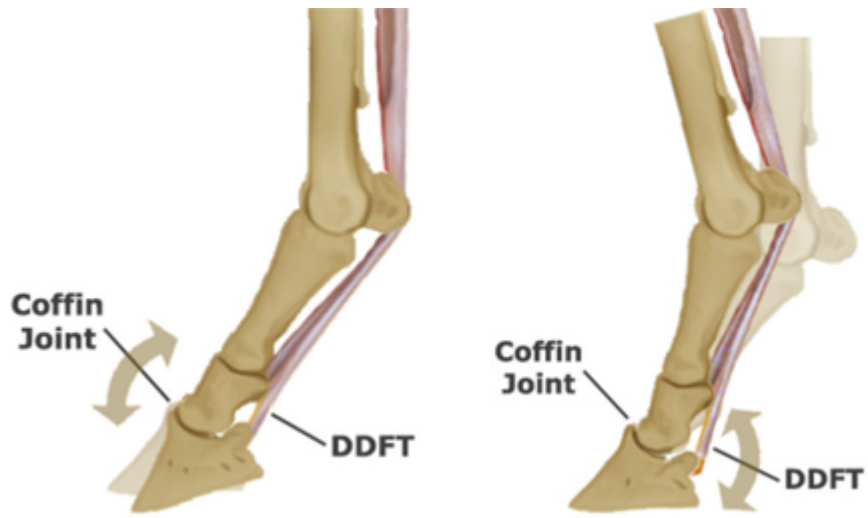


A. The superficial digital flexor tendon functions to flex the horse's pastern joint.
B. The superficial digital flexor tendon also serves to prevent hyperextension of the pastern joint.

The SDFT experiences greatest tension during extension of the pastern joint (fig. 17.2 B). Structural impairment of the SDFT can appropriately result in compromised flexion of the pastern during protraction and lowered (more shallow) pastern angle, hyperextension of the pastern joint, and (in severe cases) **dorsal subluxation** of the middle phalanx during stance.

The deep digital flexor tendon (DDFT) enforces the action of its parent structure (the deep digital flexor muscle) by flexing the horse's coffin (or distal interphalangeal) joint. It inserts along the underside of the third (or distal) phalanx (P3) and functions to move this bone relative to the first and second phalanges residing above (fig 17.3 A). The navicular bone serves as a "fulcrum" for the DDFT as the latter courses around the back of the horse's foot. Synovial fluid within the navicular bursa provides essential lubrication for the physical interaction between the DDFT and navicular bone at this level.

17.3 Basic Effects of the Deep Digital Flexor Tendon (DDFT)

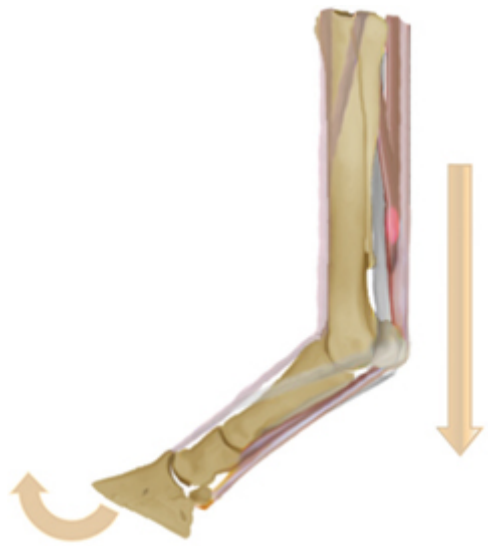


A. The deep digital flexor tendon functions to flex the horse's coffin joint.
B. The deep digital flexor tendon also serves to prevent hyperextension of the coffin joint.

Excessive DDFT tension over the long term (such as may occur in a case of "clubbed" foot) can lead to increased hoof angle (which becomes steeper/more upright) and reduced fetlock "drop" during stance. The tendon experiences greatest tension during extension of the coffin joint (fig. 17.3 B). Structural impairment of the DDFT can appropriately result in compromised flexion of the lower limb during protraction and lowered (more shallow) pastern angle, hyperextension of the coffin joint, and (in severe cases) forward repositioning of the hoof during stance.

Rupture of the DDFT generates a pathognomonic gait abnormality. Lack of flexural tension on P3 afforded by the tendon during the flight phase of the stride causes the foot to flip forward in the air prior to striking the ground surface, often resulting in a heel-first landing. The same absence of tension during stance induces the horse's toe to lift up and off of the ground (fig. 17.4 and **VL 17a**).

17.4 Pathognomonic Appearance of Deep Digital Flexor Tendon (DDFT) Rupture



Visible elevation of the horse's toe off the ground during stance is pathognomonic for rupture of the deep digital flexor tendon (DDFT).

VL 17a

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Altered function of the flexor apparatus has the potential to produce both pain-mediated and biomechanical lameness, depending on the nature of the insult. Since the flexor apparatus is employed during flight and stance, pathology associated with any of its components will generate combination gait abnormalities.

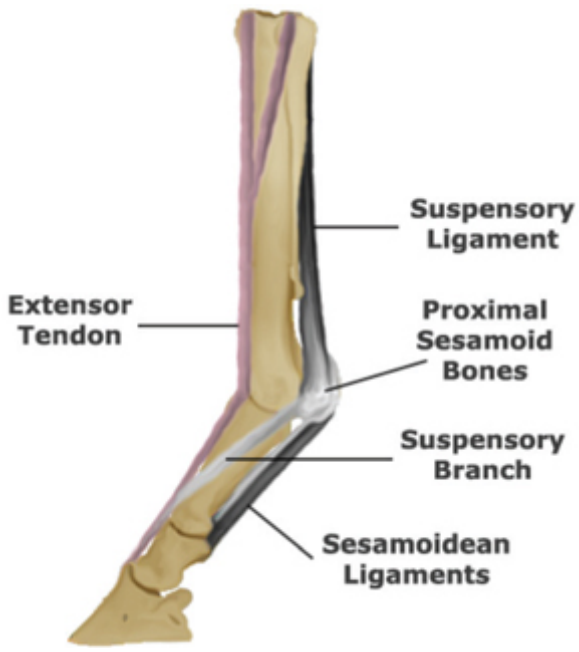
The Check Apparatus

The accessory (or “check”) ligaments function to indirectly attach the flexor tendons to bone, thereby governing eccentric action of the former. In the horse’s forelimb, the check ligaments also serve as tension bands to stabilize the carpus, fetlock, and digit, thereby transmitting the effects of the primary and secondary components of the stay-apparatus to the lower limb. The proximal (SDF) accessory ligament establishes a ligamentous connection between the horse’s lower radius and pastern that can help to discourage inadvertent overextension of the carpal and fetlock joints during stance. The distal (DDF) accessory ligament provides a ligamentous connection between the horse’s cannon (third metacarpal) and coffin bones, thereby preventing exaggerated extension of the fetlock, pastern, and coffin joints.

The Suspensory Apparatus

The suspensory ligament, proximal sesamoid bones, and distal sesamoidean ligaments comprise the foundation of the horse's suspensory apparatus (fig. 17.5). These structures, which are physically linked to one another, work in unison (as a single unit) to maintain normal lower-limb posture during stance. As a conglomerate of ligaments, this apparatus is primarily engaged for support and not voluntarily applied for the purpose of moving discrete portions of the horse's limb.

17.5 Basic Elements of the Suspensory Apparatus



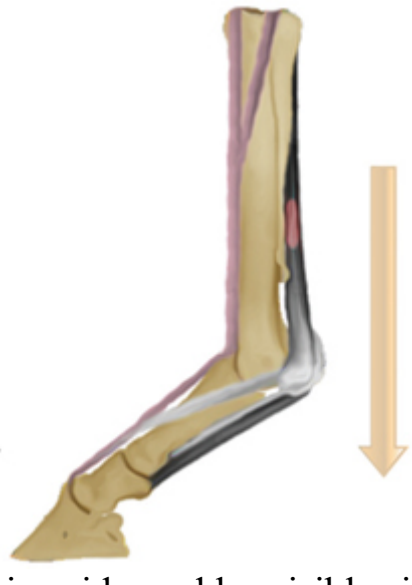
The primary function of the suspensory apparatus is to support (or literally "suspend") the fetlock joint, which is being driven downward toward the ground surface with great force while under the exorbitant weight of the horse. This force is contested by employment of the apparatus, which effectively prevents hyperextension and excessive "dropping" of the fetlock joint.

In addition to supporting the horse's lower limb, the suspensory apparatus also retains an involuntary spring-like effect that is implemented during locomotion. While the fetlock is extended under weight-bearing load, the apparatus stretches, thereby storing potential energy for future use. This energy is returned to the limb as the fetlock begins to flex during the latter part of stance (just before the limb leaves the ground surface). This both facilitates limb action and improves stamina.

Aside from its elastic effects, the suspensory apparatus (comprised exclusively of ligamentous tissue) is not directly exploited during movement. That said, associated pathology does have the potential to generate biomechanical lameness either through excessive or restricted activity. The elastic effects, for instance, may be dramatically diminished by the presence of scar tissue (which can form adhesions) within or between aspects of the apparatus that normally move independently of one another. A ligament that cannot contort or deform normally during limb movement may over-stabilize (or "bind") the structures to which it is attached, thereby abbreviating their respective activity. In many cases, a reduction in the limb's range of motion due to the presence of adhesions can generate a visible alteration in the horse's way of moving.

As we might guess, excessive laxity affiliated with the suspensory apparatus (via loss of structural integrity associated with one or more of its components) will also produce lameness. Loss of suspensory support is typically demonstrated by excessive "fetlock drop" during stance (fig. 17.6 and **VL 17b**). In the case of complete breakdown of the apparatus, the fetlock may ultimately sink near or to the level of the ground surface, precipitating a pathognomonic gait deficit.

17.6 Pathognomonic Appearance of Severe Compromise or Rupture of the Suspensory Apparatus



Deterioration of the suspensory apparatus is evidenced by visible sinking of the fetlock joint. In severe cases, the fetlock may drop until the flexor apparatus is maximally engaged and/or until the fetlock contacts the ground surface. If the deep digital flexor tendon (DDFT) is intact, the foot will remain flat on the ground surface.

VL 17b

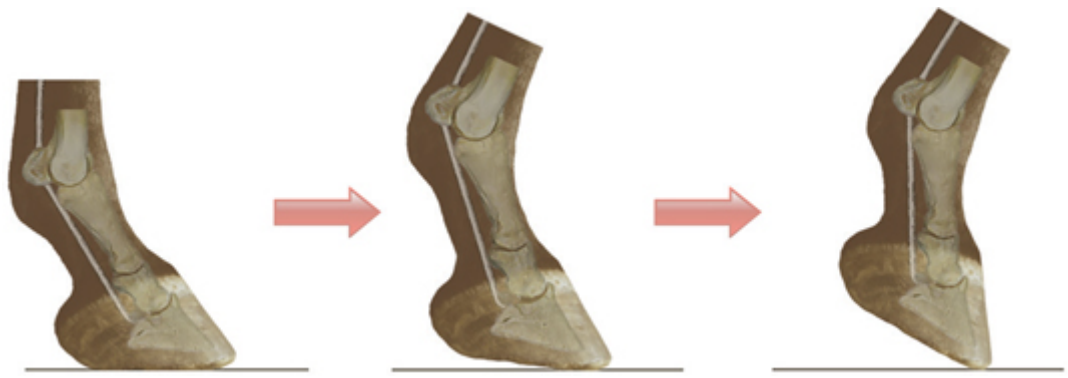
Scan/Click to view video.

www.getsound.com/tutorials/17b

Breakover

During ambulation, the horse's hoof works like a lever with the toe acting as the fulcrum (or pivot point). The term *breakover* refers to the action of the hoof as it pivots over the toe to lift and move the limb forward (fig. 17.7). This process is initiated via combined tension within the deep digital flexor muscle and distal accessory (inferior "check") ligament, both of which act through the deep digital flexor tendon (DDFT) and suspensory ligaments of the navicular bone (fig. 17.8).

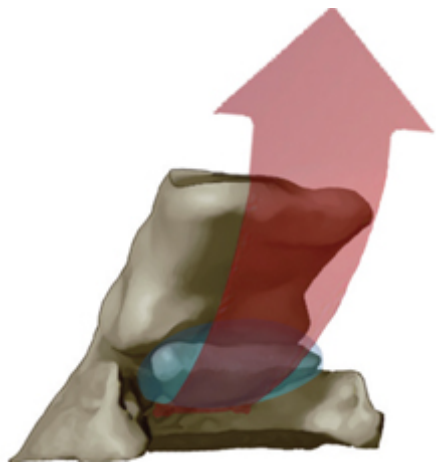
17.7 The Process of Breakover



A. Before breakover: The limb is in mid-stance.
B. Early breakover: This is just after heel-off, which represents the instant that the heel leaves the ground surface.
C. Late breakover: This is just prior to toe-off, which represents the instant that the toe leaves the ground surface.

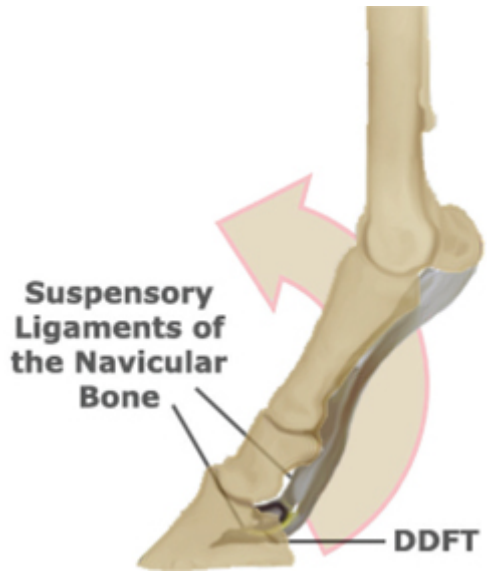
We use the term *breakover* to describe the phase of the stride between the moment that the horse's heel raises up and off the ground, and the moment that the toe is lifted. During this phase, the toe acts as a pivot point (or fulcrum) around which the heel rotates.

17.8 Sequence of Physiologic Events Preceding Breakover



A. Initial Phase
1. The deep digital flexor muscle tightens.
2. The deep digital flexor tendon (red) tightens.
3. Pressure is applied to the navicular bursa (blue).
4. Hydraulic pressure is applied to the navicular bone through the navicular bursa.

B. Middle Phase
5 A. The distal (impar) and proximal suspensory ligaments of the navicular bone tighten (green).
5 B. Tension increases at the insertion of the deep digital flexor tendon (orange).



C. Final Phase

6. The coffin bone rotates in response to these forces, thereby initiating the breakover process.

The timing of breakover (the point at which the hoof tips forward to begin the cranial or flight phase of the stride) is dictated by DDFT/navicular ligament tension combined with the amount of force required to overcome the leverage that is intrinsic to the horse's foot and limb. Differences in limb length/conformation, hoof wall length/angle, hoof-pastern limb angle, footing type/consistency, and trimming/shoeing strategy(ies) can all affect the timing of foot breakover.

On a hard surface, the hoof remains flat on the ground until heel-off (i.e. when the heel leaves the ground). On a softer surface, the toe rotates into the footing prior to heel-off, thereby attenuating tension affiliated with the DDFT and navicular ligaments. This, in turn, alleviates pressure along the navicular region. This is one reason why softer and deeper footing often benefits horses experiencing pain within this area. Concurrent application of **bar shoes** can discourage sinking of the heel(s) into the footing (via the "snow-shoe effect"), thereby alleviating navicular discomfort even further.

Contrary to what we might predict, decreasing external leverage associated with the horse's foot does not always accelerate its breakover. For instance, wedged pads are commonly used to "quicken" heel-off, especially in the forelimbs. Although application of a wedged pad increases hoof angle and decreases external hoof leverage, it also mitigates tension along the deep digital flexor tendon and distal accessory ligament (both of which instigate the breakover process). The increase in hoof angle certainly decreases the amount of DDFT/DAL tension required to initiate breakover, but the actual timing of breakover might be prolonged in this setting. In the majority of cases, however, natural breakover is delayed as a result of long-toe/low-heel conformation: more time and effort is required to rotate the heel around a longer lever arm. This configuration imposes further challenge to the structures responsible for administering the breakover process and may even predispose animals to tripping (see chapter 12, p. 52).

> *Pathology affiliated with DDFT tension and/or navicular ligaments can cause horses to falter (trip) in the forelimbs. A sense of pain that intensifies as natural breakover is approached (and these structures encounter increased tension) may overwhelm the animal and prompt premature lifting of the affected limb(s). This, in turn, increases the horse's risk for tripping. An attempt to avoid this form of discomfort is the reason why many horses with deep digital **flexor tendonitis,** navicular inflammation, etc., will not only pick the affected limb(s) up early, but walk and trot with their forefeet extended out in front, clearly avoiding the caudal (or posterior) phase of the stride.*

In general, the timing and pattern of foot breakover reflect both the extrinsic and intrinsic influences acting on the foot's internal anatomy at any point in time. The structures that govern foot breakover must be engaged to the appropriate degree and at the proper time for the horse to perform naturally.

18
Basic Axial Anatomy

The orientation, function, and comfort of the median anatomy directly influences the way in which horses move their limbs. In reciprocity, the effects of limb action are continually endured and managed by the horse's neck and back. Accordingly, issues involving the limbs and median structures regularly go hand in hand. The vertebral column effectively converts horizontal impetus from the hind limbs into vertical energy through a multitude of harmonized (and mostly imperceptible) movements. It also works to prepare the hind limbs for proper engagement and impulse. The concerted effort put forth by the axial and **appendicular** skeletons visually communicates the relative status of the horse's musculoskeletal system and separates "good movers" from "poor movers."

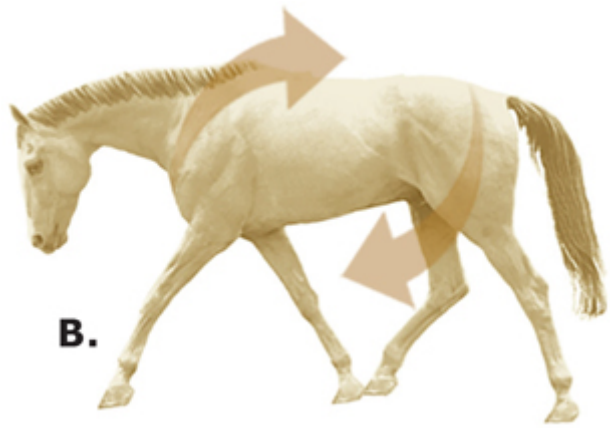
Lame horses can display multiple variations of axial pathology: some are primary in nature and some develop secondary to issues originating in the limbs. Regardless of the underlying cause, most forms significantly impact the horse's overall performance (especially under saddle). In order to establish the location and nature of any related problem, we must first understand the basics of how horses ordinarily move their median anatomy in addition to the typical strategies they use to circumvent pain. Since operation of the axial anatomy is primarily a result of joint, muscle, and ligament action, we should be extra attentive with regard to our functional anatomic consideration of these structures.

On account of the fact that the axial skeleton is oriented along the dorsal plane (horizontally) and doesn't directly engage the ground surface, we might be hesitant to acknowledge its role as a weight-bearing structure. The back does transmit the rider's weight to the limbs, however, and is therefore influenced by forces that prevail during the stance phase of the stride. It also serves to delegate impetus from the limbs to the rider's seat. The horse's axial bones, joints, muscles, and ligaments are regularly challenged during both the flight and stance phases of locomotion, particularly when activity occurs under rider direction.

From a passive standpoint, it is often helpful to view the horse's thoracic and lumbar spinal regions as sections of a platform or bridge that is suspended between the horse's four limbs (which act like structural pillars). The attachment of the bridge to the forelimbs is strictly muscular and relies on a combination of *serratus ventralis* and *pectoral* employment. Hind limb connection occurs without the assistance of muscles but instead through the sacroiliac (SI) joint, which physically links the vertebral column to the horse's pelvis. The pelvis serves as the base for hind limb action through the hip joints. The central aspect of the bridge is supported via the coordinated effort of muscles, ligaments, and joints, which constantly adapt to both extrinsic (rider) and intrinsic demands (fig. 18.1 A).

18.1 Basic Roles of the Horse's Median Anatomy

A. While under saddle, the back functions as a physiologic mediator between the rider's position, weight, and cues (coming from above) and input from the horse's limbs (coming from below). Both normal and abnormal activity is constantly managed in this way.



B. Horses employ their axial anatomy to facilitate and increase engagement of the hind limbs, which serve as their "engine" during proper movement. Hind-limb engagement is promoted via favorable lumbosacral (LS) orientation, which is achieved through ventroflexion (or "rounding") of the horse's lower back.

Flexibility of the back not only provides for passive support of the trunk and rider but also plays an active role in propulsion (forward movement) of the animal (fig. 18.1 B). Axial muscle operation orients the body and positions the limbs so that power, control, and efficiency are maximized. In this setting, the back and lumbosacral joint are ventroflexed and the hind limbs become the "engine" by applying the horse's weight to push the body up and forward. The front end is effectively lightened, thereby augmenting its role as the veritable "steering wheel." The orientation of the lumbosacral joint is integral to the nature and degree of hind limb engagement, which in turn governs the strength of the horse's engine and the direction of its propulsive force.

Axial pathology can have a number of deleterious consequences with respect to the horse's performance, not excluding visible "hollowing" of the back, loss of hind limb engagement, an inability to perform collected movements, and an unpleasant "front-wheel drive" way of moving. As we learned in chapter 12 (see p. 52), pain associated with the horse's median anatomy can also induce behavioral resistance during work.

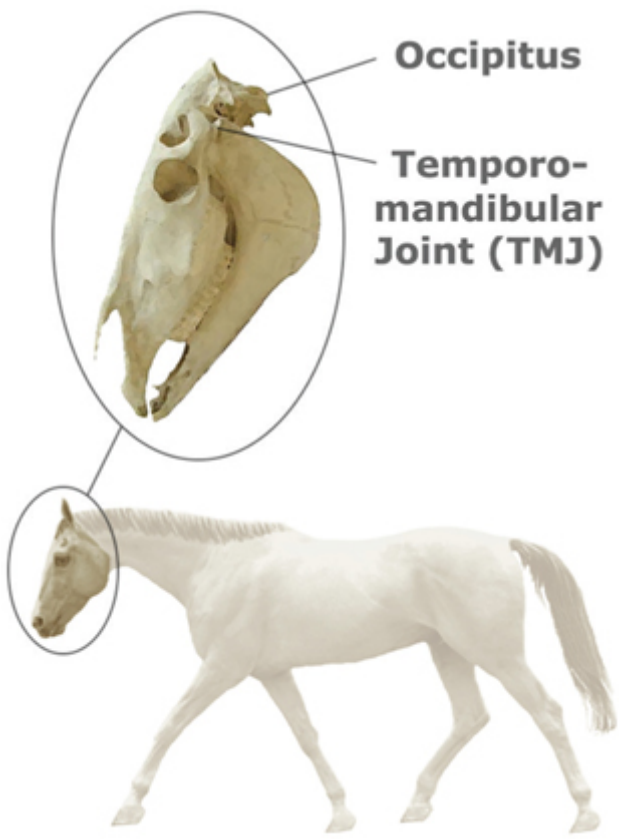
Axial Bones

The horse's skull (at the front end), the hyoid apparatus (associated with the larynx or voice box), the sternum (keel), a continuous series of cervical, thoracic, and lumbar vertebrae (along the length of the neck and back), the ribs, the sacrum, pelvis, and coccygeal vertebrae (at the "tail end") comprise the horse's axial skeleton. The number of vertebral bodies making up the spinal column may vary slightly depending on the individual, although the average horse owns about fifty-four. These bones are divided into five basic groups:

- Seven *cervical* (or neck) vertebrae.
- Eighteen *thoracic* (or upper back) vertebrae.
- Five to six *lumbar* (or lower back) vertebrae.
- Five (fused) *sacral* vertebrae (constituting the sacrum).
- Fifteen to twenty-five (in most cases eighteen) *coccygeal* (or caudal) vertebrae.

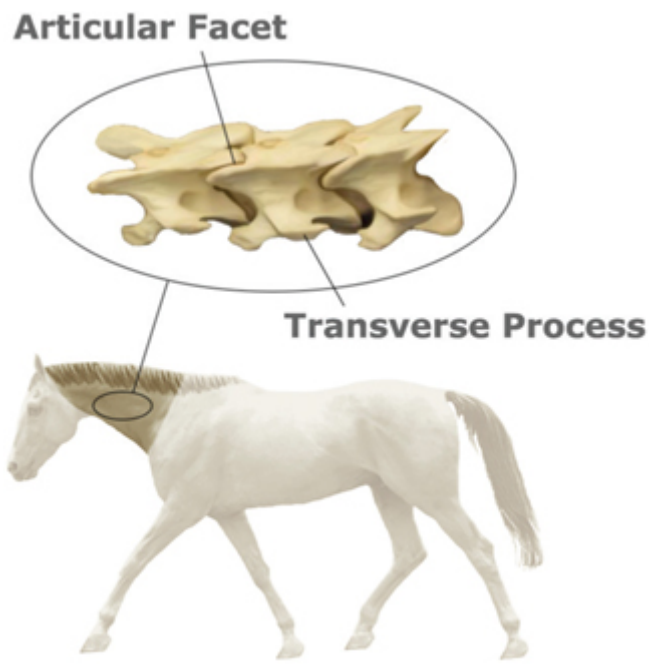
The skull is composed of many bones, most of which interact through synarthrodial (immobile) joints (fig. 18.2). The cervical spine links the head with the chest through a series of seven vertebrae, the majority of which articulate with one another via right and left facet joints (fig. 18.3). The vertebral bodies contain a central canal through which the spinal cord passes. Fracture, intervertebral disc disease, vertebral malformation (abnormal bone development), and bone cyst formation are some forms of pathology affecting the neck bones that can generate pain-mediated and/or neurologic lameness in the horse.

18.2 The Horse's Skull



The horse's skull comprises many bony plates that are fused together. The right and left temporomandibular joints (TMJs) permit movement between the horse's upper and lower jaws. The occipitus at the back of the skull articulates with the first vertebra of the cervical spine (in the neck).

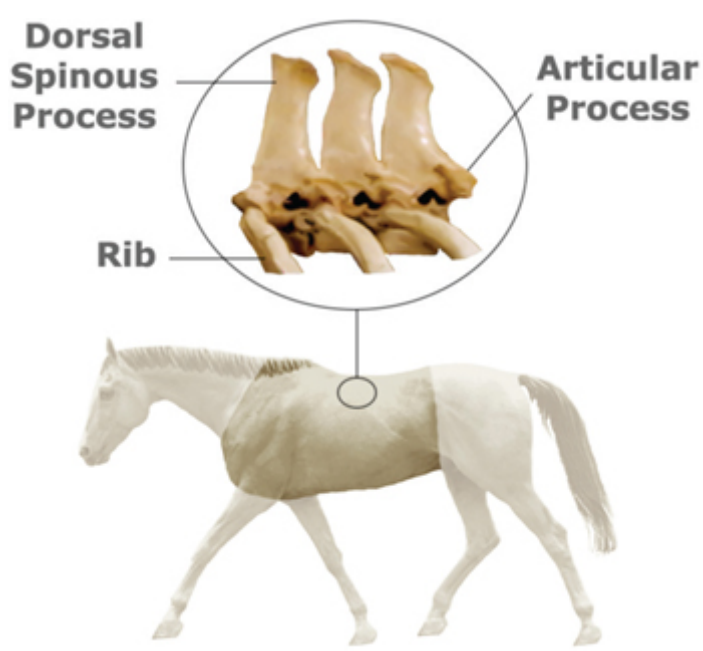
18.3 The Horse's Cervical Spine



The horse's neck comprises seven vertebrae. The first vertebra (called the atlas or C1) articulates with the occipitus along the back of the skull through a *condyloid joint.* The atlas also articulates with the second vertebra (called the axis or C2) through a *pivot joint.* The remaining neck vertebrae articulate via a combination of gliding articular facet joints and intervertebral disks.

The thoracic spine starts at the base of the horse's neck (near the point of the shoulder) and continues backward to incorporate about two-thirds of the length of the back. The dorsal spinous processes of the thoracic vertebrae are comparatively large and form the framework for the horse's withers (fig. 18.4). *Fistulous withers* denotes an infection often involving these bony processes.

18.4 The Horse's Thoracic Spine



Each thoracic vertebra accommodates six articulations: right and left facet joints (2) that link it to the adjacent vertebra in front, right and left facet joints (2) that link it to the adjacent vertebra behind, and right and left articulations with the respective ribs (2).

Five to six lumbar vertebrae comprise the posterior third of the horse's back. The dorsal spinous processes of the lumbar vertebrae are not as prominent as those in the thorax, although these structures are prone to similar problems arising as a consequence of direct pressure (rider weight) and/or abnormal spinal conformation. Impingement of the dorsal spinous processes (often referred to as "kissing spine syndrome") is a condition most often observed between the T10 to L6 articulations in the horse (the region of the back directly influenced by saddle pressure). Pain developing at and around sites of bony overlap can generate visible (axial) lameness, often accompanied by a significant behavioral component. This condition is more common in horses that regularly encounter excessive rider weight, ill-fitting tack, and/or **spinal lordosis.** *Ankylosing spondylitis* refers to chronic inflammation associated with the vertebral bodies, an ailment that can eventually lead to **fusion** at single or multiple levels.

The sacrum, which resides behind the lumbar back, is comprised of five individual vertebrae that are fused together. It sits within the bony "cage" formed by the pelvis, articulating with the latter via the right and left

sacroiliac (SI) joints. The coccygeal vertebrae form the bony column within the horse's tail.

Axial Joints

Axial joint pain is a common cause of poor performance in the horse and has the potential to develop anywhere along the median plane where two bones move with respect to one another. Relative movement within the horse's head occurs in two locations:

1. Between the hyoid apparatus and temporal bone of the skull. The hyoid apparatus is comprised of five separate bones that function to maintain the position of the horse's larynx (voice box) and support the horse's pharynx and tongue. Its cartilagenous (synchondrotic) attachment to the skull affords very little motion and, therefore, does not routinely factor into our visual assessment of lameness.

2. Between the temporal bone of the skull and mandible (lower jaw bone). The horse's pair of synovial *temporomandibular joints* or "TMJs" permit free movement of the upper and lower jaws with respect to each other, such as occurs during chewing. Each TMJ sits just below and in front of the base of the respective ear along the side of the head (see fig 18.2, p. 116).

The temporomandibular joints are subjected to considerable torque in response to rein tension, which (through action of the bit) applies pressure to the lower jaw. As with any joint, the horse's TMJs can succumb to extreme and/or atypical forces, thereby becoming unstable, inflamed, and a potential source of discomfort. Accordingly, TMJ pain is frequently implicated as a cause of axial lameness in the horse. Affected horses often express behavioral resistance during moments of increased rein tension.

The *atlanto-occipital joint,* a condylar synovial articulation, connects the horse's head and neck and is principally employed during flexion and extension of the poll (fig. 18.5 A). This joint also supports extensive lateral (side-to-side) movement. The *atlanto-axial joint* between the first and second vertebrae utilizes a pivot synovial articulation to permit thorough rotation of the horse's head relative to the neck (fig. 18.5 B). The articulations between the remaining cervical, thoracic, and lumbar vertebrae entail the application of gliding, synovial facet joints in combination with cartilaginous intervertebral joints. The disks that separate and cushion the vertebral bodies do not encase smooth cartilage or synovial fluid (which are the basic components of diarthrodial joints).

18.5 Basic Atlanto-Occipital and Atlanto-Axial Joint Action

A. The atlanto-occipital articulation between the skull and first cervical vertebra employs a condyloid joint that permits extensive up-and-down and side-to-side movement of the head.

B. The atlanto-axial articulation between the first and second cervical vertebrae employs a pivot joint that permits extensive rotation of the head in both directions.

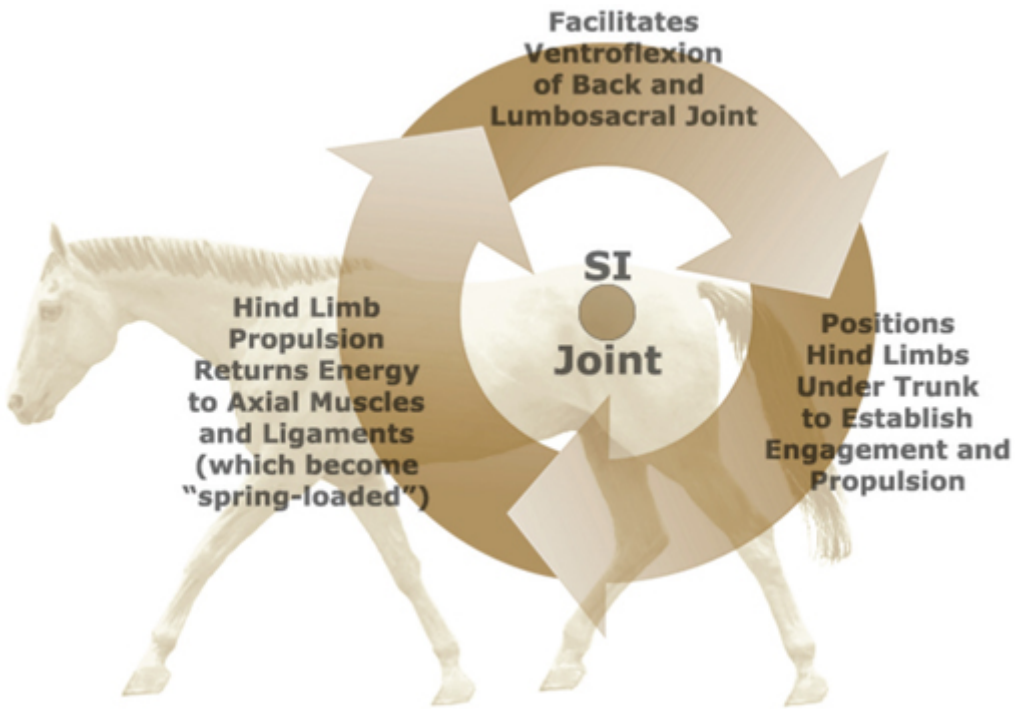
Arthritis associated with the cervical facet joints is a relatively common cause of neck-related lameness, particularly in older horses. Trauma, dislocation, and developmental orthopedic issues (such as OCD) can all lead to painful joints in the neck, although chronic (long-term) degeneration due to increased age and use is the most common cause. Related symptoms include general neck stiffness, decreased range of motion (especially from side to side), and resistance to contact with the bit. Arthritis within the caudal (posterior) facets can generate characteristic non weight-bearing gait deficits associated with the ipsilateral forelimb (on the same side of the horse) on account of related nerve impingement.

Each thoracic vertebra accommodates six articulations: right and left facet joints (two) that link it to the adjacent vertebra in front, right and left facet joints (two) that link it to the adjacent vertebra behind, and right and left articulations with the respective ribs (two). The joints along the horse's thoracic and lumbar regions provide limited lateral flexion (from side to side along the dorsal plane); they do, however, enable considerable dorso-ventral movement (up and down along the median plane).

Inflammation associated with the inter-vertebral joints is a relatively common cause of axial lameness in the horse. The sense of discomfort is closely related to tension experienced by joint-capsule fibers at their bony attachment sites. Many nerve endings reside within the joint capsule and are stimulated to a greater extent during periods of stretching (as opposed to compression). This is an important detail, as it often exposes the affected side of a horse that consistently chooses to bend in one direction.

Sacroiliac (SI) joint discomfort is another common source of axial lameness. This structure constitutes the articulation between the sacrum (below) and iliac wings of the pelvis (above), thereby serving as the juncture between the vertebral "bridge" and the hind limbs. Tremendous forces are mediated through the SI joint whilst the horse is in motion (fig. 18.6). This structure regulates the transfer of energy from the horse's median muscles and ligaments through the hind limbs to the ground. It subsequently returns energy in the form of propulsion from the ground back and into the horse's median anatomy. Disproportionate transmission of this energy dramatically influences the interaction between the back and hind limbs, which may, in turn, impair the strength, efficiency, and symmetry of movement (see **VL 5d**, p. 28).

18.6. The Role of the Sacroiliac (SI) Joint in Managing Normal Equine Locomotion



The horse's sacroiliac (SI) joint mediates the physiologic interaction between the horse's back and hind limbs. It is the first to encounter the effects of hind-limb lameness, which are mitigated as they pass through and into the back. Physical adjustments made in response to primary back pain are also moderated by the SI joint as the horse attempts to sustain an appropriate degree of power and engagement behind. In this context, the SI joint serves as a veritable *transmission* for the horse, transferring available power between the median anatomy and the hind limbs.

The SI joint can become unstable and/or painful in chronic cases of back pain and/or hind-limb lameness, thereby impairing its ability to reconcile forces being exchanged between these regions. For instance, back pain has the potential to precipitate instability within the SI joint, which consequently relinquishes the capacity to promote active engagement of the hind limbs. Hind-limb lameness can also instigate SI joint pain, weakening the ability of the latter to return propulsive energy back into the median anatomy. In both cases, affected horses are unable to effectively generate power from behind and often deliver the impression that they are "pulling themselves around with the front end." Due to the intimate relationship between the horse's median anatomy, hind limbs, and sacroiliac joint, problems in all three areas frequently occur simultaneously.

Axial Muscles

Somewhat dissimilar to the reciprocating action of most appendicular (limb) muscles, activity between flexors and extensors along the axial skeleton doesn't naturally alternate. Rather these muscles work in unison to maintain proper tension and orientation of the vertebral platform between the front and hind limbs at all times. Pathology associated with any element of this "muscular ensemble" has the potential to disrupt the horse's natural way of going.

Axial muscles perform two primary functions (fig. 18.7):

1. To stabilize the median anatomy and prevent excessive motion.

2. To move the median anatomy in response to normal locomotive provocation.

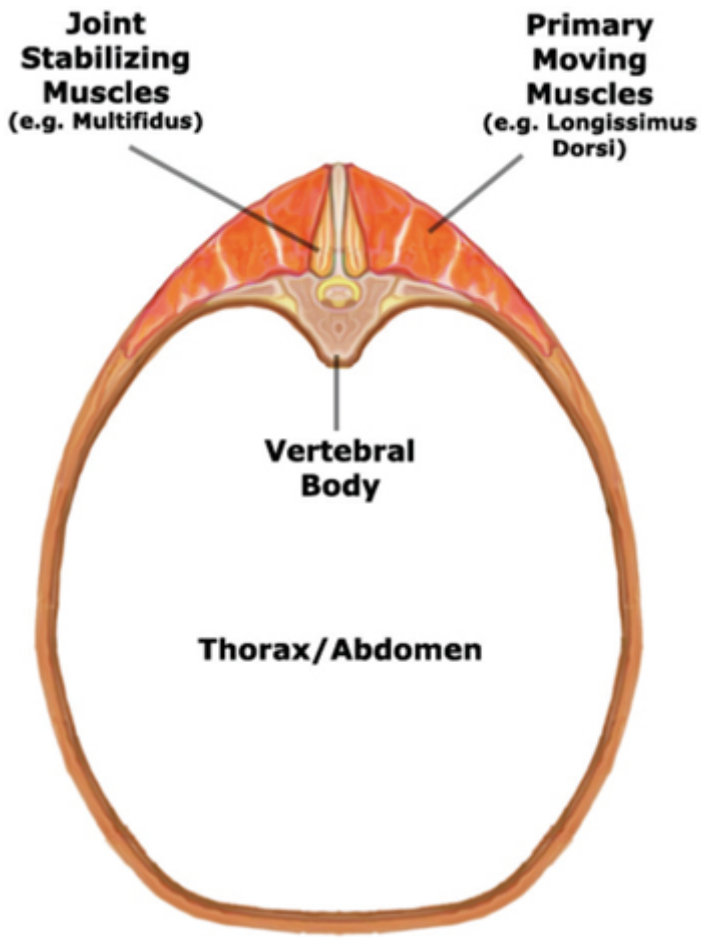
18.7 Basic Anatomic Function of the Horse's Back Musculature

Basic Composition	Basic Characteristics	Typical Location	Example Structures
Primary Function: Joint Stabilization			
Type I ("slow-twitch") muscle fibers	• Short in length • Slow contraction • Limited range of motion • **Pennate** fiber arrangement • Generate small movements • Very strong action • Excellent stamina (they don't fatigue easily) • Extremely well-innervated • Highly anticipatory action and response	Located deep in the tissue, close to bones and joints. These muscles only cross one to three articulations.	*Longus colli, transverse abdominis,* and *multifidus* muscles.
Type IIA ("fast-twitch") muscle fibers	• Short in length • Moderately fast contraction • Limited range-of-motion • Pennate fiber arrangement • Generate small movements • Very strong action • Fair stamina (relatively quick to fatigue) • Well-innervated • Anticipatory action and response		
Primary Function: Axial Movement			
Type IIB ("fast-twitch") muscle fibers	• Long in length • Rapid contraction • Excellent range of motion • Parallel fiber arrangement • Generate large movements • Very strong action; provide power and speed to movement • Poor stamina (they fatigue easily) • Less innervation • Reactive action	Located superficially, near the skin surface. These muscles cross multiple articulations.	*Rectus abdominis, iliocostalis, longissimus, external abdominal oblique, internal abdominal oblique,* and *iliopsoas* muscles.

Numerous muscles work collectively to manage axial activity in a very sophisticated and orchestrated manner. Movement typically progresses along multiple planes and across multiple articulations simultaneously. Interestingly, an important aspect of normal back function entails the *resistance* of movement against natural forces (as opposed to the *instigation* of movement). By securing the horse's spine, the short joint-stabilizing muscles help to discourage the development of facet arthritis and associated soft tissue (primarily ligament)

damage that can progress in the face of chronic instability. The stabilizers are sensitive to various forms of local affliction, however, upon which they may become "decommissioned" or deactivated. In these instances, animals will often solicit their primary "movers" to help maintain spinal stability. The result is long muscle spasm and a braced, sore, and stiff-moving animal (see VL 5e, p. 29). Unfortunately, resolution of the underlying pathologic cause doesn't always trigger reactivation of the stabilizing muscles, a task generally accomplished through rehabilitative exercise and physical therapy. Short axial myopathies, therefore, usually require intervention beyond just the successful management of their primary (underlying) cause.

18.8 The Stabilizers and Movers of the Horse's Back



The *short, deep stabilizing muscles* regulate axial movement so as to protect the intervertebral joints and ligaments from injury. The *long, superficial actuating muscles* serve to move the back in response to external (e.g. rider) and internal stimuli.

The long muscles are employed during voluntary movement of the axial skeleton, which usually entails dorsoventral (up-and-down progression along the median plane), lateral (side-to-side progression along the dorsal plane), and/or rotational (twisting along the transverse plane) activity. Contraction of muscles situated above (or *dorsal* to) the level of the spine induces dorsiflexion (or "hollowing") of the vertebral column (see fig. 14.11 A, p. 83). These muscles also serve to elevate the front end of the horse whilst the hip joint is flexed and the hind limbs are actively engaged. The *splenius* and *semispinalis* muscles perform this function along the horse's neck, while the *erector spinae* assumes the majority of this role in the thoracolumbar region. Contraction of the *erector spinae* counteracts the concentric effects of muscles located beneath the vertebral column and promotes spinal dorsiflexion, closer juxtaposition of the dorsal spinous processes (exacerbating cases of impingement), depression of the anterior pelvis, and dorsiflexion (or extension) of the lumbosacral joint.

Muscle contraction below (or *ventral* to) the level of the spine yields ventroflexion (or "rounding") of the median anatomy (see fig. 14.11 B, p. 83). This action is accomplished via employment of the *brachiocephalicus* and *sternocephalicus* muscles in the neck and the abdominal *(rectus abdominis* and *oblique abdominis)* and sublumbar *(iliopsoas)* muscles along the thoracolumbar segment. Contraction of these muscles promotes flexion of the lumbosacral joint, thereby depressing the posterior aspect of the pelvis and positioning the hind limbs farther forward under the body of the horse (where they can be more effectively employed for propulsion). The *iliopsoas* muscle, which inserts along the upper aspect of the femur, further bolsters engagement of the hind limbs by helping to initiate their forward progress. This muscle works in conjunction with the *erector spinae* to achieve concurrent lumbosacral flexion and thoracic extension, thus forming the corporal foundation for collected movement(s).

Dorsoventral manipulation of the horse's spine can also be influenced by:

- Relative positioning of the limbs.

    - Gathering of the limbs underneath the body (retraction of the forelimbs and protraction of the hind limbs) accentuates the horse's ability to round the back during movement. This configuration appropriately enhances the ability of the pelvis to transfer energy from the hind limbs to the spine.
    - Forward placement (protraction) of the forelimbs and rearward placement (retraction) of the hind limbs accentuates the horse's tendency to hollow the back during movement. This arrangement hinders the ability of the pelvis to communicate power to the horse's trunk during movement.
- Positioning of the horse's head and neck.
    - Elevating the head and neck:
        - Decreases overall axial mobility (especially along the lumbar region).
        - Induces extension (hollowing) in the thoracic region and flexion (rounding) in the lumbar region.
    - Depressing the head and neck:
        - Increases overall mobility of the vertebral spine.
        - Induces flexion (rounding) in the thoracic region and extension (hollowing) in the lumbar region.

It would be logical to assume that lateral (side-to-side) movement of the spine occurs in response to alternating contraction of muscles located along either side of the vertebral column. Indeed no such muscle exists. Rather, this role is adopted by concerted action of the extensors and flexors along each side, which combine to produce the overall effect of a single (theoretical) laterally situated structure. For instance, simultaneous contraction of the right *erector spinae* and oblique abdominal muscles will incite the horse's thorax to bend toward the right. Symmetric and asymmetric engagement of these muscle pairs enables the horse to maintain the appropriate axial contour when traveling in straight lines or around turns, respectively.

Due to the unique composition of the vertebral articular facets, rotation of the spine (along a transverse plane) typically occurs in conjunction with lateral flexion. Accordingly, pathology that is provoked by vertebral rotation will impact the horse's ability to bend laterally and vice versa. At the trot, an upward impetus generated by the weight-bearing limb and a lack of force associated with the non weight-bearing limb naturally causes the lower (ventral) aspect of the spinal column located between the limbs to rotate toward the weight-bearing side. The dorsal spinous processes, situated on top of the vertebrae, would accordingly rotate toward the non weight-bearing side. Based on the anatomical mechanics of the vertebral articulations, this engenders passive lateral flexion (or bending) toward the horse's weight-bearing forelimb. In other words, the sound horse's body naturally bends to the right when the right forelimb is in contact with the ground surface, and to the left when the left forelimb is bearing weight.[10]

Axial Ligaments

Numerous short ligaments link individual vertebrae and work to maintain appropriate alignment and stability of the spinal column. Two long ligamentous structures further operate to provide strong, elastic tension along the top border of the neck and back. The *nuchal ligament* extends from the back of the skull to the point of the withers. It facilitates counter-levering of the head and neck, which pivot about the horse's **cervicothoracic** junction. During movement, the nuchal ligament receives and returns energy to the head and neck via its outstanding viscoelastic properties, thereby attenuating regular oscillatory instigation from the forelimbs. The ligament is heavily engaged upon flexion of the poll and neck (such as occurs while in a collected frame). Related pathology, known as *nuchal desmopathy,* can discourage this form of desired movement and motivate the horse to carry the head high and the neck in an inverted (or dorsi-flexed) manner.

The *supraspinous ligament* enjoys similar viscoelastic properties to those of the nuchal ligament. This structure extends from the point of the withers to the sacrum and provides passive stability to the thoracolumbar spine. It stabilizes the horse's back when loaded in compression, but also returns essential energy to improve trunk posture and hind limb engagement. Ligament pathology typically occurs between the T15–L3 articulations, which comprise a region of the spine that normally retains a considerable degree of mobility (as compared to the anterior segment of the thorax, for instance). Accordingly, the obvious restriction in vertebral "swing" often attributed to supraspinous desmopathy is readily perceived by the observer.

Although the nuchal and supraspinous ligaments are separate structures, their common attachment at the point of the withers integrates their resilient influence across the entirety of the spine from the head to the sacrum. They cooperate to flex the thoracolumbar region when the head is lowered, thereby increasing the relative space between dorsal spinous processes and alleviating articular facet joint capsule tension. **Desmopathy** of one or both structures will often persuade the horse to reduce associated tension, which is most effectively accomplished via elevation of the head and hollowing of the neck, thorax, and lumbar spine.

19
Altered Muscle Anatomy and Function

The relative status of the horse's musculoskeletal anatomy dictates what we see with respect to both normal and abnormal movement of the body and limbs. At the root of this movement are the horse's muscles that function to influence the position of bones relative to one another. Muscles can attach directly to bone or indirectly through their associated tendon structure (see fig. 14.1, p. 74). They rely on the accessory roles of joints, tendons, and ligaments to perform their assignments properly.

Accurate recognition of biomechanical lameness requires some basic knowledge of the horse's muscular anatomy. Maladies that alter the conventional **biomechanics** of the horse's muscles have the potential to generate visible gait deficits, either by restricting normal movement or permitting abnormal movement.

Biomechanical lameness occurs, therefore, as a result of the inability of one or more muscles to perform its intended function in the intended manner (fig. 19.1).

19.1 Intrinsic (Muscular) Causes for Biomechanical Lameness

Biomechanical Effect	Potential Underlying Pathology
Concentric Causes	
The muscle cannot contract to its minimum length.	• Myopathy resulting in decreased myofiber contractility. • Corresponding joint has limited range of motion.
The muscle contracts beyond its normal (minimum) length.	• Corresponding (displaced) bone fracture with loss of architectural integrity, allowing the attachment points to move too close together. • Corresponding joint dislocation or loss of collateral support, allowing the attachment points to move too close together.
The muscle does not contract at the normal rate (too slow).	• Myopathy resulting in muscle atrophy/weakness. • Neuropathy resulting in muscle paresis.
The muscle does not contract at the normal rate (too fast).	• Neuropathy resulting in hyperactive muscle function.
The muscle cannot contract at all.	• Ruptured or detached muscle structure. • Neuropathy resulting in muscle paralysis.
Eccentric Causes	
The muscle cannot stretch to its maximum length.	• Myopathy resulting in decreased myofiber elasticity. • Corresponding joint has limited range of motion.
The muscle stretches beyond its normal (maximum) length.	• Corresponding (displaced) bone fracture with loss of architectural integrity, allowing the attachment points to move too far apart from one another. • Corresponding joint dislocation or loss of collateral support, allowing the attachment points to move too far apart from one another.
The muscle does not relax at the normal rate (too slow).	• Myopathy resulting in decreased myofiber elasticity.
The muscle does not relax at the normal rate (too fast).	• Myopathy resulting in decreased myofiber tone.
The muscle cannot relax at all.	• Neuropathy resulting in hyperactive muscle function. • Myopathy resulting in restrictive tissue fibrosis.

It might be helpful to consider the action of a bungee cord and how it not only influences, but is likewise counter-influenced, by the items to which it is attached on either end. By stretching and recoiling, this appliance affects the way that the two articles maneuver with respect to one another. When operating normally, the cord allows two items to be separated at a defined rate and to a fixed distance, for example. The eventual rate and distance of separation may be directly dictated by the degree of tension encountered by the individual bands that comprise the appliance. Tension of these bands may also serve to draw the articles closer together at a specific rate and to a predetermined length, both of which would certainly depend on the relative positions, weight, and mobility of the coupled items.

A variety of circumstances could change or govern the way that the bungee cord influences reciprocal movement of its linked items. Loss of band elasticity or tensile strength due to excessive age or repeated hyperextension, for instance, may slow or decrease convergent migration of the attached items with respect to one another, but at the same time accelerate divergent progression. Adjusting the weight, position, or mobility of one or both items would also alter their relative activity. Cutting the bungee cord in half would eliminate any influence it had on the attached articles. Depending on the anomalous conditions at hand, a disparity in movement (from what we would expect to see) may be perceptible.

Muscles, which contract to shorten and relax to lengthen, manipulate the relative positions of bones in analogous fashion to that of a bungee cord influencing the maneuverability of two connected items. As observers, we use our eyes to detect unexpected modifications in movement of certain bones arising from aberrant muscle action. A visible gait deficit will manifest if these bones are employed during locomotion and if their mobility is altered in a substantial way. We can appropriately implicate biomechanical lameness when irregular musculoskeletal activity is localized to a specific region and expressed in a predictable way and on a consistent basis.

We should note that inflammation and pain may or may not accompany biomechanical forms of lameness. Indeed, most pathognomonic deficits are not uncomfortable (see chapter 27, p. 195). When inflammation is present, though, horses will consciously resist excessive contraction or relaxation of the *myofibers* (muscle tissue) in an attempt to avoid pain. If the affected muscle comprises part of the horse's stay-apparatus (the triceps muscle in the forelimb for instance), then weight-bearing lameness will be evident. If the muscle is not a part of the horse's stay-apparatus but is employed during locomotion then associated pain usually generates non weight-bearing deficits (see **VL 16i**, p. 104). Either way, biomechanical lameness, like other forms of asymmetry, has the capacity to generate secondary deficits, including those that are referred (or artificial). Thus even non pain-mediated problems can cue the horse to unexpectedly shift weight in an attempt to facilitate travel.

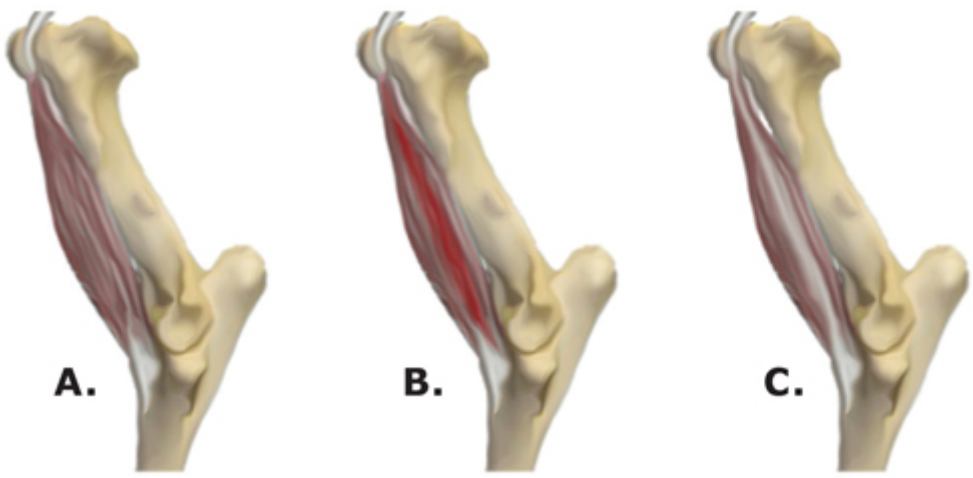
Fibrotic Myopathy

Injury to muscle will influence the way that it manipulates the horse's bones as well as the way it is counter-affected by bone movement. Local tissue damage can occur as a result of both intrinsic (e.g. limb hyperextension, congenital disease, internal abscessation, etc.) or extrinsic (e.g. blunt trauma/bruising, complications associated with injection, laceration, etc.) causes. Some forms of insult give rise to the development of scar (or *fibrotic*) tissue that can form between muscle fibers within a single structure **(intramuscularly)** and/or between separate muscle bellies **(intermuscularly).** Fibrotic tissue is composed of **collagen fibers,** which do not possess the same inherent qualities as normal muscle fibers, particularly with respect to contractility (the ability to shrink) and pliability (the ability to stretch). As a consequence, the presence of scar tissue within or around muscle can alter normal function of the latter.

> *Since mature scar tissue doesn't contract to any appreciable degree, its presence can govern the concentric action (shortening) of muscle. It also doesn't stretch significantly, thereby limiting the eccentric action (lengthening) of muscle. In a sense, scar tissue acts more like a ligament (which is designed to stabilize two structures relative to one another) as opposed to a muscle (which is intended to enable movement between two structures relative to one another).*

Fibrotic myopathy refers to muscle pathology arising from the abnormal development of associated scar tissue (fig. 19.2). This disease can affect any muscle tissue, although the presence of scar tissue within certain structures will have a greater impact on their employment. Inability of muscle to contract or relax at the appropriate rate and/or to the expected degree will produce altered movement of the bones to which it is attached. This, in turn, can generate gait abnormalities that become apparent as the horse ambulates. The conspicuity of corresponding lameness will depend on a number of factors:

19.2 The Evolution of Fibrotic Myopathy



A. Pre-injury: Healthy muscle prior to insult. No evidence of associated lameness is observed.
B. Acute myositis: Hemorrhage, swelling, and pain often accompany the onset of injury to the muscle. Pain-mediated lameness may be observed.
C. Chronic fibrosing myopathy: Ensuing development of scar (fibrotic) tissue, which takes about eight weeks to fully mature and organize, may eventually lead to restricted muscle action and an associated (biomechanical) gait deficit.

- *Which bones are moved by the affected muscle(s)? Are they large or long bones that are employed during locomotion?* Abnormal motion associated with bones that are long and directly utilized during ambulation is more easily discerned with our eyes.
- *What type of bone movement do the affected muscles generate? Does muscle activity create movement along the sagittal plane (in a front-to-back direction) or from side-to-side?* Since the limbs typically travel the greatest distance along the sagittal plane of the horse, abnormal movement of bone with respect to this plane is easiest to see.
- *Which aspect(s) of the affected muscles is fibrotic? Is the scar tissue located within a small portion of the muscle near its tendon insertion or does it extend along the entire length of the belly?* The discrepancy in bone movement will depend on how much the function of the muscle is compromised by the presence of scar

tissue. If the muscle is still able to perform its duty with relative competence, then corresponding alterations in gait may be difficult to perceive.

- *What percentage of muscle fibers are fibrotic? Are just a few fibers affected or is a significant percentage of the muscle involved?* As the percentage of muscle fibers that are replaced with collagen fibers increases, the overall functionality of the structure decreases. This translates into more obvious gait deficits.
- *What degree of fibrosis is present? Are a few collagen fibers interposed with normal muscle fibers, providing it with some degree of contractility/pliability? Or is the injured muscle replaced with dense, thick scar tissue that cannot shorten or lengthen to any appreciable degree?* Mature scar tissue that is comprised entirely of collagen fibers will demonstrate limited ability to move bones, both from concentric (moving the bones closer together) and eccentric (permitting the bones to move farther apart) standpoints. This degree of pathology has the potential to generate pathognomonic alterations in limb action.

> *Since many bones are not directly employed by the horse during locomotion, fibrosis of their governing muscle(s) may not generate visible lameness, even if pathology is severe. On the other hand, if the horse relies heavily on movement of the attached bones to ambulate normally, even mild disease has the potential to produce noticeable gait deficits.*

In all cases of fibrotic myopathy, the presence of organized scar tissue can impose biomechanical restrictions on the affected muscle(s). Associated lameness, therefore, is usually a result of physical limitations within the muscle rather than inflammation or pain. In other words, fibrotic myopathies typically don't hurt. Appropriately, their accurate diagnosis relies chiefly on our ability to recognize and characterize correlating alterations in the horse's expected way of moving.

This book will highlight two forms of fibrotic myopathy commonly associated with equine lameness. Both forms affect movement with respect to the sagittal plane of the horse and demonstrate purely non weight-bearing deficits: shortened cranial (forward) and lengthened caudal (backward) phases of the stride.

Fibrotic Myopathy of the Hamstring Musculature

The most common manifestation of fibrotic myopathy in the horse is that affecting the hamstring musculature of the hind limb, which is composed of the *semitendinosis, semimembranosis,* and *biceps femoris* groups. Of these groups, the *semitendinosis* is most frequently affected. This muscle serves to extend (straighten) the hip joint and flex (bend) the stifle joint. It also helps to medially rotate the tibia (mid-limb) when the stifle is flexed and medially rotate the femur (upper limb) when the hip is extended.

Gait deficits arising from fibrosis of the hamstring musculature are most frequently observed in Quarter Horses due to the type of work that they perform. Hyperextension of the hind limb, one of the prevailing causes of this affliction, often occurs during reining, cutting, and roping activities. Non Quarter Horse breeds commonly hyperextend their hind limb(s) while trying to abruptly stop after running up to a fence. In these instances, one or both hind limbs will often slide up and underneath the horse's belly (especially if the footing is slippery), inadvertently causing the horse to "sit down." Interestingly, the acute inflammatory reaction (myositis) that ensues immediately post-injury may not produce visible gait deficits and often goes undetected due to the relative size and location of the musculature involved. Rather, lameness usually emerges after restrictive scar tissue has developed and adequately matured during the chronic phase of healing.

> *In most cases of hamstring fibrotic myopathy, corresponding gait deficits emerge as a consequence of affected musculature being unable to perform its eccentric duties: it cannot lengthen appropriately.*

Healthy hamstring muscles move independently of one other and can each stretch to allow adequate cranial extension of the hind limb (in the forward direction). However, injury leading to the development of scar tissue within or between hamstring muscle bellies can restrict these actions, leading to dramatic shortening of the hind stride. Since the hamstrings reside along the backside of the limb, forward movement (protraction) of the limb is predominantly affected.

As scar tissue organizes and matures, it can also contract, forming a "rope-like" band where pliable muscle tissue once existed. This band may or may not be visibly evident as it courses along the back and inside of the horse's inner thigh (fig. 19.3 A). In any event, its inherent inability to lengthen prevents the horse from completing the forward (flight) phase of the stride (fig. 19.3 B). The hind limb is pulled caudally (backward) before full stride length is reached, causing the respective foot to "slap" the ground surface upon impact. This

action is often labeled *goose-stepping* and is a classic (pathognomonic) manifestation of this muscle disease. It is most clearly expressed at the walk (**VL 19a**).

19.3 Visible Evidence of Fibrotic Myopathy of the Hamstring Musculature

A. A visible defect (depression) can sometimes be discerned along the back and inside of the horse's lower thigh, corresponding to the location of the medial head of the *semitendinosis* muscle. This indication is not evident in all cases of hamstring fibrotic myopathy, however.

B. As the horse extends the limb forward during protraction (flight), scar tissue associated with the hamstring musculature eventually becomes engaged. The affected limb is pulled backward before the stride is completed, provoking exaggerated impact ("slapping") of the foot on the ground surface. In the case of unilateral disease (affecting only one hind limb), the horse will often overextend the contralateral (other) hind limb in an attempt to support its weight and maintain balance.

VL 19a
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Fibrotic Myopathy of the *Biceps Brachii* Musculature

Dissimilar to that observed with reference to the horse's hamstring musculature, forelimb lameness resulting from *biceps brachii* myopathy is more often attributed to inflammation and pain accompanying an acute insult, such as blunt trauma, laceration, or hyperextension (**VL 19b**). Gait deficits can also reflect chronic, internal muscle

fibrosis, however, which customarily results in compromised protraction, stride hypometria, and a toe-first landing in the affected limb(s). Also in contrast to the hamstring musculature, concentric action (contraction) of the biceps muscle is primarily influenced by this form of pathology. Loss of contractility decreases the rate and extent to which the biceps muscle shortens, thereby reducing the rate and degree to which the forelimb is protracted (fig. 19.4). The result is explicit non weight-bearing lameness.

VL 19b

Scan/Click to view video.

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19.4 Visible Evidence of Fibrotic Myopathy of the *Biceps Brachii* Musculature

The biceps musculature is situated along the front of the forelimb and is principally engaged during protraction. Fibrotic myopathy of the biceps affects its ability to contract normally, thereby precipitating a delayed, hypometric stride with a toe-first landing.

In most cases of biceps fibrotic myopathy, corresponding gait deficits emerge as a consequence of affected musculature being unable to perform its concentric duties: it cannot shorten appropriately.

In severe/chronic cases of fibrosing myopathy, scar tissue can organize to the point of **ossification,** during which it assumes the characteristics of bone. This can take many months or even years to develop, whereas "soft" fibrosis can generate related gait deficits within six to eight weeks following injury. As you might expect, ossified tissue enjoys even less pliability than fibrotic tissue, exacerbating visible alterations associated with the horse's gait.

SECTION V

The Fundamentals of Productive Observation

We can learn a lot by watching how the horse moves. Exactly how much we learn relies on the art of pattern recognition, which is elementary once we know when, where, and how to look for potential problems. The process gets even easier as we create and manipulate a setting with the intention of further increasing the conspicuity of the horse's irregular movement. Our primary assessment objectives are two-fold:

1. To detect potential lameness.

2. To identify and classify as many characteristics of the horse's gait as possible.

We're going to outline some tips that will both expedite and facilitate our realization of these goals. Ultimately, it's about making things as easy and as obvious as possible.

20
The Game Plan

We visually assess our horses with the intention of recognizing potential lameness and surmising the likely source(s) of the problem. Satisfying our ambition is relatively painless when the horse is noticeably "off"; it can be considerably more difficult when gait abnormalities are visibly faint. Fortunately, we can make lameness more conspicuous by:

- Improving our ability to see it.
- Maximizing the horse's expression of it.

Choosing the best approach, gait, and setting for our assessment will decidedly support our efforts.

Choosing the Approach

Lameness evaluation is a daunting venture for many of us because there are so many visual components to the horse's gait. Making rhyme or reason out of what we're seeing can sometimes seem to be an insurmountable task. We should realize, however, that we are not required to process all of the visual input simultaneously. In fact, our optical acuity is significantly sharpened when we are only asked to assess one thing at a time. By following a step-wise approach, we force ourselves to interpret each aspect of the horse's gait independently from one another, thereby simplifying the overall process. A single, large, complicated exercise essentially becomes many small simple exercises. In the end, we can use our compilation of impressions to complete our assessment and formulate an opinion with respect to how the horse is moving.

Once we've established the necessary steps, we can then arrange them in a way that maximizes the efficiency of our examination. It is important that we follow the same basic procedure with each and every assessment, whether it is performed on a single animal multiple times or many different animals. With practice, the examination process will become very familiar and progress quickly for you. Most seasoned performance veterinarians, for instance, can navigate through a multitude of "sub-examinations" to complete their overall visual assessment of a horse in less than 10 minutes.

Each of the evaluation steps is intended to distinguish a specific feature of the horse's lameness:

- Which regions are affected?
  - Is the horse lame in the forelimbs?
  - Is the horse lame in the hind limbs?
  - Is the horse expressing axial lameness?
- What is the nature of the lameness?
  - Is there a weight-bearing component?
  - Is there a non weight-bearing component?
- What is the severity or grade of lameness?
  - Is it evident while the horse is standing still?
  - Is it evident at the walk?
  - Is it evident at the trot?
  - Is it only evident under certain circumstances?
- At which gait(s) is lameness observed?
  - Is it evident at the walk?
  - Is it evident at the trot?
  - Is it evident at the canter?
  - Is it evident during any other gait(s)?
  - Is it evident during upward or downward transitions?
- What factors exacerbate the lameness?
  - How does the lameness change with regard to surface?
  - How does the lameness change with regard to direction?
  - How does the lameness change with regard to velocity?
  - How does the lameness change with regard to acceleration or deceleration?
  - How does the lameness change under saddle?
  - How does the lameness change with regard to duration of activity?
  - How does the lameness change with regard to temperature?
- Are there any distinguishing traits to the lameness?

Answering each of these questions separately takes the complexity out of the process but still enables us to perform a comprehensive evaluation of the horse's gait. Things begin to make sense once we integrate all of the

puzzle pieces that we've gathered during the course of our assessment.

Choosing the Gait

Most observers find it easiest to observe horses at the trot because it is the simplest gait, comprising a *two-beat stride pattern* in which the horse's weight is distributed evenly between diagonal pairs of limbs (see fig. 10.1 and **VL10a**, p. 47). The left hind and right front limbs comprise one diagonal pair, whereas the right hind and left front limb comprise the other. Assessing movement of the horse in motion requires that we evaluate both diagonal pairs, visually comparing one with the other. The slower the horse moves during our assessment, the more time our eyes have to pick up on relative discrepancies.

At the trot, the two limbs constituting each diagonal pair have a special relationship with each other:

- A horse that chooses to underload one limb of a diagonal pair will proportionately overload the other so as to support its own weight (fig. 20.1 A). It is important to remember that lameness is defined as any alteration in gait and can manifest as a result of underloading in one limb and/or overloading in a compensating limb—the visual interpretation of bearing not enough weight in one limb or too much weight in the other limb may be *identical.*
- Any alteration in the timing and length of one limb's stride will be mirrored in the stride of its diagonal counterpart so as to maintain synchroneity and balance (fig. 20.1 B). This principle constitutes the basis for *diagonal synchrony of stride,* which was previously discussed in chapter 10 (see p. 46).

20.1 Physiologic Relationship Between Diagonal Limbs

A. A horse that underloads one limb within a diagonal pair will proportionately overload the other at the trot. **B.** The timing and length of one limb's stride will be mimicked by the other limb within the same diagonal pair at the trot.

Once the relationship between diagonally paired limbs is acknowledged, detection of a limb's altered response to factors affecting its diagonal counterpart can actually *facilitate* (rather than complicate) our visual interpretation of lameness.

Choosing the Venue

In order to get the most out of our assessment, we should go to the trouble of making the horse's asymmetric movement as transparent as possible. Most of us have the choice of speed, gait, surface congruity, footing, and direction at our disposal. Choosing the right venue for our assessment can make all the difference when it comes to recognizing important visual markers (fig. 20.2). Periodically modifying environmental factors as we navigate through the evaluation process, as we'll see in chapter 26 (p. 183), can also be extremely rewarding.

20.2 Setting for Basic Visual Lameness Assessment

Gait	Walk Trot Canter	
Direction	Straight line	Walk Trot
	Clockwise circle	Trot Canter
	Counterclockwise circle	Trot Canter
Surface	Hard	Walk on a straight line Trot on a straight line
	Soft	Trot in circles Canter in circles

Increasing the horse's asymmetric movement can transform an obscure lameness into one that is obvious, thereby simplifying our job as effective observers. Altering the horse's environmental setting can also facilitate our isolation, identification, and classification of significant gait characteristics.

21
Rules of Effective Lameness Assessment

As with any technique, there are inherent *dos* and *don'ts* when it comes to observing the lame horse. Adhering to a few basic guidelines can both simplify and enhance our visual judgment and clinical reasoning.

Basic Rules of Observation

Rule #1: Practice, practice, practice. Work hard to sharpen your observation skills. Observe as many lame horses as you can, even if it requires you to seek out video footage to review online. A setting in which a single examiner is able to evaluate a large number of subjects has been suggested to be a crucial part of refining one's subjective diagnostic accuracy.[11]

Rule #2: Watch your horse move on a regular basis (at least weekly). This practice will enhance your ability to discern gait alterations during the early stages of lameness, before multiple primary or secondary issues have as much chance to develop. With fewer areas of the horse being affected, lameness evaluation is considerably simplified. Moreover, changes in your horse's movement will become more obvious once you've established a visual baseline. Video acquisition can facilitate this process by allowing for day-to-day comparisons of your horse's gait.

Rule #3: Employ the help of your veterinarian whenever possible. It is very likely that your equine practitioner has observed many lame horses and could provide further insight into what you're seeing. If you've acquired video footage, pertinent clips can easily be sent to your veterinarian for expeditious review and consultation.

Rule #4: Look for consistent patterns of abnormal movement as opposed to brief flashes of lameness. Stepping on a rock or reacting to some other temporary environmental impediment can generate an obvious, short-lived gait deficit that might disappear within a few minutes. Remember, we're not looking for an odd step here or there. Rather, we are looking for consistent *patterns* of movement, both normal and abnormal. If you can't convince yourself that there is some degree of regularity associated with the altered movement you're seeing, it probably doesn't deserve your undivided attention. That said, if you notice the lameness for more than a few hours or over multiple days, it likely carries clinical significance. As we learned in chapter 12 (p. 52), some intermittent forms of lameness occur very sporadically and only when they are triggered by a specific set of circumstances. It is important to pay attention to these potential instigators with the intention of establishing a pattern for what you're seeing.

Rule #5: Avoid limb gear. Abstain from applying equine supportive or protective wear for the purpose of your evaluation. Polo wraps or brushing/splint/bell boots can be visually distracting for the observer, especially when brightly colored. Moreover, both tactile and weight stimulation afforded by the gear can artificially alter the horse's movement. You'll get a more authentic portrayal of the horse's gait when it is "naked."

Rule #6: Start by evaluating the horse "cold" (e.g. after several hours of stall confinement). Most horses are "stiffest" in the beginning and accordingly display more obvious gait deficits before they warm up. Moreover, the appearance of lameness may change with prolonged activity, thereby complicating your assessment in the event that a "cold baseline" impression had not been preemptively established. Valuable information can usually be gleaned through observing any gait mutation that evolves over the course of examination.

Rule #7: Keep it slow. Start by moving the horse as slowly as possible for the purpose of your assessment. This tactic will help to accentuate the appearance of asymmetry, especially at the trot. Slowing the subject's movement gives our eyes more time to process visual input. In addition to abating the horse's movement, try to keep the velocity consistent throughout each facet of your evaluation.

Rule #8: Start big and work your way down to the small things. Inspect the horse in a deliberate, logical, and consistent manner. Watch the horse move as a whole at first, then start visualizing the smaller pieces:

- Try not to zero in on any particular aspect of the horse to start. Rather, begin by evaluating the horse's entire frame to get a feel for how the body is moving through space. This will help you to identify the horse's lame side (left or right). Subtle asymmetries will be much easier to depict if you can discern a one-sided dip of the horse's entire skeleton.
- Secondly, assess each end (front and back) of the horse independently of one another, starting with the front. Determine if the horse is lame up front, then move to the back and determine if the horse is lame behind. Make sure that you always take the time to assess both halves. Remember, we have to determine the affected end(s) of the horse in addition to the affected side(s).
- Once you've established the lame side(s) and end(s) of the horse, then evaluate motion of the limbs. This is the time to determine the nature and severity of the horse's asymmetry. Since alterations in axial motion will

almost always manifest in limb-lame horses (secondarily), we'd want to confirm regular and symmetric limb dynamics prior to implicating another region (such as the back) as the primary cause of the problem.

- Once you've isolated the lame limb(s), assess the horse's median activity. In cases of primary axial pathology, you may perceive alterations in flexion, bending, and/or carriage of the neck and back in the absence of overt limb asymmetry. If limb lameness was previously established, accurate characterization of axial movement will contribute further insight into the overall nature of the problem.
- Wait to assess distinguishing gait characteristics (such as stride length and height) until after you have identified the affected region(s) of the horse. If you observed altered movement affiliated with more than one region (e.g. two separate limbs), inspect each one individually. It is much easier to characterize the movement of one structure as opposed to defining the activity of multiple structures simultaneously.

Rule #9: Recognize the comfortable (rather than the lame) side of the horse. In other words, don't try to find the horse's lame limb. Remember, we're not trying to see the horse's problem but rather how the horse avoids the problem and/or facilitates movement in the face of the problem. *Look for the sound limb:* this is the one that the horse transfers weight *onto.* From a visual standpoint, it is decidedly easier to watch the horse descend or "fall into" the comfortable side as compared to favoring the affected side (fig. 21.1 and **VL 21a**).

21.1 Establishing the Sound Side

It is much easier to see the horse "fall into" the comfortable side as opposed to favoring the lame side. In this instance, the horse is sinking heavily into the left forelimb, indicating lameness in the right forelimb.

VL 21a
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Rule #10: Answer as many questions relating to the horse's lameness as you can, but don't worry if you can't answer all of them. As we'll see in section VI (p. 157), our evaluation process will entail answering a series of predetermined queries. Our responses will lead us to the likely location(s) and, in some cases, the source(s) of the horse's lameness. Fortunately, we're not always obligated to provide feedback with respect to each and every inquiry. In some instances, answering just one or two questions enables us to make accurate conclusions with regard to the basis for the observed lameness.

Rule #11: Don't fixate on one thing. Avoid the temptation to focus on the most obvious gait deficit throughout the course of your assessment. In many cases, the most pronounced abnormality is *secondary* and therefore should not be the sole focus of investigation. An obvious forelimb lameness, for instance, could easily go unsolved in the event that it is *referred* from behind and the examiner doesn't take the time to separately assess hind limb activity

(VL 21b). Make sure that you take the time and energy to examine each and every aspect of the *entire* animal prior to determining where to target your attention.

VL 21b
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Rule #12: Consider relationships between coexisting deficits. The elements of a complicated lameness (in which multiple limbs are affected) may be related in some way, especially if the degree and nature of asymmetry among them is comparable. Thinking along these lines will help to prevent you from needlessly pursuing secondary or tertiary components of the problem. If you perceive forelimb lameness, for instance, always try to find an excuse for the abnormal movement by assessing hind gait and vice versa. There's a very good chance that two coexisting asymmetries are physiologically linked; it is undeniably rare to find multiple independent problems manifesting within the same horse at the same time.

Rule #13: Watch the horse move in a figure-eight pattern whenever possible, with each circle being approximately 8–10 meters (or 25–35 feet) in diameter (fig. 21.2). This will allow you to easily compare differences in the horse's movement from one direction to the other, as well as detect any deficits that emerge during directional or lead changes. This exercise necessitates the cooperation of a rider or someone who is willing to acquire video footage of you riding the horse.

21.2 Trotting in a Figure Eight

Progression in a figure-eight pattern allows you to quickly compare the differences in the horse's weight distribution and stride length from one direction to the other. Ideally, the diameter of the loops at each end should be 8–10 meters (or 25–35 feet). This activity naturally requires the assistance of a rider.

Rule #14: Know your subject. Background knowledge of the breed as well as any exclusive traits of the horse undergoing evaluation is very conducive to effective observation. It also helps to know the variety of problems that your equine athlete is likely to encounter based on the discipline in which he or she is participating. For instance, racehorses are more likely to develop *palmar* metacarpal condyle bone disease (along the *back* of the fetlock joint), whereas show jumpers are more likely to develop *dorsal* metacarpal condyle bone disease (along the *front* of the fetlock joint). A career that imposes extreme rotational forces (torque) on the hind limbs (such as cutting, roping, barrel racing, and dressage) may predispose a horse to lower hock pain (distal tarsitis). Biomechanical interference of the stifle is more often observed in horses asked to collect heavily, such as gaited horses and those performing upper-level dressage. Access to archived (baseline) video footage of the subject moving soundly can further facilitate the recognition and characterization of recently acquired abnormalities.

Rule #15. Don't give up! Avoid the temptation to convince yourself that abnormal or asymmetric movement is inherent to (or "just part of") the horse's genetic makeup. It is easy to implicate an extraneous reason for the horse's lameness, especially if you're having difficulty locating its source. You should realize, however, that your inability to establish the reason behind the gait deficits does not mean that there isn't one. Genetic causes are

extremely rare, especially in older animals. Careful, diligent investigation will usually reveal a more common and logical source to the problem.

22
Key Visible Elements of the Equine Gait

Like any definitive mark, the horse's gait comprises an array of visible features. These features reflect movement of the median anatomy, the quality of stride, limb **kinetics,** and foot positioning. As competent observers, we're steadily hunting for visual asymmetry: we're looking for something on one side of the horse that moves differently than the same thing on the other side. In order to recognize discrepancies in motion, however, we first have to know the features of the horse's gait that warrant our scrutiny.

Let's review the forms of asymmetry that we're likely to notice during our assessment:

- Asymmetry in *axial movement.*
- Asymmetry in the *quality of stride.*
- Asymmetry in *limb gesturing.*
- Asymmetry in *foot dynamics.*

Asymmetry in Axial Movement

The walk and trot are two gaits that enable us to easily compare symmetry between the horse's right and left sides and right and left diagonals. Equal activity observed between both sides and diagonals would imply that the horse is sound. Irregular activity that appears consistently during every other stride would signify that the horse is lame.

Asymmetry in median excursion. Regular up-and-down movement (or **oscillation**) of the horse's axial anatomy occurs at the walk and trot (fig. 22.1 and **VL 22a**). The amplitude (or height) of these oscillations may differ between diagonal strides in the lame horse (figs. 22.2 A–D and **VL 22b**). In the case of weight-bearing lameness, the sound (comfortable) limb pushes up harder (causing increased elevation of the trunk during takeoff) and bears more of the weight (causing excessive dropping of the trunk during stance). This depression (or dropping) of the median anatomy is easier for most of us to see (and sometimes hear). The opposite effect occurs in the lame (uncomfortable) limb. Correlating asymmetry observed in the axial oscillation between the horse's strides is an indication of lameness.

22.1 Median Excursion of the Sound Horse

A. Forelimb Soundness: Regular, symmetric movement of the head and withers suggests that the front end of the horse is sound.

B. Hind Limb Soundness: Regular, symmetric movement of the croup suggests that the back end of the horse is sound.

C. Overall Soundness: Regular, symmetric movement of the head, withers, and croup suggests that the horse is sound.

VL 22a
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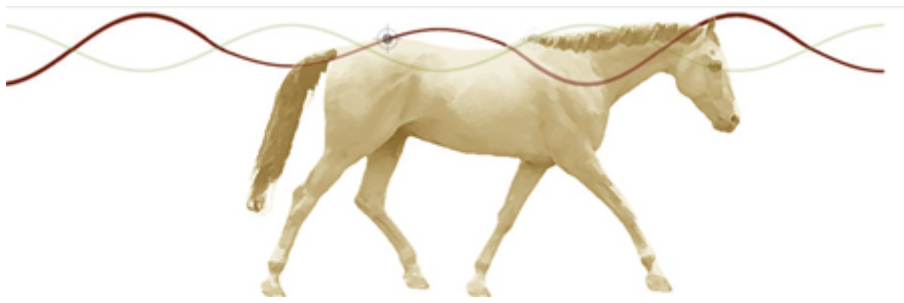
Figure 22.2 Median Excursion of the Lame Horse

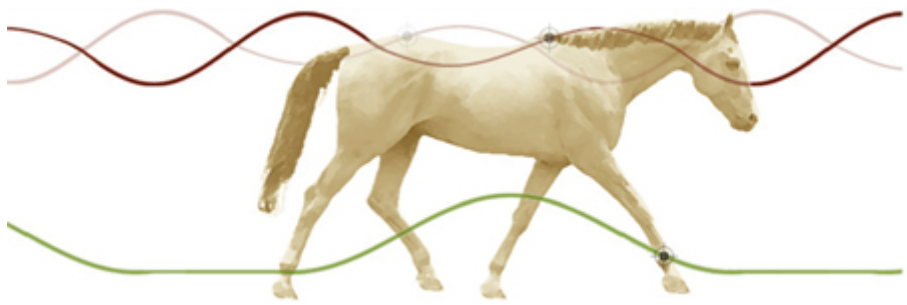
A. Forelimb Lameness

Regular, asymmetric movement of the withers suggests that the front end of the horse is lame. Regular, symmetric movement of the croup suggests that the back end of the horse is relatively sound. In this instance, the right front limb is in contact with the ground surface as the withers drop excessively. This would signify weight-bearing lameness of the left front limb.



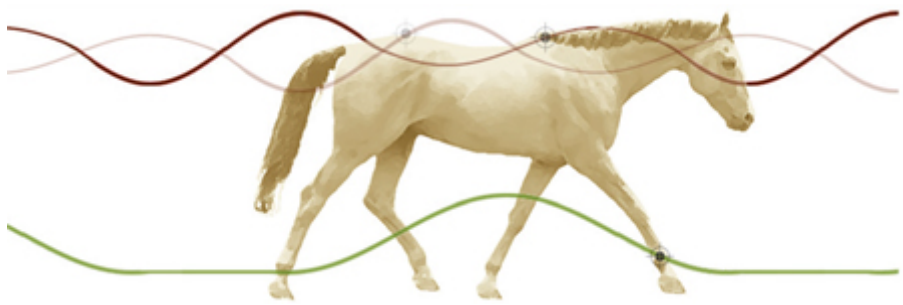
B. Hind Limb Lameness

Regular, symmetric movement of the withers suggests that the front end of the horse is relatively sound. Regular, asymmetric movement of the croup suggests that the back end of the horse is lame. In this instance, the left hind limb is in contact with the ground surface as the croup drops excessively. This would signify weight-bearing lameness of the right hind limb.



C. Complicated Lameness (Authentic)

Regular, asymmetric movement of the withers suggests that the front end of the horse is lame. Regular, asymmetric movement of the croup suggests that the back end of the horse is also lame. In this instance, the right front and left hind limbs are in contact with the ground surface as the withers and croup drop excessively, respectively. This would signify concurrent weight-bearing lamenesses of the left front and right hind limbs. *Since all gait deficits are weight-bearing and coexist in limbs comprising the same diagonal pair, it is unlikely that they are physiologically related.*



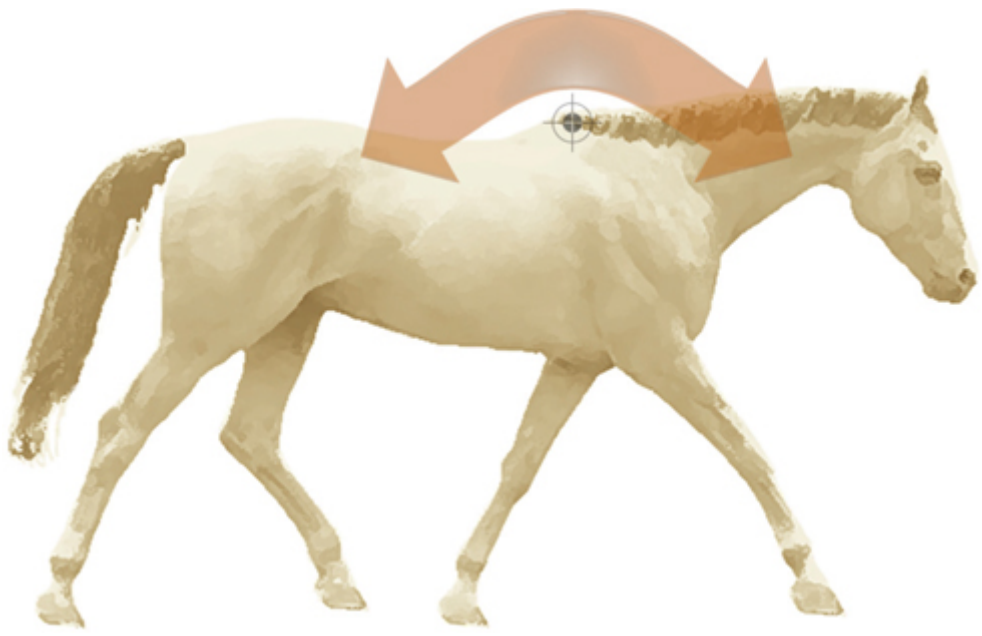
D. Complicated Lameness (Artificial)

Regular, asymmetric movement of the withers suggests that the front end of the horse is lame. Regular, asymmetric movement of the croup suggests that the back end of the horse is also lame. In this instance, the right front and right hind limbs are in contact with the ground surface as the withers and croup drop excessively, respectively. This signifies concurrent weight-bearing lamenesses of the left front and left hind limbs. *Since all gait deficits are weight-bearing and coexist in limbs comprising different diagonal pairs, it is likely that they are physiologically related and that left-fore lameness is referred from the left hind.*

VL 22b
Scan/Click to view video.
www.getsound.com/tutorials/22b

Most of us are visually drawn to the horse's head, which tends to migrate over appreciable distances whilst the horse is walking and trotting. We should note, however, that abnormal head movement (even when significant) is not always conveniently ascribed to a specific side or diagonal of the horse. In many instances, the lame side of the horse cannot be determined via assessment of head action alone. The best place to focus our attention (at least at first), therefore, is the horse's withers. In most cases of forelimb lameness, the observer can perceive the horse's neck and back flexing around the point of the withers during the stance phase of the comfortable limb (fig. 22.3 and **VL 22c**). Indeed it is very helpful when movement of the head and neck endorse our findings, although this doesn't always happen.

22.3 Targeting the Withers to Discern Forelimb Asymmetry



Focusing on the action of the horse's neck and back as they flex over the point of the withers often provides the most accurate appraisal of forelimb soundness.

VL 22c
Scan/Click to view video.
www.getsound.com/tutorials/22c

In the sound horse, there is no appreciable difference in the degree of excursion (up-and-down movement) of the thoracic median anatomy (the withers) between diagonal strides. Regular, asymmetric excursion of the withers is observed in the horse exhibiting front end lameness, however. Concurrent irregular movement of the head will be recognized in more severe (and obvious) cases, but not necessarily in all instances.

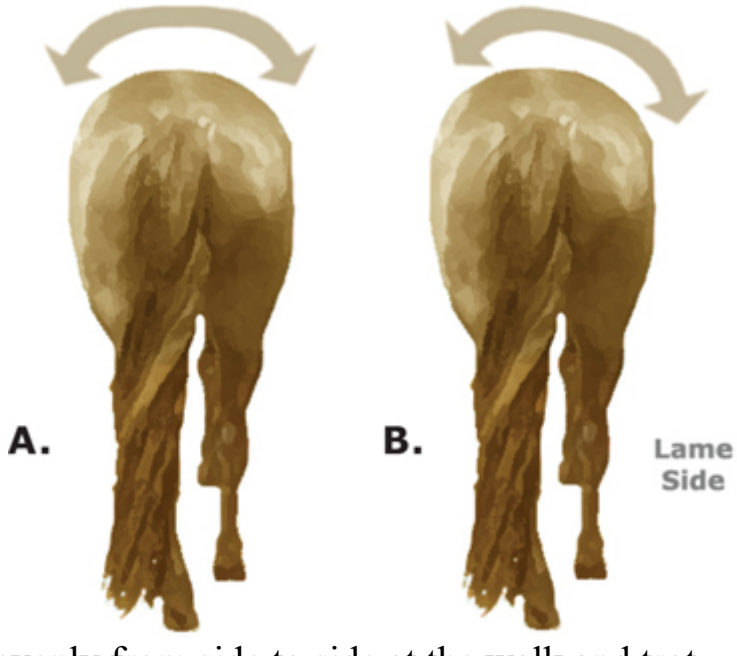
Since the pelvis functions as the base for the horse's hind limb activity, it also serves as our focal point when assessing the symmetry of oscillations between strides. Specifically, we should fixate on the highest point of the pelvis (known as the croup or *tuber sacrale*). In the sound horse, we are unable to discern a significant difference in the degree of excursion (up-and-down movement) of the pelvic median anatomy (the croup) between diagonal strides. Regular, asymmetric movement of the croup is observed in the horse displaying hind limb lameness,

however (VL 22d). If concurrent forelimb lameness is evident, there's a good chance that the two are physiologically related (one is causing the other).

VL 22d
Scan/Click to view video.
www.getsound.com/tutorials/22d

Asymmetry in median rotation. As the horse alternately engages diagonal pairs of limbs, the median anatomy rotates toward the side corresponding to the weight-bearing forelimb.[10] This means that the body regularly tips back and forth (from left to right and vice versa) as the horse trots along. In the sound horse, the degree of rotation is similar from one side to the other (fig. 22.4 A). On the other hand, a horse displaying limb lameness will usually bear more weight on (or "drop into") the comfortable side, often causing the trunk to tip farther toward the opposite (affected) side (fig. 22.4 B and VL 22e).

22.4 Rotation of the Pelvis at the Trot



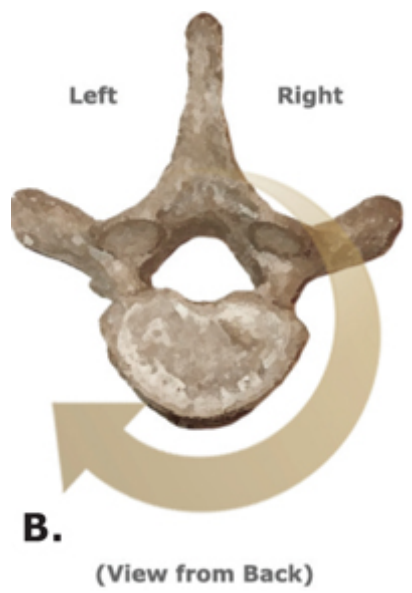
A. The sound horse vacillates evenly from side to side at the walk and trot.
B. The lame horse may tip farther toward the affected side at the walk and trot. In this instance, the spine is rotating toward the sound side (based on definition).

VL 22e
Scan/Click to view video.
www.getsound.com/tutorials/22e

Rotation and lateral flexion of the axial skeleton are closely linked and can often be discerned simultaneously by the acute observer. As the spine rotates toward the weight-bearing forelimb, concomitant flexion (or curving) will also progress toward the same side of the horse (fig. 22.5). In other words, the horse's spine simultaneously tips and swings back and forth at the trot.

22.5 Concomitant Rotation and Lateral Flexion of the Spine

A. The horse's axial skeleton flexes or bends to the left during the stance phase of the left forelimb.



B. The horse's axial skeleton rotates to the left during the stance phase of the left forelimb (see also fig. 14.6 c, p. 79).

Horses with unilateral (one-sided) limb lameness tend to bear more weight on the comfortable side and less weight on the affected (or painful) side. *This provokes greater rotation and flexion of the medial anatomy toward the sound side of the horse's body.* The discrepancy between the degree of axial leaning and lateral deviation from one side to the other can sometimes be appreciated by those observers who carefully monitor this activity. Visible asymmetry in median rotation is especially apparent with respect to the horse's pelvis. As the horse trots away from us, we watch the pelvis vacillate from side to side, assessing the degree of lateral roll along a transverse plane (see **VL 22e**, p. 148).

Horses with primary back pain will often try to avert tension (or stretching) of affected tissues (e.g. muscles, ligaments, and joint capsules), which they may manage by voluntarily electing to bear less weight on the forelimb along the opposite side. For instance, a horse with right thoracic facet arthritis may resist axial flexion to the left, an activity that would presumably accentuate tension of the affected joint capsule (and, therefore, any related pain). Accordingly, this horse might be motivated to underload the left forelimb, thereby curtailing the degree of lateral flexion to the left and abating discomfort associated with the right facet joint. In this instance, limb lameness may manifest as a secondary component to primary axial lameness (**VL 22f**).

VL 22f
Scan/Click to view video.
www.getsound.com/tutorials/22f

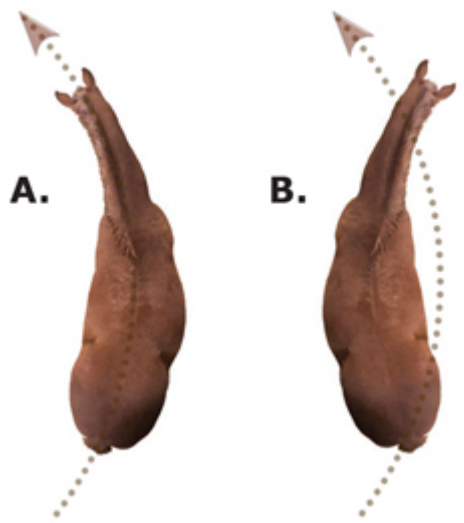
Asymmetry in median orientation. With respect to movement of the axial anatomy, we often refer to the three "Bs": *Bending, Banking,* and *Bearing.*

- **Bending:** Horses should bend their bodies to correlate with the directional path of their movement. Many horses will reshape their median anatomy to avoid pain or accommodate some form of pathology.
- **Banking:** In addition to bending their axial anatomy, horses will also *lean into* turns to help navigate the diversion. Normal horses will slant their bodies up to 15 degrees while turning sharply. Beyond that, horses will usually augment lateral flexion (bending) of their axial skeleton to accommodate the abrupt change in direction. However, if lateral bending induces pain or engages a biomechanical restriction, the horse may reconcile the situation by leaning excessively into the turn.
- **Bearing:** Unless asked otherwise by the rider, horses should always be pointed in the direction to which they are traveling (**VL 22g**).

VL 22g
Scan/Click to view video.
www.getsound.com/tutorials/22g

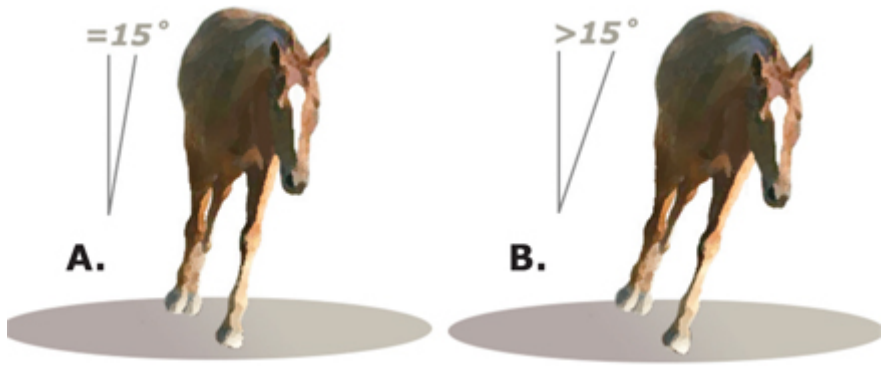
22.6 The Three "Bs" Pertaining to the Horse's Axial Orientation

Axial *Bending*



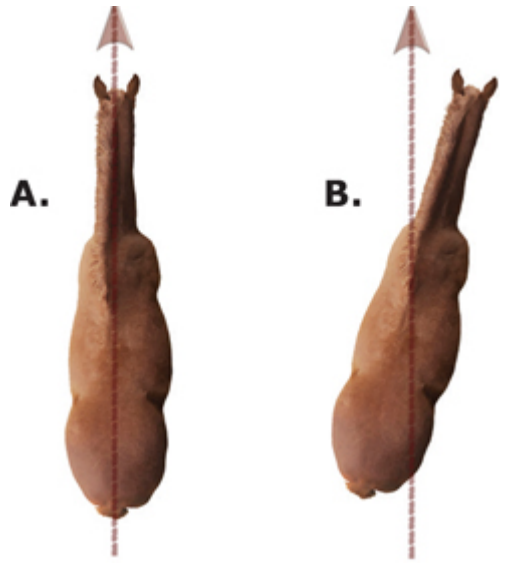
A. Normal: The horse's back should bend to correspond with the directional path of movement.
B. Abnormal: Horses often alter the way in which they bend to protect pathologic regions of their anatomy and/or avoid pain.

Axial *Banking*



A. Normal: Horses will normally lean to the inside of a circle or turn at an angle of approximately 15 degrees. Leaning occurs in combination with an appropriate degree of lateral flexion (or bending) of the back.
B. Abnormal: Horses that resist lateral flexion of the back (usually due to pain) will accommodate turns or circles by leaning excessively inward, often exceeding 15 degrees of angulation.

Axial *Bearing*



A. Normal: The horse's axial skeleton should remain aligned with the directional path of movement.
B. Abnormal: Horses often alter their axial orientation to protect pathologic regions of their anatomy and/or avoid pain.

Asymmetry in the Quality of Stride

As observers, we should pay extra close attention to the characteristics of the horse's stride. Differences in appearance between right and left limbs, and right and left diagonal pairs, are usually fairly conspicuous. Moreover, we can make effective use of these differences when establishing the nature and location of the horse's lameness.

Asymmetry in stride length. Aberrations in the horse's expected stride length can provide valuable insight into the nature of the lameness, especially if we can establish the phase of the stride that is most defective. For instance, the caudal (or weight-bearing) component of the stride will be visibly shortened in most cases of weight-bearing lameness (**VL 22h**). Contrarily, horses suffering from non weight-bearing pathology will often demonstrate insufficient cranial (or flight) stride length (**VL 22i**). As you might expect, both aspects of the stride will be influenced by combination problems (**VL 22j**).

VL 22h
Scan/Click to view video.
www.getsound.com/tutorials/22h

VL 22i
Scan/Click to view video.
www.getsound.com/tutorials/22i

VL 22j
Scan/Click to view video.
www.getsound.com/tutorials/22j

Asymmetry in stride timing. A shorter stride takes less time to execute and will accordingly appear to be hurried. Similarly, a hasty stride will not have the time to travel far enough to achieve normal length and will, therefore, appear to be abbreviated. Appropriately, the timing and length of the horse's stride are complementary. Evaluation of one feature allows us to skillfully estimate the other. Horses that display altered timing of stride will often appear to "skip" along at the trot, deserting the balanced tempo that is expected between diagonal strides at this gait (**VL 22k**).

VL 22k
Scan/Click to view video.
www.getsound.com/tutorials/22k

Asymmetry in stride metricity. Difference in stride height between left and right sides can be very evocative with regard to certain forms of pathology. A horse that is not raising the affected limb competently is demonstrating *hypometricity* (see fig. 14.10 B, and VL 14f, p. 82). In some cases, disproportionate toe wear is evident, implying that the horse is dragging the respective limb along rather than clearing the ground surface. Excessive elevation of the limb (i.e. picking it up too high) represents a *hypermetric* stride (see fig. 14.10 A and VL 14e, p. 82).

Asymmetry in Limb Gesturing

Each of the horse's limbs will follow a specific pattern of movement, both while in the air and on the ground. The particulars of these patterns enable us to accurately define the distinguishing features of the horse's gait. Once we establish these attributes, we can often implicate the underlying motive for the horse's movement.

Asymmetry in flight path of the limb. Rarely does the limb follow a straight line during protraction. Contralateral limbs that trail dissimilar flight paths likely do so for a reason. Winging-in, winging-out, plaiting, and circumduction are all terms used to describe altered progression of the limb during flight. It will behoove the observer to pay close attention to how each limb tracks (**VL 22l**).

VL 22l
Scan/Click to view video.
www.getsound.com/tutorials/22l

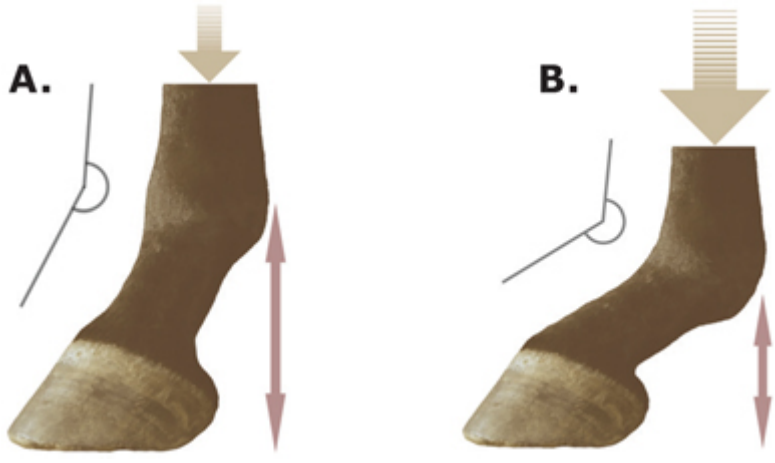
Asymmetry in limb conformation during stance. Horses suffering from pathology that alters function of the stay-apparatus may demonstrate abnormal limb configuration (such as hyperextension or hyperflexion) during the weight-bearing phase of the stride. Medial-to-lateral deviation of the loaded limb may also be indicative of explicit problems (**VL 22m**).

VL 22m
Scan/Click to view video.
www.getsound.com/tutorials/22m

Asymmetry in limb loading during stance. In some instances, the degree of fetlock extension can be used to appraise the magnitude of load-bearing experienced by the horse's weight-bearing limb. The horse is inclined to assign less weight-bearing load to the uncomfortable (or lame) limb, which consequently demonstrates reduced fetlock extension during stance (fig. 22.7 A). In compensation, additional weight applied to the comfortable (or sound) side generates increased downward force, which in turn, "pushes" the respective fetlock joint closer to the ground surface, thereby increasing its degree of extension (fig. 22.7 B). This action may be difficult to perceive, especially if the horse is moving swiftly (**VL 22n**).

We should note that a false impression of the horse's lameness may be procured by employing this visual technique, especially if there is structural compromise of the flexor and/or suspensory apparatuses. In these instances, increased fetlock drop occurs as a consequence of slackened support rather than limb overloading (see chapter 17, p. 106). Accordingly, the limb demonstrating increased "fetlock drop" in this scenario is actually the one that is impaired (**VL 22o**). Thus, this visual marker should not be employed as an exclusive means of assessing the degree of limb loading in the horse.

22.7 The Effect of Weight-Bearing Force on "Fetlock Drop"



A. The uncomfortable limb: The downward force applied to the uncomfortable (or lame) limb is tempered in an attempt to abate pain. As a consequence, the respective fetlock encounters less downward "push" from above. In this instance, you will observe minimal settling of the joint toward the ground surface.

B. The comfortable limb: Weight is transferred off the uncomfortable and onto the comfortable limb, effectively increasing the downward "push" encountered by the latter. The comfortable fetlock visibly sinks toward the ground surface, resulting in excessive extension of the joint.

VL 22n

Scan/Click to view video.

www.getsound.com/tutorials/22n

VL 22o

Scan/Click to view video.

www.getsound.com/tutorials/22o

Asymmetry in Foot Dynamics

A difference in the placement and action of the feet between right and left sides provides the observer with additional clues relating to the location and origin of the horse's lameness.

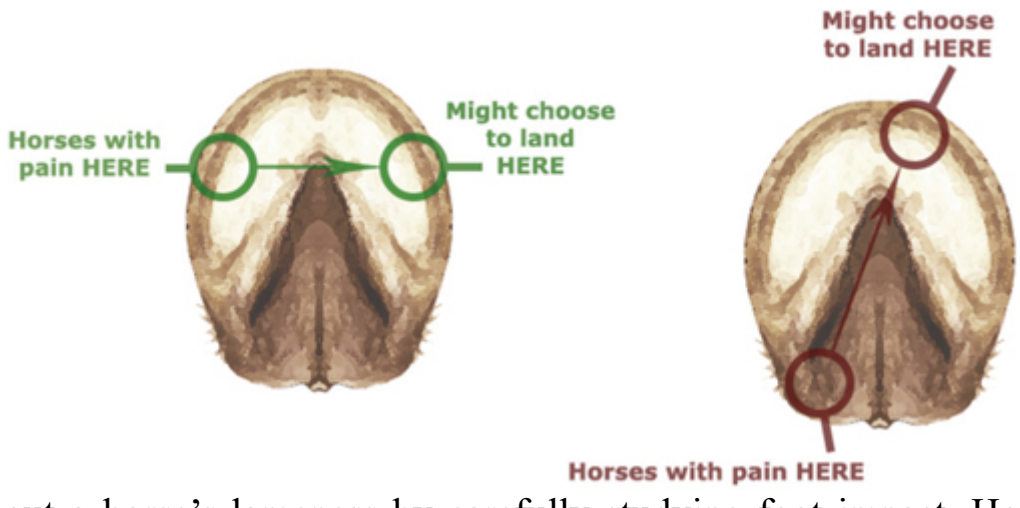
Asymmetry in foot placement. In general, the horse's feet should land relatively flat upon impact with the ground surface. Toe-first landings often occur in a horse experiencing heel pain, flexor tendonitis, suspensory desmitis, or negative third phalangeal (P3) angulation (**VL 22p**). In fact, we can usually surmise with confidence that a heel bruise/abscess or flexor and/or suspensory disease is *not* present in horses demonstrating a flat landing with the respective foot. Some horses experiencing pain and/or biomechanical interference associated with the stifle joint will also choose to land toe-first behind (**VL 22q**).

VL 22p
Scan/Click to view video.
www.getsound.com/tutorials/22p

VL 22q
Scan/Click to view video.
www.getsound.com/tutorials/22q

Close scrutiny of foot placement can be extremely helpful in estimating the approximate location of potential pain within the foot, as most animals will choose to strike the ground with the opposite side first (fig. 22.8). For example, a horse that initially contacts the ground with the toe may be attempting to circumvent pain emanating from the heel (**VL 22r**). Contrastingly, horses with tenderness along the toe of the foot (such as can occur in cases of laminitis) may elect to land heel-first (**VL 22s**).

22.8 Scrutinizing the Manner of Impact to Establish the Specific Site of Foot Pain



You can learn a lot about a horse's lameness by carefully studying foot impact. Horses will often elect to strike the ground surface with the most comfortable aspect of the foot first in an attempt to avoid pain. This area is invariably located directly opposite (diagonal) to the uncomfortable side of the foot.

VL 22r
Scan/Click to view video.
www.getsound.com/tutorials/22r

VL 22s
Scan/Click to view video.
www.getsound.com/tutorials/22s

Many animals may also land along the medial (inside) or lateral (outside) aspect of the foot first, depending on the location and quality of pain that they're trying to avert (**VL 22t**). A horse accommodating desmopathy of the lateral (outside) collateral ligament of the distal interphalangeal (or coffin) joint, for instance, may elect to contact the ground surface with the medial aspect (inside) of the foot foremost. In some cases, the observer can make highly explicit conclusions through fastidious inspection. For example, an outside (lateral) heel quarter landing may imply discomfort along the inside (medial) toe quarter of the foot (**VL 22u**).

VL 22t
Scan/Click to view video.
www.getsound.com/tutorials/22t

VL 22u
Scan/Click to view video.
www.getsound.com/tutorials/22u

In most cases, the angle of the horse's third phalanx (or coffin bone) should be slightly positive in relation to the ground surface. This means that the front of the bone should be slightly depressed (or lower) than the back of the bone as it rests within the hoof capsule. Horses with negative coffin bone (P3) angulation (in which the front of the bone is perversely elevated) will experience increased deep digital flexor tendon (DDFT) tension, which may prompt them to land toe-first upon impact (**VL 22v**). *Since the DDFT also contributes to the tertiary component of both fore and hind stay-apparatuses, negative P3 angulation can influence both the stability and action of these mechanisms (particularly in the hind limb).*

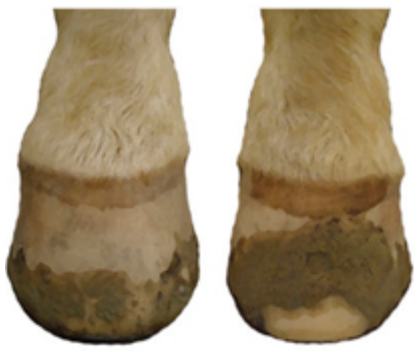
VL 22v
Scan/Click to view video.
www.getsound.com/tutorials/22v

Asymmetry in foot movement during stance. Medial (inward) or lateral (outward) rotation of the foot during stance often reflects distinct forms of pathology higher up in the horse's limb. As we learned in chapter 14 (p. 74), appendicular rotation actually transpires at the level of the shoulder in the forelimb and at the level of the hip in the hind limb. Horses experiencing pain associated with the lower hock joints (a condition known as *distal tarsitis*), for instance, will often medially rotate the limb and foot during the stance phase of the stride (see fig. 14.6 A, p. 79). Accordingly, the toe of the foot can be visually traced by the observer as it twists inwardly toward the horse's midline (**VL 22w**). This action often promotes excessive wear along the lateral aspect (or outside) of the foot, a side effect that is ordinarily discovered by the farrier. Lateral (or outward) rotation of the foot during stance is sometimes observed in cases of moderate to severe hip pain.

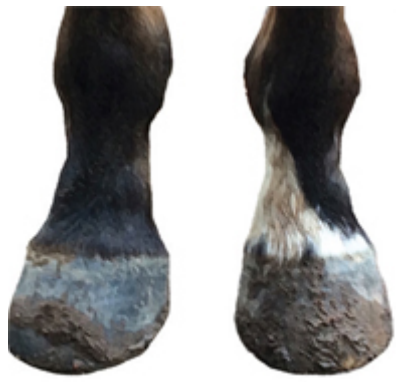
VL 22w
Scan/Click to view video.
www.getsound.com/tutorials/22w

The observer may also notice a difference in the way that the toe or heel of the foot sinks into softer footing (as opposed to remaining level) during the stance phase of the stride (figs. 22.9 A & B). Regular depression of the toe may evoke excessive abrasion and wear along this region of the sole or shoe and hint to potential discomfort affiliated with the heel of the foot or the flexor/suspensory apparatuses. This visual impression can sometimes be corroborated by examining the horse's footprints in wet or malleable footing (fig. 22.9C and **VL 22x**). Disproportionate erosion of the toe will prompt many farriers to check the respective P3 angles to confirm that the horse is not experiencing undue DDFT tension.

22.9 Static Markers for Aberrant Foot Dynamics

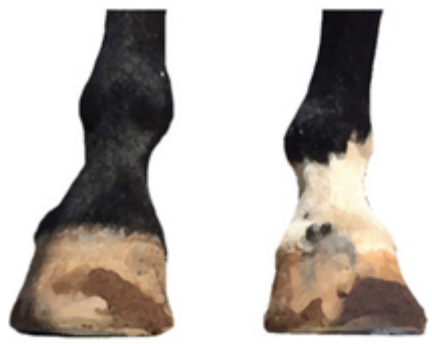


A. Excessive left hind toe-drop was observed in this horse while trotting in soft footing. Since the footing was wet at the time of evaluation, visual impressions could be confirmed by evaluating the static appearance of the feet immediately following examination. Radiographic examination (X-rays) of this horse revealed osteochondrosis within the left femoropatellar (stifle) joint (between the femur and knee cap); the appearance of the right stifle joint was normal.



B. A toe-first landing with subsequent toe-drop was apparent in the left hind during evaluation of this horse, which was also suffering from stifle joint pain. In this case, the lateral (outside) femorotibial joint (between the femur and tibia) harbored a subchondral cyst that generated the observed gait deficits. In an attempt to underload the lateral aspect of the stifle (during stance) and abbreviate flexion of the joint, this horse laterally rotated the left hind toe (outwardly) resulting in a medial (inside) foot breakover. He also abducted the limb during protraction to clear the ground surface and complete the stride.

C. It can be very helpful to assess the horse's footprints (when available). This image depicts footprints made by the horse's hind limbs; the closest print is of the right hind. You can deduce that the horse initially struck the footing with the lateral (outside) toe quarter of the foot, appropriately "splashing" sand in front and to the outside of the landing site. You can also see that the toes of the hind feet dropped into the footing during stance, creating an angled (toe-deep) print. Comparable footprints are often formed by horses experiencing lower hock pain (distal tarsitis).



D. Front feet of a horse suffering from moderate right carpal joint disease. With the assistance of "sticky sand," you are able to confirm that this horse is breaking over the outside of the left front foot and the inside of the right front foot. This discrepancy signifies asymmetric movement between the forelimbs. To avoid flexion of the right front limb (and pain associated with the respective carpus), this horse would rotate the foot laterally and break over the medial toe to enable (shoulder-generated) swinging of the limb outwardly.

VL 22x

Scan/Click to view video.

www.getsound.com/tutorials/22x

Excessive heel-drop into the footing during stance is a relatively rare occurrence, although this action can be a reflection of severe deep digital flexor tendon compromise in certain instances. Many farriers counter this activity via the application of a *bar shoe,* which furnishes extra heel support during stance (thereby mimicking the effect of a snowshoe). The opposing strategy is implemented in cases of *suspensory desmopathy,* in which the farrier might apply a *suspensory shoe* to encourage the heel to sink under weight-bearing load. This approach is intended to alleviate challenge to the suspensory apparatus via the transference of load-bearing tension to the flexor apparatus.

Asymmetry in foot breakover. Inspecting the breakover pattern of each of the horse's feet independently can be very constructive, especially if differences are noticed between contralateral limbs. Most horses will break over just to the outside of center (the dorsolateral toe quarter) in the fore and hind feet (see chapter 17, p. 106). In the author's experience, centrally oriented breakover (straight over the toe) is relatively uncommon in horses, even though it might seem the most natural path.

Some forms of pathology can prompt the horse to break over farther to the outside than expected, an action that is often accompanied by *toed-in* conformation. Horses that choose to break over the inside (medial aspect) of the foot may be attempting to avoid proper flexion and/or full engagement of the respective limb. In these instances, the horse may voluntarily rotate the foot laterally, break over the inside of the foot, and "swing" the limb to the outside so as to establish adequate clearance over the ground surface during protraction. This action is commonly observed in the forelimbs of horses with moderate to severe carpal (knee) joint disease (fig. 22.9 D and VL 22y). It may also be appreciated in the hind limbs of horses that cannot appropriately flex the stifle joint (due to interference of the stay-apparatus) or that resist flexion of the stifle joint (due to arthritic discomfort).

VL 22y
Scan/Click to view video.
www.getsound.com/tutorials/22y

SECTION VI

The Method of Visual Lameness Assessment

When my children were a little younger they would play the "20 Questions" game, a revived form of entertainment based on a television show from the fifties. The modernized version of this game amounted to a small, plastic, computerized ball that would ask a serious of "yes" or "no" questions with the intention of guessing an object in the mind of the player. The kids were invariably amazed with how this little plastic ball seemed to read their minds. In reality, however, it was a very calculated strategy: By simple process of elimination, the device would use a series of questions and answers to guide it to what seemed to be the inevitable correct match. Since each of the questions was structured to eliminate approximately half of the remaining possibilities, the computer was able to distinguish between over a million objects by the time the last query had been answered. Computer scientists perform comparable feats of analysis by employing a computation known as a binary search algorithm.

As observers, we apply a similar systematic strategy when evaluating gait deficits in the horse. During the course of assessment, we ask ourselves a series of visual questions and use a process of elimination to arrive at likely answers, which in our context provide insight into the nature, location, and possible cause of the horse's lameness (fig. VI.1). The queries are basically the same for all evaluations, with mild variations:

- Is the horse lame in the forelimbs?
- Is the horse lame in the hind limbs?
- Is the horse expressing axial lameness?
- What is the nature of lameness?
- What is the severity of lameness?
- What factors exacerbate the lameness?
- Does the gait have any distinctive features?

VI.1 Basic Lameness Evaluation Protocol

Primary Query	Secondary Queries	Possible Results
Is the animal lame?	• Is there an asymmetry in excursion between right and left sides? • Is there an asymmetry in movement between opposing diagonal pairs? • Is there altered movement, orientation, or carriage associated with the axial anatomy (head, neck, back)? • Are any of the limbs falling into an imaginary hole? • Are any of the limbs dragging an imaginary brick?	YES or NO
Which regions and/or limbs are affected?	• To which side (left or right) does the animal transfer weight? • To which end (front or back) does the animal transfer weight? • Why does the animal transfer weight (e.g. to underload a limb, maintain diagonal synchrony, or retain balance)?	LF, RF, LH, RH; multiple limbs; axial anatomy (head, neck, back)
What is the nature of lameness?	• How does the animal transfer weight (e.g. by dropping below the normal plane or by rising above the normal plane)? • Does the limb lameness have a *weight-bearing* component (i.e. is the comfortable limb dropping into a hole)? • Does the limb lameness have a *non weight-bearing* component (i.e. is the lame limb dragging a brick)? • Is the lameness purely *axial* in nature?	Weight-bearing (WB); non weight-bearing (NWB); combination (WB & NWB); axial
What is the severity or grade of lameness?	• How much abnormal movement/median excursion is demonstrated? • *Weight-bearing* component: How deep is the hole into which the comfortable limb drops? • *Non weight-bearing* component: How heavy is the brick attached to the lame limb? • *Axial* component: What degree of median deviation (from normal) is evident?	0, 1, 2, 3, 4, 5

Which gaits are affected?		Walk; trot; pace; canter; gallop; rack; multiple gaits; other
What is the consistency of the lameness?	• Is lameness evident during every stride? • Is lameness evident on a regular basis (from one day to the next)? • Is lameness evident in every environmental circumstance?	Consistent; intermittent; infrequent/occasional
What factors exacerbate the lameness?	Surface Influences	Hard; soft; congruency
	Directional Influences	Straight line; turning right; turning left
	Grade Influences	Flat surface; going downhill; going uphill
	Other Influences	Obstacles; under saddle; temperature; mosture/humidity; miscellaneous
Are any distinguishable traits displayed?	• Is there a characteristic limb appearance during flight? • Is there a characteristic limb appearance during stance? • Is there a characteristic carriage of the axial anatomy?	Goose stepping of the hind limb, etc.

Although a veterinary degree is required to perform diagnostic imaging and local anesthesia ("blocks"), a degree certainly *isn't* required to formulate an opinion as to the possible source(s) of a horse's lameness based on visual perception. All of us assuredly have access to the optimum diagnostic imaging tool for this exercise: *our eyes.*

The following chapters will take us through the various steps comprising an effective visual assessment of the horse in motion. We can simplify the process immensely by interpreting one aspect of the horse's gait at a time. Although it may take some time to navigate through the material, the actual process takes the seasoned observer less than 10 minutes to complete. Abnormalities usually become very obvious once we know where, when, and how to look for them.

The average horse owner may only care to know if her horse is sound or lame. She might not be interested in the specifics of the problem. The serious upper-level competitor, on the other hand, might want to know a little more with regard to the severity and nature of the horse's pathology, thereby providing the means for quicker and better management decisions. In both instances, the process of visual assessment is fundamentally the same.

The most basic lameness assessment would entail identifying the location, nature, and severity of asymmetric movement. At the minimum, these three characteristics should be highlighted in the verbal or written characterization of any equine lameness, for example: *grade 3/5 right front weight-bearing lameness.* These facets of our assessment will be discussed in the first three chapters of this section.

The last three chapters of this section are directed toward those enthusiasts seeking to further interpret gait deficits with the intention of postulating probable causes. For all of us, it is not only important that we listen closely to our subjects, but that we listen with our *eyes* instead of our ears.

23
Identifying the Affected Area(s)

Foremost, we should decide if the horse is lame. This is most easily accomplished by comparing the general movement between diagonal pairs of limbs at the walk and trot. One pair should visually mirror the other in the sound horse: LH–RF action will mimic RH–LF action (see **VL 3a**, p. 22). Similar motion should be perceived in the pairs of limbs as well as both sides of the horse's head, neck, body, and pelvis (which comprise the axial skeleton). Dissimilar movement between dichotomized halves of the horse is the most conspicuous indicator of lameness (see **VL 3b**, p. 22).

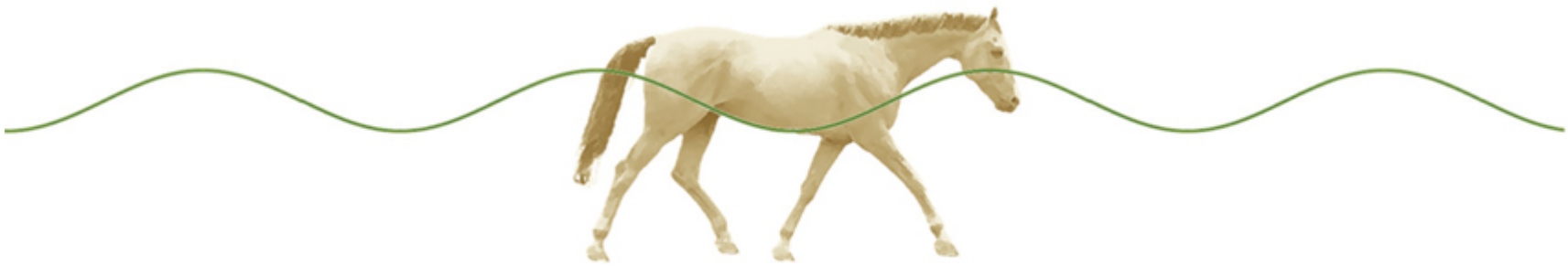
Identifying the Lame Limb(s)

Once we confirm that one diagonal looks different than the other, we should then survey the physical oscillations of the horse's body relative to the position and activity of the limbs comprising each pair. *It is much easier for our eyes to perceive the horse "falling into" the comfortable side as opposed to favoring the uncomfortable side.* The popular "down-on-sound" phrase commonly used to delineate the appearance of lameness in the horse typifies this concept (**VL 23a**). We should note, however, that downward movement toward the "sound" side of the horse does not just refer to the head and neck. In many cases of forelimb lameness, for example, the obvious "head bob" is not perceivable. Our ability to detect depression (or dipping) of the horse's overall body structure (rather than a specific segment) dramatically enhances our ability to depict even marginal lameness. This requires that we first assess the general progression of the horse's body through space.

VL 23a
Scan/Click to view video.
www.getsound.com/tutorials/23a

The horse's body will move up and down (oscillate) with each trot step. The vertical distance of this movement (often labeled *excursion*) can be visually approximated by the observer. In the sound horse, the degree of excursion is similar between the right and left sides (fig. 23.1 A). By contrast, increased skeletal excursion is noted during every other stride in the lame horse (fig. 23.1 B).

23.1 Excursion of the Horse's Body at the Trot



A. The horse's frame moves up and down (oscillates) with each trot step. In the sound horse, the amplitude of oscillation (excursion) is similar between right/left sides and right/left diagonal pairs.

B. A visible discrepancy in the degree of excursion between the horse's right/left sides or right/left diagonal pairs at the trot is an indication of lameness.

Since the forelimbs and hind limbs provide dissimilar physiologic functions for the horse, each end will respond differently while under the influence of musculoskeletal pathology. In most disciplines (reining not included), the forelimbs function to steer and brake the horse, whereas the hind limbs are used principally for propulsion. The appearance of lameness associated with each end of the horse differs as a consequence of these disparate roles. We will, therefore, describe the visual features of fore and hind lameness separately.

Detecting Forelimb Lameness

The horse's head, neck, and thorax begin to descend each time one of the forefeet impacts the ground surface and reaches the lowest point at or near mid-stance. Just beyond mid-stance these structures begin to rise and eventually reach their highest point at or near lift-off of the foot.[12] The upward and downward movement of the horse's front end generates regular oscillations that have similar timing and intensity between diagonal pairs in the sound horse.

Forelimb lameness that manifests every other stride disrupts the regular symmetric movement of the horse's head, neck, and thorax. Disruption is regular and peaks at the instant the insult (e.g. biomechanical restriction or pain) is most intense.[13] Descent of the head, neck, and thorax is regulated during the lame limb's stance phase so as to reduce loading. The weight is transferred to the contralateral (or compensating) limb, which displays increased head/neck/thorax descent during its respective stance phase (**VL 23b**). The accentuated drop of the front end (into the comfortable limb) is highlighted in cases of weight-bearing lameness and gives the appearance of the horse *stepping into a hole.* The front of the horse moves down less during the stance phase of the lame limb and up less after the stance phase of the lame limb when maximum pain is experienced at hoof impact or within the first half of the stride's stance phase.[14]

VL 23b
Scan/Click to view video.
www.getsound.com/tutorials/23b

As the contralateral or compensating limb pushes off, the horse's head and neck swing upward, thereby increasing the downward force of the hoof against the ground surface (**VL 23c**). This action helps to lift the trunk as well as facilitate protraction of the affected forelimb. This visible element is highlighted in cases of non weight-bearing lameness and resembles the horse's attempt to *drag weight with the lame limb.* The front end of the horse moves up more following the stance phase of the lame limb when maximum pain is experienced during the second half of the stride's stance phase.[14]

VL 23c
Scan/Click to view video.
www.getsound.com/tutorials/23c

As a consequence of forelimb lameness, movement of the horse's trunk is visibly disparate between the two diagonals. The horse's head, neck, and withers are highest at the moment that the lame limb comes into contact with the ground surface. By supporting the frame in a higher position at the beginning of the lame diagonal stance phase, the horse is able to prevent the accumulation of downward momentum.[12] This results in shortening of the stride, a feature that can be visibly appreciated during most lameness assessments.

Evaluation of the horse's forelimbs requires that we visibly discern asymmetric oscillation between diagonals to determine which limb is lame. Does the front of the horse (comprising the head, neck, chest, and/or withers) drop excessively when one fore-limb contacts the ground surface? If so, the opposite forelimb is lame. For instance, the horse "falling into" the right forelimb would signify lameness in the left forelimb (see **VL 3b**, p. 22).

Detecting Hind-Limb Lameness

We use the same observation techniques when assessing the horse's hind end, which is performed subsequently and separately from the front end. We monitor oscillations between up-and-down movement of the horse's croup (pelvis) to determine if asymmetry between diagonals can be detected. Since we're assessing a single region that is intimately "tied into" the horse's axial skeleton, hind limb lameness may be more challenging to perceive with the naked eye. Even so, careful observation will usually reveal asynchronous and/or asymmetric movement.

While trotting, a sound horse's pelvis generates regular oscillations similar to those produced in the front end. The pelvis begins to descend just before each hind foot strikes the ground surface, reaches its lowest point at or near mid-stance, rises during the second half of the stance phase, and eventually reaches its highest point after push off.[12]

Consonant with the front end, regular disruption of up-and-down pelvic movement signifies hind limb lameness. Disruption peaks at the moment of maximum insult (e.g. pain). Although the horse can use the head and neck to counter-lever off the back end to some degree (manifesting in referred forelimb lameness), most weight transfer behind occurs via relative engagement of the hind extensor musculature.[13] When the insult is most intense during the first half of stance, ground reaction force experienced by the lame limb can be reduced by increasing extensor muscle activity. This action prevents descent of the pelvis on the lame side of the horse. Disproportionate pelvic dropping and tilting that occurs during the stance phase of the compensating (comfortable) stride affords the appearance of that limb *sinking into a hole.* This movement is highlighted in cases of weight-bearing hind limb lameness (**VL 23d**).

VL 23d
Scan/Click to view video.
www.getsound.com/tutorials/23d

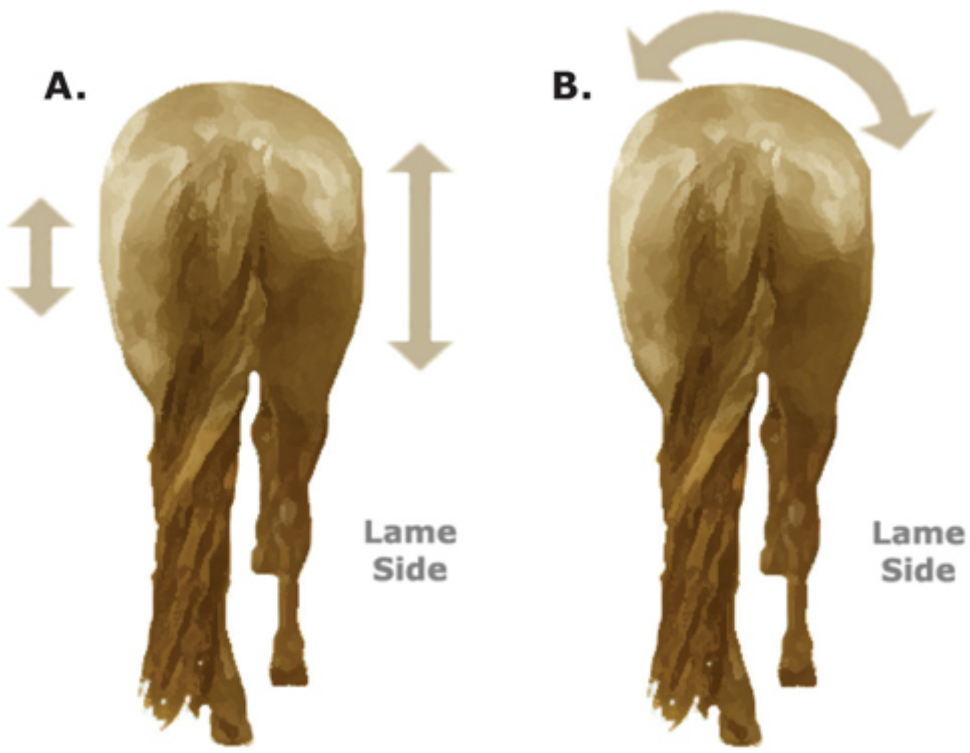
Interestingly, ground reaction forces are most effectively reduced during the second half of stance via extensor muscle *disengagement.* In this case, the pelvis pushes less as the affected limb takes off, giving the impression that the limb is being *dragged along* by the horse's body. This action is highlighted in cases of non weight-bearing hind limb lameness (**VL 23e**).

VL 23e
Scan/Click to view video.
www.getsound.com/tutorials/23e

When assessing hind limb lameness, some observers focus on the degree of vertical pelvic drop below the horse's normal or expected plane of descent. Others fixate on the extent of pelvic rotation (or "tipping") toward the lame side of the horse. For the purpose of accurate assessment, it is probably wisest to evaluate both parameters:

- Is the overall up-and-down movement of the pelvis consistent between right and left strides? If not, more pelvic excursion will be visible on the lame side of the horse (fig. 23.2 A and see **VL 23d**).
- Does the pelvis tilt more toward one side (as opposed to the other) in the course of every other stride? If so, the pelvis will tip toward the lame side of the horse (fig. 23.2 B and see **VL 22e**, p. 148).

23.2 Asymmetric Excursion and Rotation of the Pelvis Amid Hind Limb Lameness



A. The horse's pelvis will display greater excursion (up-and-down motion) along the side of the affected (lame) hind limb.
B. The horse's pelvis will display greater rotation (slanting) toward the side of the affected (lame) hind limb.

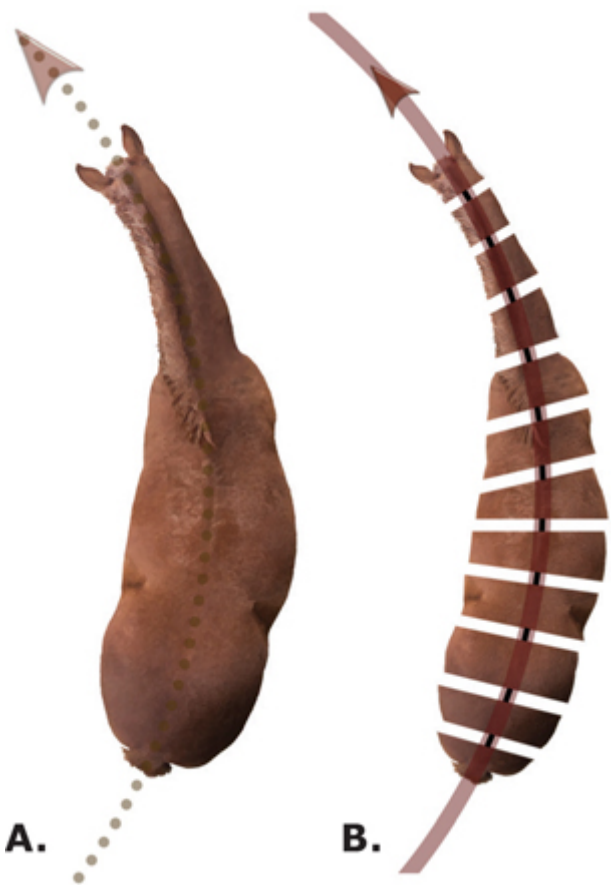
Excessive pelvic rotation toward one side of the horse is often denoted as a "hip hike," "pelvic hike," or "gluteal rise" and can be artificially generated in horses with preexisting asymmetric pelvic anatomy (in which one side of the pelvis passively rests lower than the other). In these cases, the sunken side will usually display an increase in vertical movement, thereby mimicking rotational asymmetry and imparting the false impression that the horse is lame behind. Appropriately, horses with anatomic asymmetry that display increased pelvic rotation but not an increase in overall vertical movement of the pelvis may actually be sound. It is, therefore, imperative that we assess both components of pelvic movement, particularly in horses that are physically "lopsided" behind. Any structural imbalances associated with the horse's pelvis should be noted and recorded prior to lameness assessment so that aberrant movement(s) can be judged fairly.

Detecting Axial Lameness

Revised activity of the median anatomy is an expected consequence of disproportionate delegation of weight and balance among the limbs in the lame horse. Therefore, we will usually observe concurrent axial lameness in limb-lame horses (particularly when the primary issue is behind). As effective observers, it is integral that we criticize **median movement** in this context. Dynamic evaluation of the horse's neck and back is, therefore, most appropriately performed *after* our assessment of the *appendicular skeleton.* Only in cases in which the horse is sound in the limbs should our sole diagnostic and therapeutic attention be directed at the median anatomy.

Carriage and movement of the horse's head, neck, and back should mimic that of a series of train cars traveling along a railway: These structures should align, bend, and straighten in accordance with the directional path upon which they're being led (fig. 23.3).[15] Abnormalities are most easily depicted when the horse deviates from the expected track, alters body angle inwardly or outwardly, or bends in a way not consistent with the contour of the (imaginary) railway (fig. 23.4).

23.3 Demonstration of Proper Axial Orientation on the Longe Line



A. The horse's median anatomy should bend in a way that is commensurate with the circumferential path of the longeing circle.
B. Technically speaking, each segment of the horse's axial skeleton should conform to its respective slice of the longeing circle, similar to the way a series of train cars travels along a railway.

23.4 Demonstration of Improper Axial Orientation on the Longe Line

A. In accordance with the rest of the horse's median anatomy, the neck should conform to the section of the longeing circle upon which it advances. Horses that bend the neck excessively inward (while refusing to tip the head) may be protecting an issue between the first and third cervical vertebral levels (C1-C3).

B. Most horses will tip the head slightly in response to tension applied at the attachment of the longe line. Excessive tipping of the head is sometimes displayed in horses that are evading appropriate flexion of the neck. This symptom may imply pathology between the fourth and seventh cervical vertebral levels (C4-C7).

C. Similar to a motorcycle navigating a corner, the horse will lean the body toward the inside of a longeing circle to counteract the centripetal effects, which "push" the body in the opposite direction (toward the outside of the circle). Sound horses often lean at an angle of up to 15 degrees depending on the size of the circle. When longed in tighter circles, the horse will bend or curve the body more to allow for consistent tracking along the shorter circumference. At some point during concentric longeing, the back-sore horse will cease to bend further and start to lean inwardly excessively. This response may suggest pain along the thoracic (T1-T16) region of the back.

D. The curvature of the horse's neck and back should match that of the circle along which they're being longed. *Counterbending* is often observed in horses attempting to protect the back (particularly the side corresponding to the outer aspect of the circle). Some of these horses will concurrently alter their body angle to face inwardly (*drift*), suggesting the possible presence of pain along the lower back (thoracolumbar) region.

Primary back pain can also be appraised via the study of hind limb position and impulse. Serving as the horse's engine, the hind limbs must be adequately engaged to provide ample drive and power (see chapter 18, p. 114). This requires appropriate positioning up and under the body, where the hind limbs can assume enough of the horse's weight to generate forward impetus (momentum). Productive hind limb positioning is achieved through proper orientation (ventroflexion) of the horse's lower back and lumbosacral (LS) joint. Horses unable to attain this axial posture (for whatever reason) may not be able to afford favorable positioning of the hind limbs, thereby attenuating the ability of the latter to drive the horse onward. As a consequence, the horse will be forced to "pull itself around" with the forelimbs (which are usually designated for steering and braking in most disciplines). This manifestation of lameness is fairly obvious to the observer who can watch the horse's pelvis passively "float over" each hind limb during its respective weight-bearing phase. In this instance, the hind limbs behave more like passive pillars as opposed to working parts within a power-generating motor. Comparable action is often observed in horses trotting on a flat, mechanized treadmill, during which the ground surface is actively traveling beneath them. Since propulsive forces are chiefly assigned to the treadmill, the horse merely has to move its limbs so as to resist faltering. It is important to remember that a lack of hind limb engagement could be due to factors other than axial pathology, not excluding hind limb pain, neuropathy, myopathy, biomechanical interference, and general weakness.

Detecting Complicated Lameness

In many instances, lameness will be apparent in more than one end (both the front and back) and/or more than one side (both the right and left) of the horse. It is important to remember that gait abnormalities associated with multiple limbs frequently possess a physiologic relationship with one another. Our capacity to link coexisting deficits will be easier after we have established the severity and nature of each independently.

In the case of multifactorial lameness, a relationship between two or more abnormal patterns of movement cannot be contrived. Horses with discrete issues affecting both limbs of a single diagonal pair are particularly challenged at the trot, since neither limb can unconditionally compensate for the other (see **VL 5c**, p. 27). Under these circumstances, our ability to visually isolate each limb's irregular movement (independently from one another) becomes critical to the success of our assessment (see chapter 28, p. 202).

Breed Considerations

Observing horses at the basic trot is preferable due to the symmetric movement affiliated with this gait. Assessment of gaited horses performing at the rack and slow gait is considerably more challenging because movement is displayed in a four-beat rather than a two-beat rhythm (**VL 23f**). This means that each of the four feet strikes the ground at a different time and that the horse's weight is balanced among individual limbs as opposed to diagonal pairs throughout a complete stride.

VL 23f
Scan/Click to view video.
www.getsound.com/tutorials/23f

The fundamental principles of lameness identification still apply, although visual markers may be more challenging to detect in the gaited athlete. The effective observer should possess baseline knowledge of the subject's *normal* movement patterns (both from individual and breed standpoints) so that any alteration(s) can be analyzed appropriately (fig. 23.5). Preemptively cataloging baseline video footage (which characterizes normal or expected movement) is the easiest way to satisfy this obligation.

23.5 Basic Gaits of the Horse

Natural Gaits	
Walk	A slow, flat-footed natural four-beat **lateral gait**.
Trot	A two-beat **diagonal gait** that varies in speed.
Canter	A controlled three-beat diagonal gait with periods of suspension (during which time no feet are in contact with the ground surface).
Gallop	A fast three-beat gait; the first beat is made by the trailing hind foot followed by the leading hind foot with its diagonal fore foot, succeeded by the remaining fore foot.
Artificial, Manufactured or Ambling Gaits	
Amble	A four-beat lateral gait similar to (but slower than) the rack; also known as the slow gait or single foot.
Counter-Canter	A canter in which the horse deliberately takes the outside lead.
Extended Gait	Strides occur at the same rate but are lengthened, thereby enabling the horse to increase velocity without altering tempo.
Fox Trot	A four-beat diagonal broken trot. The horse appears to simultaneously walk with the forelimbs and trot with the hind limbs.
Hauchano	A two-beat lateral gait (pace) demonstrated by the Peruvian Paso horse.
Pace	A two-beat lateral gait.
Paso Corto	A four-beat lateral (and extended) gait that is slightly faster than the Paso Fino gait.
Paso de Andatura	A high-stepping four-beat diagonal gait demonstrated by the Andalusian horse; also known as the Spanish walk.
Paso Fino	The slowest gait of the Paso Fino horse; a four-beat lateral (and highly-collected) gait also known as the fine walk.
Paso Largo	One of the faster gaits of the Paso Fino horse; a four-beat lateral gait.
Paso Llano	The slowest gait of the Peruvian Paso horse; a four-beat (broken) lateral gait during which the hind limb moves prior to the complementing forelimb.
Rack	A fast four-beat lateral gait demonstrated by the five-gaited Saddlebred horse.
Running Walk	A four-beat lateral gait demonstrated by the Tennessee Walking Horse and related breeds.
Slow Gait	A four-beat lateral gait similar to (but slower than) the rack; also known as the amble or single foot.
Sobreanado	A fast broken two-beat lateral gait (pace) demonstrated by the Peruvian Paso horse.
Termino	A desirable action in the Peruvian Paso horse; outward swinging of the forelimb that emanates from the shoulder joint.
Tolt	A running walk demonstrated by the Icelandic Horse; similar to the rack.
Abnormal or Inappropriate Gaits	
Cross-Canter	The horse simultaneously assumes one lead up front and the opposite lead behind.
Run	A fast (three-beat) gallop evolving into a four-beat gait as the diagonal pair becomes dissociated; the hind foot strikes the ground before its complementary forefoot.

Trocha	An overly-swift gait demonstrated by the Paso Fino horse; a two-beat lateral gait identical to the pace.
Trocha y Galope	A broken gait performed by the Paso Fino horse in which the pace, canter, and gallop are commingled.

24
Determining the Nature of Lameness

In addition to identifying the affected limb(s), the observer should attempt to determine exactly *how* the horse is limping. *Designating the nature of the horse's lameness is vital to isolating the locality and functionality of its cause.* Fortunately, this is a relatively simple assignment. All lame horses display one of the following three gait attributes:

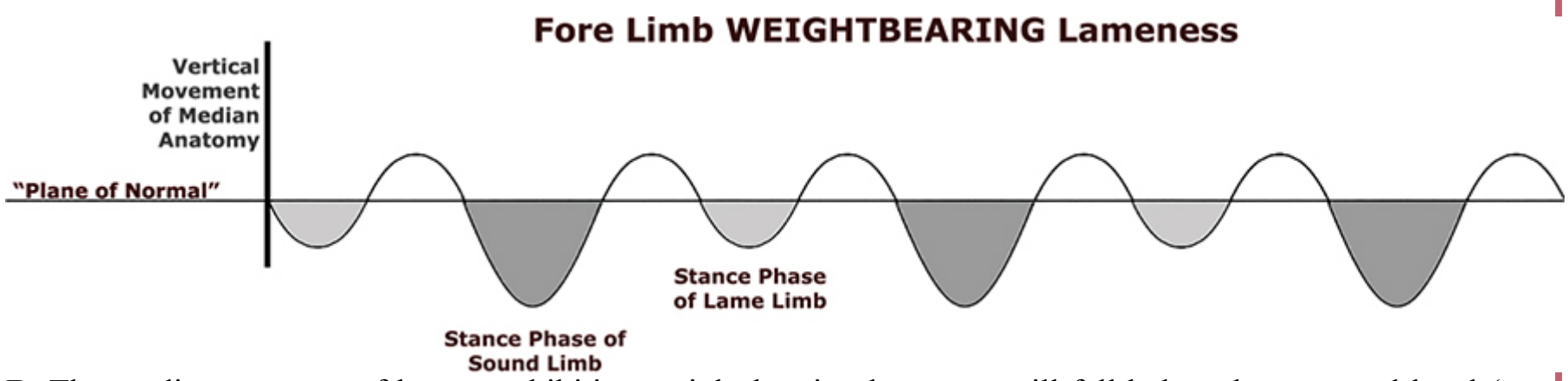
- Weight-bearing lameness
- Non weight-bearing lameness
- Combination lameness (which comprises both weight-bearing and non weight-bearing components)

Weight-Bearing Lameness

This form of lameness (often denoted as *impact lameness*) is a visible expression of the horse's attempt to reduce the load encountered by the affected limb, which experiences maximum insult during the stance (or load-bearing) phase of the stride. Appropriately, the horse distributes less weight to the affected side, thereby limiting the visible drop displayed by the median anatomy (e.g. head, neck, back, and/or pelvis) while the lame limb is in contact with the ground surface. This load is delegated to the contralateral (comfortable) limb, which then experiences additional burden during its corresponding stance phase. The observer can witness the horse's median anatomy plunge below the expected level of descent as the sound limb begins to bear weight (figs. 24.1 A & B).

24.1 General Appearance of Weight-Bearing Lameness in the Horse

A. Weight-bearing lameness manifests as the horse transfers weight from the affected side to the sound side of the body. Weight is transferred through the horse's median anatomy, which sinks dramatically while the comfortable limb is in contact with the ground surface.



B. The median anatomy of horses exhibiting weight-bearing lameness will fall below the expected level (or plane) during stance of the unaffected (sound) limb.

C. The horse visibly "falls into" the sound side, affording the appearance that he/she is stepping into a hole with the sound foot.

D. The exclusive demonstration of weight-bearing deficits suggests that the source of the horse's lameness is within the distal limb (below the level of the fetlock joint).

The head, neck, and withers serve as powerful visual markers for this action in cases of forelimb weight-bearing lameness. The pelvis, which demonstrates excessive excursion and rotation during every other stride, is most easily assessed in the hind limbs. In both cases, median activity renders the appearance of the horse stepping into a hole with the sound foot (figs. 24.1 C & D and **VL 24a**). *You could self-impose this gait deficit by placing a small rock in one of your shoes.*

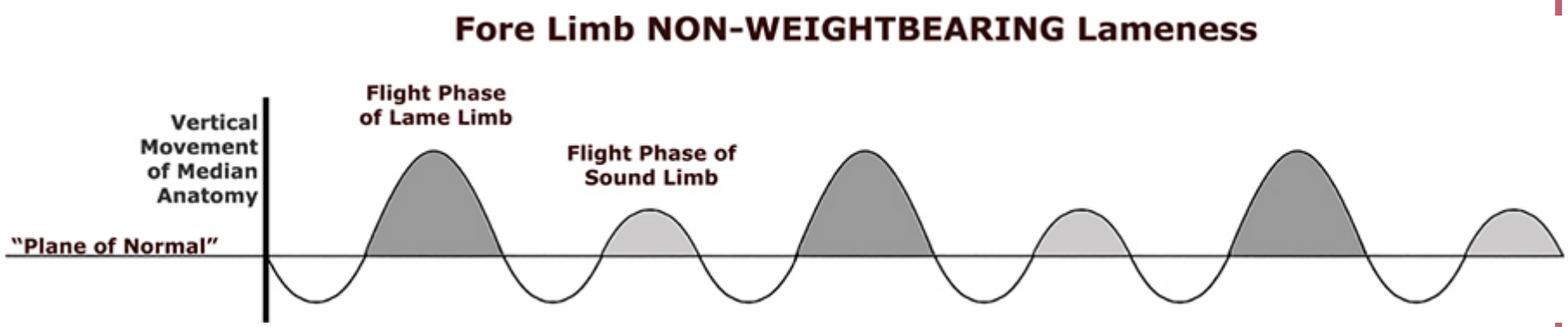
VL 24a
Scan/Click to view video.
www.getsound.com/tutorials/24a

Non Weight-Bearing Lameness

This form of lameness (often denoted as *push-off lameness*) is a visible expression of the horse's attempt to protract the affected limb, which experiences maximum insult during the flight (or non load-bearing) phase of the stride. In this instance, **ascending movement** of the median anatomy is exaggerated as the horse struggles to advance the affected limb in a forward direction; axial structures will rise above the expected level of ascent (figs. 24.2 A & B). In the forelimb, the head and neck are often exploited to assist in this effort. Elevation of the horse's pelvis is less conspicuous than that of the head and neck and may be more difficult to perceive when assessing hind limb lameness, although it does occur. In both cases, the horse appears as though it is dragging a weighted object (such as a brick) with the affected limb (figs. 24.2 C & D and **VL 24b**). *You could self-impose this gait deficit by applying a stiff brace over one of your knees.*

24.2 General Appearance of Non Weight-Bearing Lameness

A. Non weight-bearing lameness arises as the horse attempts to facilitate protraction (advancement) of the affected limb. The median structures (most notably the head and neck) are solicited in this effort and rise excessively in an attempt to assist in forward "swinging" of the affected limb.



B. The median anatomy of horses exhibiting non weight-bearing lameness will rise above the expected level (or plane) during protraction of the affected limb.

C. The employment of median anatomy to incite limb protraction renders the appearance of the horse dragging a brick with the affected limb.

D. The exclusive demonstration of non weight-bearing deficits suggests that the source of the horse's lameness is within the upper limb (above the level of the carpus or tarsus in the fore or hind limbs, respectively).

VL 24b

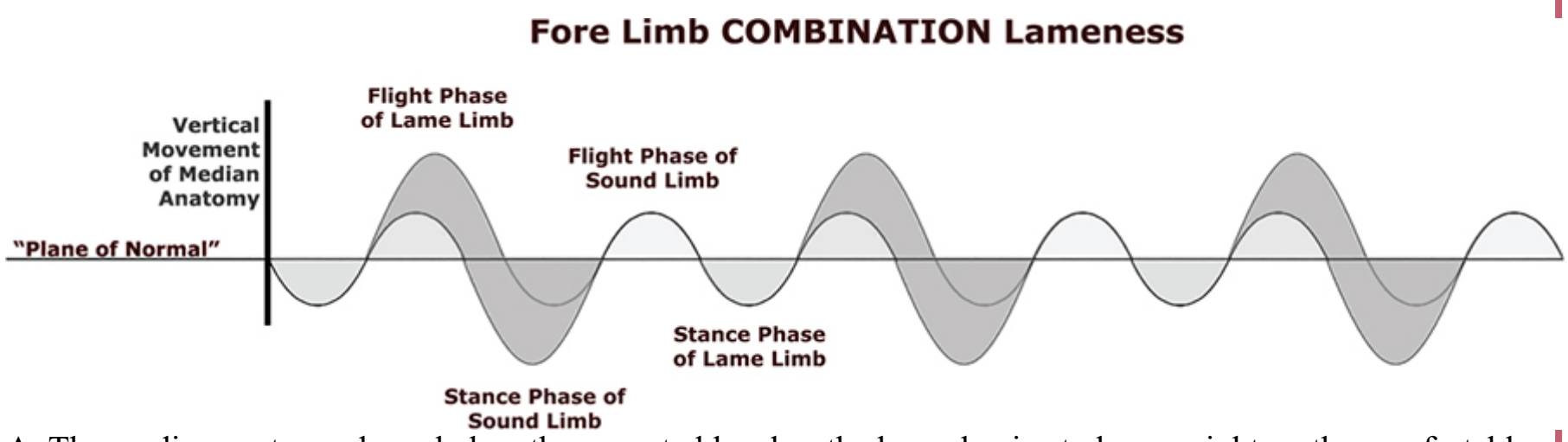
Scan/Click to view video.

www.getsound.com/tutorials/24b

Combination Lameness

Gait abnormalities that are displayed during both the flight and stance phases of the stride are reflected in cases of combination lameness. This form of asymmetry occurs as the horse attempts to transfer load to the comfortable limb *and* facilitate protraction (advancement) of the affected limb. Accordingly, combination lameness looks like the horse is *dragging a brick* with the affected limb in addition to *stepping into a hole* with the contralateral (comfortable) limb (figs. 24.3 A–C and **VL 24c**). *You could self-impose this gait deficit by placing a small rock in one of your shoes and applying a stiff brace over the knee of the same limb.*

24.3 General Appearance of Combination Lameness



A. The median anatomy drops below the expected level as the horse begins to bear weight on the comfortable limb and rises above the expected plane as the horse begins to advance the affected limb. As a consequence of both of these activities, the tempo (or timing) of overall body movement will vary between diagonal sides.

B. The employment of median anatomy to both transfer weight and incite limb protraction renders the appearance of the horse falling into a hole with the comfortable limb while simultaneously dragging a brick with the affected limb.

C. The demonstration of combination-gait deficits suggests that the source of the horse's lameness is within the mid-limb (above the level of the fetlock joint and below the carpus or tarsus).

VL 24c
Scan/Click to view video.
www.getsound.com/tutorials/24c

It is important to remember that a shortened stride is featured in almost all forms of lameness. The aspect of the stride that is most influenced, however, will depend on the nature of the horse's lameness:

- The caudal, posterior, or backward phase of the stride will be shortened in the case of weight-bearing lameness (fig. 24.4 A and see **VL 22h**, p. 150).
- The cranial, anterior, or forward phase of the stride will be abbreviated in the case of non weight-bearing lameness (fig. 24.4 B and see **VL 22i**, p. 150).
- Both cranial and caudal phases of the stride will be shortened in the case of combination lameness (fig. 24.4 C and see **VL 22j**, p. 150).

24.4 The Effect of Weight-Bearing, Non Weight-Bearing, and Combination Lameness on Stride Length

A. Weight-Bearing Lameness

A. Weight-bearing lameness is typically characterized by a shortened caudal (or backward) component of the stride.

B. Non Weight-Bearing Lameness

B. Non weight-bearing lameness is typically characterized by a shortened cranial (or forward) component of the stride.

C. Combination Lameness

C. Both forward and backward components of the stride are abbreviated in cases of combination lameness.

Highlighting the Nature of Lameness During Assessment

Since dissimilar forms of lameness boast distinct physiologic traits, each will respond differently to extrinsic (environmental) influences. It behooves the observer, therefore, to manipulate the horse's evaluation setting whenever possible. Employing this strategy can clarify our assessment immensely, particularly with regard to identifying the nature of lameness.

Directional influences. Horses lean into turns in an attempt to counter some of the centripetal forces encountered by the body during directional changes. During this maneuver, the body's center of gravity migrates farther toward the inside of the turn (fig. 24.5 A). Accordingly, the weight-bearing load encountered by the limbs along the inside of the horse's body is augmented, whereas the outer limbs endure less of the load. Since weight-bearing pathology is directly challenged under load, we would expect corresponding lameness to become more pronounced when the affected limb is along the inside of a turn or circle (fig. 24.5 B and **VL 24d**).

A horse that steps into an "imaginary hole" with one foot will tend to:

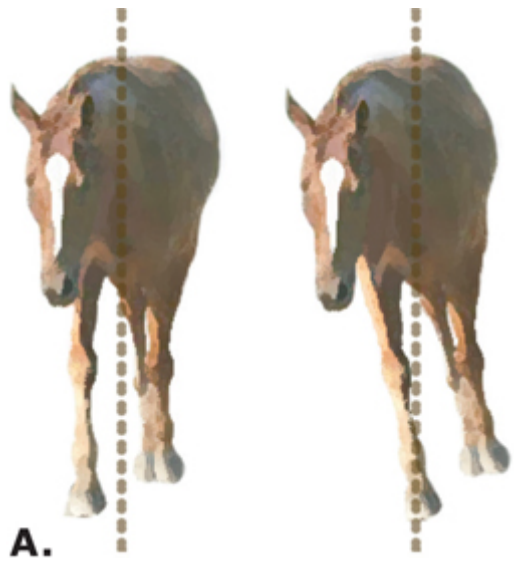
- *Bear weight on the other (affected) foot for less time.*
- *Pick the other (affected) foot up a little higher.*

A horse that drags a weighted object with one pastern will tend to:

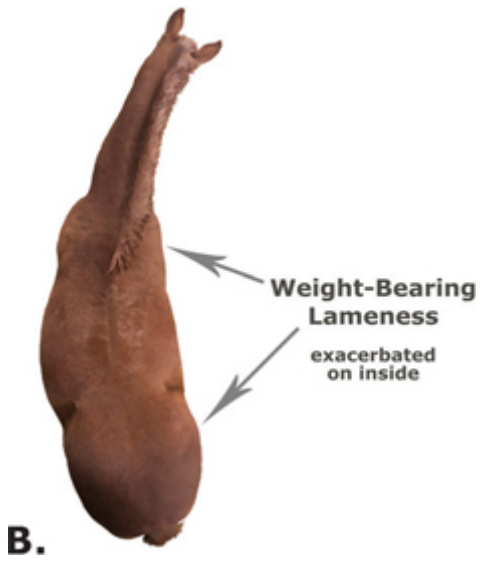
- *Be late in moving the (affected) limb forward.*
- *Not move the (affected) limb as far forward.*
- *Not pick the (affected) limb up as high.*

24.5 The Influence of Directional Changes on the Visual Expression of Weight-Bearing and Non Weight-Bearing Lameness

Weight-Bearing Lameness

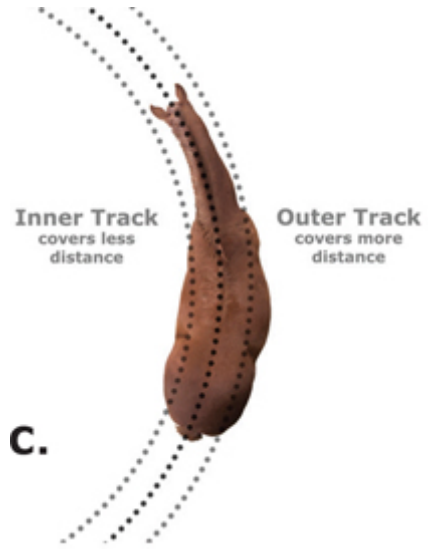


A. While navigating turns, the horse's body tends to angle inwardly. This response shifts the center of gravity toward the inside of the curve, approaching the tracking path of the horse's inner limbs.

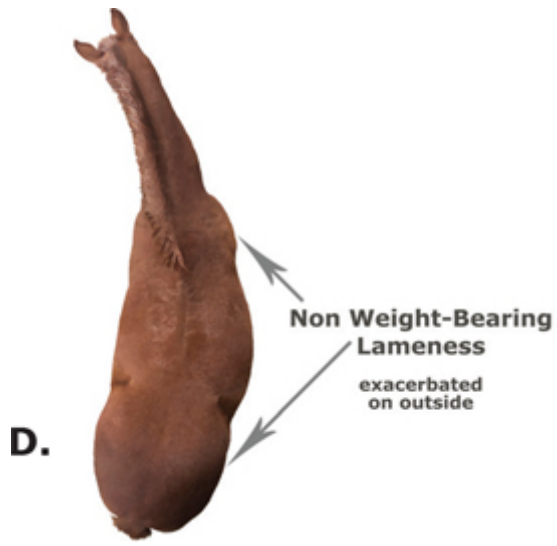


B. Interior relocation of the horse's center of gravity imposes added weight-bearing load on the limbs situated along the inside of the curve. Accordingly, lameness derived from weight-bearing pathology will be more conspicuous when the affected limbs are placed on the inside of a turn or circle.

Non Weight-Bearing Lameness



C. The horse's limbs situated along the outer aspect of the turn do not bear as much weight as those located along the inner aspect. The circumference of their tracking path, however, is longer than that of the inner limbs. The outer limbs consequently have to cover more ground in order to keep the horse moving in a balanced and synchronous manner. This translates into increased stride length.



D. Since non weight-bearing pathology is typically aggravated as a result of stride elongation, related lameness will be more obvious during periods when the affected limb is situated along the outside of a turn or circle.

24d
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Non weight-bearing issues, on the other hand, are not impacted by the degree of weight-bearing but rather the length of stride. A longer stride obligates increased protraction, upon which non weight-bearing pathology is directly challenged. The limbs situated along the outer aspect of the turn will indubitably demonstrate increased stride length, since they have more ground to cover in comparison to the limbs occupying the inside of the curve (fig. 24.5 C). Non weight-bearing asymmetry, therefore, will generally become more conspicuous when the affected limb is along the outside of a turn or circle (fig. 24.5 D and **VL 24e**).

VL 24e
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Altering the horse's directional path can be exceptionally conducive to interpreting combination deficits, because we can highlight each component of the lameness independently from one another. Placing the affected limb along the inside of a turn (or longeing circle, for instance) allows us to further illuminate the weight-bearing element. By contrast, the non weight-bearing component of the lameness will become considerably more recognizable when the affected limb is situated along the outside (**VL 24f**).

VL 24f
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Surface influences. Pathology that generates weight-bearing deficits tends to occupy the horse's lower limb and is, therefore, situated in close proximity to the ground surface. Weight-bearing issues are aroused by forces generated via the foot's interaction with the footing. Appropriately, hard footing will both create and transmit more intense concussive forces through the horse's limb, thereby accentuating the expression of any related weight-bearing gait deficits (**VL 24g**).

VL 24g
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Contrastingly, firm footing invariably facilitates flight (protraction) of the limb, which can more easily clear the ground surface during advancement. Non weight-bearing pathology is typically aggravated in horses that are forced to increase the height (in addition to the length) of the stride, such as may occur in deeper footing. It is common, therefore, for non weight-bearing deficits to become more flagrant as the ground surface gets softer (**VL 24h**).

VL 24h
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As you've probably guessed, each element of combination lameness can be distinguished by altering the density of the footing during the course of the examination. To maximize expression of weight-bearing deficits, for instance, we might longe the horse on asphalt and place the affected limb on the inside of the circle. Non weight-bearing asymmetry will become more obvious as we increase the depth and malleability of the footing while concurrently positioning the affected limb along the outside of the longeing circle.

Velocity influences. It is good practice to trot the horse as slowly as possible for the purpose of accurate evaluation. This policy is especially productive when scrutinizing weight-bearing lameness, because the affected limb is in contact with the ground surface for an extended period of time. In this context, our eyes have more time to pick up on any discrepancies in movement from one side of the horse to the other.

Increased contact time may not accentuate non weight-bearing deficits, however, since these are most heavily influenced by the length and height of the stride (rather than factors that come into play during stance). We can usually augment both the length and height of stride by quickening overall movement, an activity requiring each limb to stay in flight for a longer period of time. In many instances, this form of manipulation will induce the non weight-bearing lame horse to "skip" with the affected limb (**VL 24i**).

VL 24i
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25
Determining the Severity (Degree) of Lameness

When we observe lameness, we're not seeing the horse's problem. Instead, we're witnessing the horse's physical *response* to the problem. This reaction reflects the horse's attempt to avoid pain, accommodate biomechanical restriction, and/or maintain balance. It may or may not be commensurate with the intensity of its origin. Accordingly, when we visually judge the severity of lameness we're actually establishing the degree to which the horse reacts to pathology rather than the degree of pathology itself. A horse that is markedly lame, for instance, is communicating its response to the problem in a way that is very conspicuous to us. Another horse with the same type, location, and degree of pathology may be less expressive in their reaction and display only subtle gait deficits. As observers, we can be particularly fooled in cases of obscure (or hidden) lameness, in which the horse's expressivity is limited due to disparate complicating factors (see chapter 5, p. 26).

There is a disproportionate relationship between the degree of pathology that the horse endures and the degree of corresponding lameness that we observe.

There are two primary determinants that influence our ability to accurately and objectively evaluate equine lameness:

- Not all horses will react to the same problem in the same way.
- Not all observers will judge the same physical reaction in the same way.

The ramifications of these complicating factors are cumulative, and can make it very difficult for even the most seasoned of veterinarians to interpret the severity of gait deficits accordantly.[8] Still, assessing the magnitude of asymmetry has a number of benefits:

- It allows us to track the progression or regression of a single animal's pathology over time. By establishing a baseline "grade" of lameness, we can conclude the horse is getting better or getting worse from one day to the next.
- It allows us to compare the severity of similar gait deficits displayed by multiple horses. We utilize consistent, reproducible patterns of movement that are exhibited among a population of animals to assist in our visual interpretation of lameness. Appropriately, we know the approximate degree of lameness that horses enduring certain maladies would be expected to exhibit. This type of evidence provides us the means to further define *gait signatures* that complement unique forms of pathology (see section VII).
- It can provide insight into the particulars of the horse's problem. For example, a medial foot bruise (located along the inside of the foot) might be expected to generate very similar (if not identical) gait characteristics to those precipitated by a medial foot abscess (occupying the same location). We would expect the degree of lameness, however, to differ between the two afflictions (abscesses generally hurt more than bruises). Moreover, we would anticipate eventual resolution of the foot bruise (as demonstrated by a progressive decrease in the severity of the horse's asymmetric movement) whereas lameness evoked by the presence of a foot abscess might be expected to escalate with time.

Standardized Grading of Lameness

For the purpose of promoting and facilitating networking between horse enthusiasts, the American Association of Equine Practitioners (AAEP) has devised a grading scale pertaining to the severity of equine lameness. The scale ranges from zero to five with zero being no perceptible lameness and five being the most severe form (fig. 25.1).

25.1 American Association of Equine Practitioners (AAEP) Grading Scale[16]

Grade of Lameness	Visual Interpretation
0	Lameness is not perceptible under any circumstances.
1	Lameness is difficult to observe and is not consistently apparent, regardless of circumstances (i.e. under saddle, circling, inclines, hard surfaces).
2	Lameness is difficult to observe at a walk or when trotting in a straight line, but consistently apparent under certain circumstances (i.e. weight-carrying, circling, inclines, hard surface).
3	Lameness is consistently observable at a trot under all circumstances.
4	Lameness is obvious at a walk.
5	Lameness produces minimal weight-bearing in motion and/or at rest, or a complete inability to move.

Following is an attempt to clarify the AAEP denotations for some of the non-professional readers:

0. The observer cannot detect consistent lameness in any circumstance. *The horse is considered to be sound.*

1. The horse exhibits subtle asymmetry on occasion, although it is not always associated with the same region or limb. Moreover, the horse's lameness cannot be anticipated nor exacerbated through environmental or gait manipulation. In other words, *you know that there's something wrong but you can't put your finger on it.*

2. Lameness is not apparent until you ask for a specialized gait (e.g. canter, rack, etc.) and/or manipulate the environment in a certain way (such as trot the horse on hard ground and/or in tight circles), through which you can expose the lameness in a consistent and predictable manner. *Thus you can make the horse look lame if you want, otherwise it appears to be sound.*

3. Lameness is visible at the trot, but not at the walk.

4. Lameness is visible at the walk and trot, but not while standing still.

5. Lameness is visible in the standing horse (that is resting and/or holding up the affected limb).

As observers, our goal is not to decide *how lame the horse looks*, but rather to establish the circumstances under which we can detect asymmetric movement. We may not always agree with each other, but we are inclined to agree with our own conclusions from one day to the next and with respect to our assessment of multiple animals.

For those of us that employ the "hole-brick" phantasm (see chapter 24, p. 170) when observing lameness, estimating the severity can be a transparent exercise:

- In the case of *weight-bearing lameness* in which the comfortable limb is stepping into a hole, we simply need to determine the perceived "depth" of the hole (fig. 25.2 A and **VL 25a**). *The deeper the hole, the more severe the lameness.* One might denote grade 3/5 lameness as a 2-inch hole and a grade 4/5 as a 4-inch hole, for instance.
- In the case of *non weight-bearing lameness* in which the affected limb is dragging a brick, we simply need to determine the perceived "weight" of the brick (fig. 25.2 B and **VL 25b**). *The heavier the brick, the more severe the lameness.* One might denote grade 3/5 lameness as one-half a brick and a grade 4/5 as a whole brick, for instance.

25.2 A Simple Way to Estimate the Severity of a Horse's Lameness

A shallow hole would signify subtle (or mild) weight-bearing lameness.

A deep hole would represent obvious (or severe) weight-bearing lameness.

A. Weight-Bearing Lameness (Associated with the Left Front Limb): In cases of *weight-bearing* lameness (in which the sound limb is stepping into an imaginary hole), you simply appraise the perceived depth of the hole to estimate the severity of the horse's lameness.

A light brick would denote subtle (or mild) non weight-bearing lameness.

A heavy brick would indicate obvious (or severe) non weight-bearing lameness.

B. Non Weight-Bearing Lameness (Associated with the Left Front Limb): In cases of *non weight-bearing* lameness (in which the affected limb is dragging an imaginary brick), you simply appraise the perceived weight of the brick to estimate the severity of the horse's lameness.

VL 25a
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VL 25b
Scan/Click to view video.
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We have the capacity to influence the horse's expression of lameness by manipulating the set of circumstances under which we make our assessment. This does not exclude the intensity of expression, which can also be regulated in foreseeable fashion.

Gait influences. The "hole-brick" apparition is often very conducive to establishing the nature and severity of an animal's lameness but doesn't necessarily take into account the gait. A horse stepping into a 2-inch hole at the walk, for instance, would be considered to be more lame than a horse stepping into a 2-inch hole at the trot, particularly if the latter display of lameness is not evident at the walk. This is the reason why our ability (or inability) to perceive lameness at certain gaits shapes the infrastructure of our grading scale (**VL 25c**).

VL 25c
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Surface influences. Anything that further challenges the functional operation of the affected anatomy will influence the severity of lameness. Ironically, we (as observers) usually go out of our way to accentuate the horse's expression of gait deficits for the benefit of our assessment. After all, we are trying to make the process as easy as possible. We can usually increase the severity of weight-bearing asymmetry by evaluating the horse on a firm surface (such as asphalt or concrete). By contrast, soft surfaces will frequently stimulate the horse to display altered movement characteristic of non weight-bearing lameness.

Velocity influences. Slower movement ordinarily increases the perceived severity of weight-bearing lameness and decreases the demonstration of non weight-bearing lameness. More time spent in the theoretical "hole" means there's more time for the horse's median anatomy to descend (into the comfortable limb). Accordingly, this action becomes more conspicuous to the observer during the assessment of weight-bearing lameness.

Increasing the horse's speed naturally provokes the opposing response: the horse is less proficient at demonstrating weight-bearing asymmetry but is more adept at displaying non weight-bearing deficits. Horses hauling a theoretical brick must work more expeditiously in order to maintain the proper timing and length of stride at swift speeds. The inability of the horse to initiate movement of the brick (at the beginning of the flight phase of the stride) and nimbly drag the brick (for the duration of flight) yields gait deficits unique to non weight-bearing lameness.

Speed variation. The virtual "momentum" of the brick noticeably regulates action of the limb afflicted with non weight-bearing pathology during periods of acceleration and deceleration. A horse that is accelerating must increase the effort applied for the purpose of building up the brick's momentum. This increase in effort is visibly revealed to the observer, who may conclude that the horse's lameness is more severe during periods of a acceleration (**VL 25d**). The brick's momentum works in favor of the animal during deceleration, per contra, since the horse can passively let the object's speed slacken. Appropriately, non weight-bearing deficits tend to be less conspicuous during periods of deceleration.

VL 25d
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Horses with weight-bearing limb pathology respond antagonistically to adjustments in their rate of movement. In these cases, the intensity of compressive force applied to the affected limb(s) usually dictates any change in the degree of lateral disparity. As the horse accelerates, weight is shifted toward and onto the hind limbs, which function to drive the horse forward. The subsequential accumulation of structural load encountered by the hind limbs exacerbates any associated weight-bearing pathology. The observer readily discerns the byproduct of these events, which render more obvious hind limb weight-bearing lameness.

We can accurately surmise that the shifting of weight from front to back (transpiring during acceleration) will alleviate both front-end load and related weight-bearing lameness. The front end does encounter the ramifications of increased weight-bearing load during *deceleration*, however. Indeed, the anterior transfer of weight can impose dramatic effects on forelimb weight-bearing pathology for the following reasons:

- Horses are inherently "front-end heavy" and typically carry approximately two-thirds of their weight on their forelimbs. A moderate forward shift in weight can readily overburden structures directly employed for limb support.
- Due to their general physique, horses are much more proficient at transferring weight from the hind end forward as opposed to shifting weight in the other direction (see fig. 10.2, p. 48).
- Most horses have the ability to *decelerate* ("brake") at a much faster rate than they can *accelerate.* Tremendous compressive forces are applied to the forelimbs as the majority of the horse's weight is quickly "dumped" onto the front end.

Accordingly, forelimb weight-bearing asymmetry is usually exacerbated during deceleration as the affected areas/limbs are forced to assume more weight-bearing load. The observer can correspondingly witness the horse being "pushed farther in the theoretical hole" during this activity (**VL 25e**).

VL 25e
Scan/Click to view video.
www.getsound.com/tutorials/25e

26
Determining Extrinsic Influences on Lameness

The purpose of manipulating the horse's local environment is to make our job of seeing and characterizing lameness easier. We can accentuate the overall degree of asymmetric movement as well as the prominence of individual deficits by making deliberate alterations to our observation "stage." This approach is especially useful in cases of bilateral lameness in which the overall severity of asymmetry is reduced due to the horse's inability to favor contralateral limbs simultaneously (see chapter 5, p. 26). Ideally, the observer should assess the horse in motion as it encounters a variety of circumstantial challenges, some of which might require the assistance of a rider. (Note those environmental variations requiring the assistance of a rider are marked with an * on the pages that follow.)

Temporal and Thermic Variations

Cold. More credit is given to the movement of a horse that has not been turned-out or exercised prior to evaluation. Many issues (particularly those associated with joints) are alleviated during warm-up. For this reason, it may be helpful to perform your assessment "fresh out of the stall" whenever possible.

Warmed up. In some instances, an increase in tissue activity and blood flow can accentuate inflammation and associated pain. By contrast, other forms of pathology will become less obvious as blood flow increases and tissue pliability improves. Joint problems (such as arthritis) should be considered in horses that are considerably more comfortable following a period of warm-up.

Post exercise. Tendon and/or ligament issues should be considered in horses that display increased asymmetry following exercise, especially if associated limb swelling is evident.

Starting and stopping. Inflammation and pain associated with many structures that are challenged during exercise may have a delayed effect on the appearance and/or degree of observed lameness. It is not uncommon to observe a 1 to 2 grade increase in severity of lameness immediately following a five-minute break in the exercise routine. This observation may be suggestive of tendon, ligament, or coffin-joint inflammation.

Day-to-day changes. Regular assessment is an integral part of accurate interpretation and management of lameness. Horses should always be critiqued for differences in performance from one day to the next. Issues associated with or adjacent to the midline of the body (such as back problems), for instance, may not manifest as overt asymmetric movement between the two sides of the horse during a single evaluation/at any single point in time. Rather, the horse may merely exhibit indistinct stiffness and become "braced" in its movement. It could be construed as normal (comfortable) movement for the individual or as an indicator of pathology. A difference in this behavior (body carriage or stiffness) is more easily discerned over a period of multiple days.

Footing

Hard (concrete or asphalt). Hard surfaces function to increase the degree of concussion experienced by structures within the horse's distal or lower limb (such as the foot). Lameness attributed to these structures will accordingly become more obvious as the horse is maneuvered on a hard surface. Weight-bearing asymmetry with a shortened caudal (posterior) phase to the stride is typically observed in conjunction with an increase in severity (or degree).

Soft (arena footing). Softer footing usually mitigates weight-bearing lameness except when attributed to the proximal (upper) aspect of the suspensory ligament, the third phalangeal insertion of the deep digital flexor tendon (DDFT), the collateral ligaments of the coffin joint, and/or the distal suspensory (impar) ligament of the navicular bone. In all of these cases, dropping of the horse's heel into the footing exacerbates tension (and related pain) of the aforementioned soft tissue structures. Correlating lameness is generally more obvious with the affected limb on the outside of the circle.

Softer footing can also make it more difficult for horses with fetlock joint pain, carpal joint disease, bicipital bursitis, and stifle problems to clear the ground surface during limb protraction, thereby augmenting the non weight-bearing asymmetry affiliated with these problems.

Wet (arena footing). Sticky footing provides the observer with yet another tool for inspecting gait characteristics, particularly with respect to understated foot dynamics. It may be difficult to discern, for instance, if one toe is dropping into the footing more than another. It may also be hard to see if one foot is dragging more than another (suggesting an asymmetric hypometricity). Wet footing can effectively "paint" a picture of recent activity on the hoof wall, creating a depiction that is available for our ensuing review (after the fact). Static markings provide valuable insight into the foot's dynamic activity (see fig. 22.9, p. 155).

We can also examine the horse's footprints left in wet (or malleable) footing subsequent to active assessment. The prints serve as stationary molds that illustrate the mechanical details of the foot during the stance phase of the horse's stride. We can use our static study to confirm or deny our dynamic impressions assembled over the course of the active examination. The "sand splash" left behind by the spattering of footing debris can also reveal a lot about how the horse places, moves, and breaks over the feet during movement—all valuable clues that shouldn't be ignored (see **VL 22x**, p. 156).

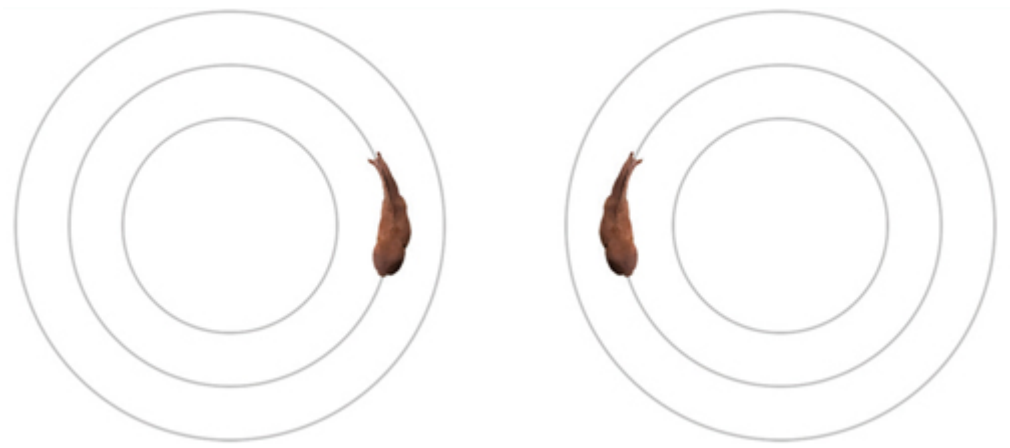
Direction

Straight line. Your subject should initially be walked and trotted in a straight line on a flat, firm surface. Some horses with suspensory desmitis (ligament inflammation) or fetlock arthritis (joint inflammation) will exhibit more asymmetry while being trotted in a straight line as opposed to being turned in either direction.

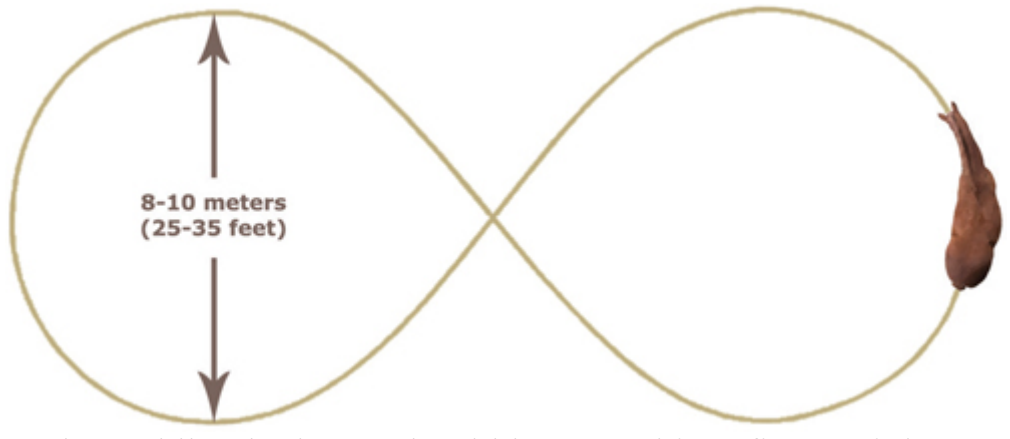
Circles. Circling (longeing) is an aggressive activity, but it can be extremely valuable for screening many forms of lameness (fig. 26.1 A). We can effectively alter lateral loading of the lower joints and feet via this activity: The outer limb experiences an increase in medial (inside) compression and lateral (outside) tension, whereas the inner limb experiences opposite forces. We also affect the overall weight-bearing load and stride length experienced by all four limbs. The inner limbs will be subjected to greater weight-bearing load as the horse's center of gravity "leans into" the corresponding side. Proportionally, the outer limbs will experience less loading as the body "leans away" from them. For this reason, most weight-bearing issues are accentuated with the affected limb toward the inside of a circle and abated when the affected limb is on the outside of the circle (see VL 24d, p. 174).

26.1 Popular Movement Patterns Used to Enhance Visual Assessment of the Lame Horse

A. Longeing in circles. The horse should be longed in both clockwise and counterclockwise directions.



B. Concentric circles. The horse should be longed in circles of varying size to assess the ability of the axial anatomy (neck and back) to bend in both directions.



C. Figure eight. While under saddle, the horse should be trotted in a figure-eight pattern on a loose rein. The diameter of each each loop should be approximately 8–10 meters.

Exceptions include those problems aggravated as a result of altered compressive loads from one side of the limb to the other: Medial foot problems, coffin joint pain, distal lateral collateral ligament compromise, lateral suspensory branch injury, proximolateral suspensory desmitis, and some forms of pathology associated with the

inside of the carpus may display increased weight-bearing lameness in a setting in which the affected limb is on the outside of the circle (**VL 26a**).

VL 26a
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Non weight-bearing issues, which are directly influenced by stride length, are almost always exacerbated with the affected limb on the outside of a circle. This is due to the outer limbs' need to accommodate a larger circumference and cover more ground (see fig. 24.5 C, p. 175). Appropriately, non weight-bearing lameness may dissipate or even disappear when the affected limb is placed on the inside of a circle.

Analyzing the horse's ability to negotiate concentric circles of diminishing size and/or traveling in a spiral pattern can also be very informative (fig. 26.1 B). We can accentuate asymmetry between diagonals as we shorten the diameter of the longeing circle, thereby precipitating exposure of obscure gait deficits. In some cases of bilateral forelimb lameness, for instance, the horse may have an increasing propensity to "look" outwardly as the circles shrink in size, even though asymmetry associated with the limbs may not be readily apparent (**VL 26b**). It should be noted, however, that sound horses can display artificial or physiologic lameness if longed in very small circles, particularly if they are moving quickly.[17,18]

VL 26b
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Horses are normally expected to lean toward the center of a circle at a slight angle (of up to 15 degrees). As the longeing circle shrinks, the normal horse will bend or curve the body more to allow for consistent tracking along the tighter circumference. At some point during concentric longeing, most back-sore horses will cease to bend farther and start to lean inward excessively. Some of these horses will concurrently alter their body angle to face inward, suggesting the possible presence of pain along the lower back region (see **VL 14g**, p. 82).[15]

Figure-eight pattern*. Observing the horse navigate a figure-eight pattern at the walk and trot is considered an essential component of the under-saddle assessment. The observer can easily compare differences in the horse's movement from one direction to the other, as well as detect any deficits that manifest during directional or lead changes. Each end of the figure eight should have a diameter between 8 and 10 meters (fig. 26.1 C).

Gradient

Flat ground. All basic evaluations should be performed on a flat surface, so as to avoid the inadvertent fabrication of deficits that arise as the horse attempts to negotiate rough or slanting terrain. The placement of each foot as it strikes the ground surface can also be more accurately assessed on a flat, firm surface.

Incline. The horse's weight is shifted in a craniocaudal (front-to-back) direction while navigating an incline. Asymmetry associated with femoropatellar (stifle) or hip joint pain may be more obvious when walking or trotting uphill.

Decline. The horse's weight is shifted in a caudocranial (back-to-front) direction while walking downhill. Horses that have trouble negotiating declines may be suffering from pain affiliated with the front feet, lumbosacral, and/or sacroiliac joint(s). Lameness resulting from biomechanical interference of the stifle (such as intermittent upward patellar fixation or proximal patellar hesitation) may be dramatically accentuated as the horse walks downhill. Neurologic deficits (especially proprioceptive and circumductive defects relating to the hind limbs) may also manifest on a decline.

Gait

Walk. It is important to initially examine the horse on a firm, flat surface at the walk so as to better assess overall motion and individual foot placement. Severe (grade 4/5) lameness will be evident at the walk (**VL 26c**). In some cases, excessive head and neck excursion attributed to pathology within the upper forelimb may generate a non weight-bearing lameness that is more conspicuous at the walk as opposed to the trot.

VL 26c
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Trot. This is the basic "lameness assessment gait," because it provides the observer with two diagonals, each of which can be visibly compared to the other. If you only have time to assess one activity, watch the horse trot in a straight line on a firm surface.

Canter. The observer should pay particular attention to back movement while the horse is cantering. In cases of sacroiliac joint pain and/or biomechanical stifle interference, the quality of canter may be worse than that of trot. Inappropriate or untimely canter-lead changes may occur in cases of acute lumbar pain, biomechanical stifle interference, excessive lower hock pain, proximal suspensory desmopathy, fetlock joint pain, and/or sub-clinical ataxia (neuropathy).

Both canter leads (left and right) should be evaluated. Differences in forelimb "scissoring" between canter leads can reveal potential front-end issues that may not be obvious at the trot. Horses that are unwilling to canter with one forelimb leading might have pain associated with the respective limb and/or the contralateral hind limb. Horses that are unwilling to canter with one forelimb trailing may be experiencing deep digital flexor tendon (DDFT) pain within the respective limb.

Four-beat gait (rack, slow gait, walk). Familiarity with these activities is required to make accurate conclusions with regard to the gaited horse's soundness, particularly if the animal cannot be persuaded to trot. If your horse is gaited, acquire baseline video footage and store it away for future comparative use.

Transitions. The horse should be observed during upward and downward transitioning in multiple gaits. Horses with biomechanical interference of the stifles or mid-back pain may express behavioral resistance when asked to pick up the canter from the trot (an upward transition). Since the extension phase of the lead hind limb stride is prolonged during downward transitions, this is a likely time for intermittent upward patellar fixation (IUPF) to occur. From a visual standpoint, this condition looks as if the hind end suddenly falls through a trap door (**VL 26d**). Most horses recover from the event within a few strides.

VL 26d
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Lateral movements*. By moving the horse laterally (shoulder-in, half-pass, etc.) we effectively increase side (medial-to-lateral) loads on the feet and lower joints in addition to influencing the degree of upper-limb adduction/abduction. We also increase the degree and duration of extension associated with the trailing limbs, thereby precipitating associated non weight-bearing pathology. Depending on the local source of pain (e.g. one side of the foot, a discrete suspensory branch or lumbar musculature along one side), lateral movements can evoke

very specific alterations to the horse's gait. When applicable, the horse should be observed performing both left and right half-passes at all gaits.

Extended movements*. Increased limb protraction is necessary to perform extended movements (such as the medium trot in dressage). Consequently, non weight-bearing lameness will often become more obvious to the observer in this setting. Shortening of the caudal or posterior phase of the horse's stride and/or "skipping" during extended movement may be suggestive of flexor-tendon pathology.

Jumping*. Jumping a horse for the purpose of visual assessment is only useful for animals that perform this activity as part of their standard riding discipline. *This activity is not recommended for animals that have not grown accustomed to it.* Horses that deviate the front end (directionally) over jumps and/or land heavier on one side may have pain associated with one of the forelimbs and/or the thoracic dorsal spinous processes (the withers).

Upon landing from a jump, the trailing forelimb experiences greater weight-bearing load (and subsequent ground reaction force). Therefore, horses will usually choose to land with the affected limb forward (or leading) in the case of weight-bearing lameness. By contrast, horses with non weight-bearing fore-limb lameness almost always prefer to trail with the affected limb when touching down.

Horses that consistently jump "crooked" might be suffering from hind limb weight-bearing lameness. A lack of propulsion afforded by the lame limb will often prompt the horse to jump toward the direction of the affected side.[19] For instance, a horse with right hind weight-bearing lameness might veer off and to the right over jumps. Horses with non weight-bearing hind-limb lameness may also have trouble negotiating jumps. Many horses with biomechanical stifle interference, for instance, will not clear the rail or may refuse to jump altogether.

Backing up. Horses that consistently move toward the right or left side while backing up may display *iliopsoas* (lower back) muscle pain upon veterinary examination. In these cases, the horse can be expected to move toward the painful side.

Velocity

Slow trot. The temporal resolution of the observer improves as the horse moves more slowly[20], meaning that it is easier to visually process slow movement as compared to swift movement. Consequently, most forms of lameness are more conspicuous in the slow-moving subject. It is for this reason that a horse moving at a slow, consistent rate equips us with a superior context for visual analysis.

Fast trot. As the subject moves more quickly, we begin to test the temporal resolution limits of our eye. Lameness may appear to be less severe at a higher rate of motion, even though it is physiologically identical to what it was at a lesser speed (**VL 26e**). This is an important concept when considering the fact that a lameness that worsens with increased velocity may actually appear unchanged (or even improved) to the observer's eye. Thus an authentic increase in the severity of lameness may be appropriately balanced by our reduced perceptual competence at higher velocity.

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Since both velocity and circle diameter can influence locomotive symmetry, it is important that both parameters remain within reasonable limits during the course of the evaluation. Horses moving at excessive speeds in very tight circles can develop artificial deficits associated with the inside limbs, thereby complicating our visual assessment.[17,18]

Deceleration. Weight-bearing lameness associated with the forelimbs often becomes more obvious while the horse is slowing or coming to a stop (see **VL 25e**, p. 182). More weight is shifted onto the front of the horse during this activity.

Acceleration. Problems associated with the horse's upper hind limbs, pelvis, and lower back may become more evident during acceleration, as body weight is shifted in a craniocaudal (front-to-back) direction (see **VL 25d**, p 181).

Form of Restraint

Free in round pen. Horses moving freely have more opportunity to express themselves through head, neck, and body motion. Scrutiny of the horse's back is considerably less complicated in this setting. It is important to encourage the subject to maintain a consistent gait and speed in the round pen so that a proper assessment can be performed.

Lead line. Make sure that the handler allows ample slack in the lead line so as to minimize influence of head and neck movement. Horses that tend to extend the neck (straight out in front) while in hand may be protecting a painful region farther back along the axial skeleton.

Longe line. The longe provides an excellent setting within which to assess the effects of direction on the nature and degree of limb lameness. It is also an effective way to evaluate movement of the horse's back. In many cases, the painful segment of the spine will deviate toward the inside of the circumferential path of the longeing circle.

Under saddle*. The rider serves several basic functions with respect to our visual assessment of the horse:

- The weight of the rider incites engagement of the *axial flexor musculature,* which helps the horse to maintain appropriate posture while under saddle. Problems associated with the horse's thoracic and lumbar spine will often materialize as the rider climbs aboard or applies leg pressure.
- The rider adds to the overall weight-bearing load encountered by the horse's limbs. Weight-bearing lameness is often accentuated under saddle, particularly when affiliated with the hind limbs. By contrast, non weight-bearing limb lameness is much less influenced by rider weight alone.
- The rider administers physical cues to influence the horse's activity (turning, jumping, collection, etc.). Retaining the assistance of a rider furnishes the observer with a tremendous advantage, as the horse can be systematically summoned to perform activities for the purpose of clarifying the visual assessment. For example, the visible expression of sacroiliac joint pain[21-23] and/or biomechanical stifle interference is often accentuated during increased hind-limb engagement and activity. Saddle slip to one side may indicate hind-limb lameness along that side, presuming that the saddle fits the horse appropriately.[24,25]
- The rider is usually willing to relate any impressions "felt" during the course of the evaluation, thereby supplementing the observer's visual findings. Rider opinion often clarifies the assessment and should be solicited whenever possible.

In some cases, the utilization of a rider can complicate the process, however. As we discussed in chapter 4 (p. 23), horses will alter their body movement to avoid pain, accommodate for biomechanical restriction, and/or maintain balance. We should remember that the human counterpart of the team is undergoing the same process of adjustment for their own pathology(ies) and that these alterations of movement will be transmitted into the horse's body. As such, the ridden horse is having to accommodate two sources of input: self and rider. Rider-induced adjustments can result in the visual manifestation of artificial equine lameness, particularly if the horse is merely trying to counteract abnormal extrinsic input. This constitutes the premise behind the horse's expression of rein lameness (see chapter 12, p. 52).

The horse will not only experience the rider's weight and body alterations under tack, but also any conscious or subconscious cues executed during the course of the assessment. Forelimb weight-bearing lameness, for instance, can be significantly (and sometimes inadvertently) mitigated via contact with the bit and/or through increased collection. Use of a weighted surcingle is an effective method for mimicking the passive impact of a rider without affording the concern associated with inconsistent rider contribution (fig. 26.2).

26.2 The Use of a Weighted Surcingle During Visual Lameness Assessment

You can reproduce the effects of rider burden via the employment of a *weighted surcingle*. This method helps to clarify the assessment by eliminating referred (artificial) movements evoked through abnormal passive and active human input.

***Loose rein*.** To accurately determine the influence of collection, the rider should first avoid contact and allow the horse's head, neck, and body to move as freely as possible while under saddle. Abnormal carriage of the neck and back in this setting can be indicative of problems associated with these areas. Those issues influenced by weight alone can also be more clearly isolated. Severe resentment of the horse-to-rider weight in the absence of contact might imply thoracic and/or thoracolumbar vertebral pain (possibly stemming from kissing spines) or rib injury. It should be noted that some horses with gastric ulcers and some cycling mares (those in estrus or "heat," for instance) also react to the burden imposed by the rider.

***Choice of bit*.** Problems affiliated with the horse's mouth, teeth, jaws, and/or temporomandibular joints (TMJs) are often conveyed through altered axial movement, and/or poor behavior (see chapter 12, p. 52). Alternating the employment of various types of bits can often guide us to the root of the problem.

***Collection*.** During collection, force vectors converge as forelimb breaking and hind limb propulsion are simultaneously employed. The culmination of the two converging forces promotes "hollowing" of the back[12], an action that is immediately countered by cues from the rider. Hind-limb activity (engagement) and suspension are increased during collected work. The horse's hocks and stifles are particularly challenged by this activity.

***Posting*.** While tracking in a figure-eight pattern, the rider may be petitioned to use the rising trot and intermittently avoid changing diagonals to enable the observer to evaluate all four limbs under increased load in both directions. Observing the animal as the rider posts on both correct and incorrect diagonals can be extremely rewarding, depending on the circumstances. For example, hind-limb weight-bearing lameness is often accentuated when the rider sits on the diagonal accommodating the lame limb. Some instances of hind suspensory desmitis will appear worse as the rider posts on the opposite (and sometimes incorrect) diagonal. This finding may be accompanied by a hypometric, non weight-bearing deficit in which the toe of the affected limb drags during the first half of the cranial (flight) phase of the stride.

***Sitting*.** Soliciting the rider to sit may eliminate any optical illusions inadvertently generated while posting. Horses with lower-back (thoracolumbar) pain may overextend the head and cranial neck during the sitting trot.[19]

***Two-point stance*.** Riding in two-point is sometimes used to indirectly assess the influence of back pressure on the horse's performance. Horses with issues affecting the mouth and/or temporomandibular joints (TMJs) may also exhibit a visible response to the revision of rein tension and its direction of application.

The observer can use a variety of strategies during evaluation to enhance the horse's expression of irregularity (fig. 26.3). Turning the horse sharply to the right, for example, might reveal lameness that was not previously evident as the horse moved in a straight line. Irregularity might further be aggravated when the horse is turning right on a hard surface, such as asphalt. In this example, the horse's response to changes in direction and surface provide valuable information with regard to the nature and location of potential pathology.

26.3 Extrinsic Influences on the Nature of Lameness

Extrinsic Feature	Nature of Lameness	Notes

	Weight-Bearing	Non Weight-Bearing	
Hard Surface	More Obvious	Less Obvious	
Soft Surface	Less Obvious	More Obvious	
Inside of Turn/Circle	More Obvious	Less Obvious	
Outside of Turn/Circle	Less Obvious	More Obvious	
Low Velocity (Slow)	More Obvious	Less Obvious*	Excluding some forms of pathology affecting the horse's stifle, shoulder, and neck regions.
High Velocity (Fast)	Less Obvious	More Obvious*	Some horses will demonstrate an artificial (or referred) "skip" associated with the affected limb if its rate of protraction cannot keep pace with the speed of travel.
Acceleration	Less Obvious*	More Obvious	Excluding cases of severe hind-limb lameness.
Deceleration	More Obvious	Less Obvious*	Excluding non weight-bearing lameness related to biomechanical interference of the hind stay-apparatus (e.g. delayed patellar release or upward patellar fixation).
Upward Transitions	Less Obvious	More Obvious	
Downward Transitions	More Obvious	Less Obvious*	Excluding non weight-bearing lameness related to biomechanical interference of the hind stay-apparatus (e.g. delayed patellar release or upward patellar fixation).

Deliberate manipulation of the horse's environment can also help to simplify the evaluation of combination lameness (comprising both weight-bearing and non weight-bearing deficits) by visually segregating the two components. The weight-bearing component might be expected to prevail when the horse's affected limb is placed on the inside of a circle, for instance, whereas the non weight-bearing component of the same lameness might be visibly dominant when the horse moves with the affected limb on the outside of a circle (see **VL 24f**, p. 176).

27
Identifying Distinctive Features of the Gait

As discussed in chapter 13 (p. 70), our perception of unique gait characteristics can dramatically facilitate and further define our visual assessment of the lame horse. Identification of "standout" traits might help to make sense of previous observations as well as confirm our suspicions with regard to potential causes. Accordingly, making an effort to discern unique deficits is invariably worthwhile. Each distinctive feature of the horse's gait maintains one of the following designations: *suggestive*, *symptomatic,* or *pathognomonic*.

Suggestive Traits

Horses can retain unique traits that depict their overall ability to perform under certain circumstances. Although suggestive traits may be exposed on a relatively consistent basis, they do not distinguish explicit forms of pathology. These features of the horse's gait are something that we often observe over time (over a series of evaluations) and/or within a population of animals. Their recognition frequently directs the observer toward other (more definitive) visual markers (fig. 27.1).

27.1 Lameness Traits That Are *Suggestive*

Visual Interpretation	Casual Deduction(s)
Forelimbs	
Intermittent lameness	Biomechanical source
Bilateral lameness	Problem is likely below the level of the fetlock joint
Bilateral weight-bearing lameness	Navicular inflammation
Lameness is worse on a hard surface	Pain in the foot
Lameness is worse when turning sharply in one direction	Pain in the foot
Horse improves with warm-up	Joint pain
Horse worsens with warm-up	Tendon or ligament injury
Horse exhibits greater degree of lameness in a straight line as opposed to turning in either direction	Fetlock joint pain, suspensory desmitis
Hind Limbs	
Intermittent lameness	Biomechanical source
Bilateral lameness	Problem is likely below the level of the fetlock joint
Bilateral weight-bearing lameness	Distal tarsitis (lower hock pain)
Bilateral non weight-bearing lameness	Proximal patellar hesitation, intermittent upward patellar fixation
Lameness is worse on a hard surface	Pain in the foot
Lameness is worse when turning sharply in one direction	Pain in the foot
Horse improves with warm-up	Joint pain
Horse worsens with warm-up	Tendon or ligament injury
Horse exhibits greater degree of lameness in a straight line as opposed to turning in either direction	Fetlock joint pain, suspensory desmitis
Axial Anatomy	
Horse exhibits behavioral resistance to riding in the absence of overt limb lameness	Axial lameness
Horse walks for a prolonged period before picking up the trot	Thoracolumbar (TL) myositis
Horse obviously prefers the canter over the trot	Thoracolumbar (TL) myositis
Horse consistently holds the tail off to one side	An issue affecting the hind limb from which the tail is pointing *away*

Symptomatic Traits

Most lame horses display gait aberrations that carry casual significance with regard to their cause. These anomalies are generally consistent from one animal to the next. For instance, we would expect two horses afflicted with the same musculoskeletal disease to exhibit similar gait deficits, even if one of the horses resides in China and the other in the United States. Since symptomatic traits are somewhat emblematic, they can be informally appropriated to rule in or rule out potential causes for lameness (fig. 27.2).

27.2 Lameness Traits That Are *Symptomatic*

Forelimbs	
Visual Interpretation	**Casual Deduction(s)**
Bilateral weight-bearing lameness in the forelimbs	Navicular inflammation
An obvious and dramatic shortening of the caudal (backward) aspect of both fore strides (bilaterally)	Severe navicular inflammation, laminitis
Horse increases angle of forefoot by sticking toe into footing	*Negative palmar angulation,* flexor tendonitis, palmar digital tenosynovitis, deep digital flexor insertional tenopathy, heel pain, navicular inflammation
Forelimb weight-bearing lameness worse with lame limb on the outside of a circle	Medial (inside) foot pain, coffin joint pain (excluding medial collateral desmitis), proximal (high) suspensory desmitis, medial carpal (knee) bone injury, medial (inside) splint desmitis/fracture
Forelimb combination lameness worse with lame limb on the outside of a circle	Fetlock joint pain, carpal joint pain, flexor tendonitis, palmar digital tenosynovitis, distal (mid and low) suspensory desmitis, splint desmitis/fracture
Foretoe dragging	Fetlock joint pain, carpal (knee) pain, elbow joint pain, severe shoulder-joint pain, bicipital bursitis, caudal cervical (neck) arthrosis, median neuropathy, proximal musculocutaneous neuropathy
Both cranial (forward) and caudal (backward) phases of the fore stride are dramatically shortened	Scapular fracture, humeral fracture, severe scapulo-humeral (shoulder) joint pain
Excessive outward bulging of the shoulder with outward heel rotation during the stance phase	Brachial plexus neuropathy
Forelimb non weight-bearing lameness with hypermetric gait	*Extensor carpi radialis* tendon rupture
Weight-bearing forelimb lameness with reduced protraction	Heel pain
Weight-bearing forelimb lameness with excessive protraction	Toe pain
Excessive upward excursion of the head and neck during forelimb protraction	Icipital bursitis, scapular (shoulder blade) issues, caudal cervical (neck) arthrosis
Forelimb combination lameness with an obvious inability to protract limb	Shoulder-joint pain, caudal cervical (neck) problem (C6-7)
Forelimb non weight-bearing lameness with an obvious inability to protract limb	Bicipital myositis/bursitis, caudal cervical (neck) problem (C6-7)
Forelimb non weight-bearing lameness with an obvious inability to protract limb and an intermittent weight-bearing component	Caudal cervical (neck) problem (C6-7)
Obvious and excessive “fetlock drop” during stance	Suspensory desmitis, superficial digital flexor tendon rupture
“Dropped” elbow with an inability to maintain fore-limb extension	Olecranon fracture, radial nerve paralysis, brachial plexus neuropathy
Hind Limbs	
Visual Interpretation	**Casual Deduction(s)**
Hind foot does not land flat	Hind foot pain
An obvious and dramatic shortening of the caudal (backward) aspect of both hind strides (bilaterally)	Laminitis
Horse increases angle of hind foot by sticking toe into footing	Negative plantar angulation, flexor myositis or tendonitis, plantar digital tenosynovitis, deep digital flexor insertional tenopathy, heel pain, suspensory desmitis, stifle joint pain, biomechanical stifle interference
Hind toe dragging during the first half of the flight phase of the stride	Proximal (high) suspensory desmitis, neurologic disease
Hind toe dragging during the majority of the flight phase of the stride	Stifle joint pain or biomechanical interference, neurologic disease
Both cranial (forward) and caudal (backward) phases of the hind stride are dramatically shortened	Pelvic fracture, severe coxofemoral (hip) joint pain
Weight-bearing hind-limb lameness with reduced protraction	Heel pain
Weight-bearing hind-limb lameness with excessive protraction	Toe pain
Bilateral lateral deviation/rotation of the hocks (often with internal rotation of the foot) during the stance phase of the stride	Distal tarsitis (lower hock pain), gastrocnemius myositis
Unilateral external hind-limb rotation with severe lameness	Pelvic fracture, proximal femoral fracture
Hind-limb adduction during flight phase of the stride: one or both	Distal tarsitis, gastrocnemius myositis, greater trochanteric bursitis

hind limbs cross under the body during protraction and then move outwardly just before landing	(whorlbone)
Hind-limb abduction during flight phase of the stride: one or both hind limbs move away from the body during protraction and then move inwardly (toward the midline) just before landing	Intermittent upward patellar fixation, neurologic disease
Hind-limb weight-bearing lameness with hypermetric gait	Distal tarsitis (lower hock pain)
Hind-limb non weight-bearing lameness with hypermetric gait	Intermittent upward patellar fixation, stringhalt, shivers
Hock points inward and toe points outward during stance	Coxofemoral (hip) joint pain
Point of the hock "pops," "vibrates," or "shimmies" at the initiation of the flexion phase of the hind stride	Proximal patellar hesitation, intermittent upward patellar fixation
Point of the hock appears "dropped" or lower than its contralateral counterpart during stance	Compromise of the gastrocnemius muscle and/or tendon, compromise of the common calcaneal tendon, sciatic neuropathy
Point of the hock appears elevated or higher than its contralateral counterpart while the limb is extended in the face of severe lameness	Pelvic fracture, coxofemoral (hip) luxation, femoral fracture
Obvious and excessive "fetlock drop" during stance	Suspensory desmitis, superficial digital flexor tendon rupture
Hind fetlock "knuckles over" during stance	Upward patellar fixation, femoral nerve paresis, peroneal neuropathy, sciatic neuropathy
Horse crouches during ambulation	Femoral nerve paresis, sciatic neuropathy, lateral patellar luxation, intermittent upward patellar fixation
Axial Anatomy	
Horse overextends neck and holds the head excessively low	Cervical (neck) issue
Horse excessively bends the neck and resists tipping or tilting the head	Cranial cervical (neck) problem (C1-3)
Horse tips or tilts the head instead of bending in a circle	Mid to caudal cervical (neck) problem (C4-7)

Pathognomonic Traits

In some instances, horses demonstrate distinctive gait deficits which, when accurately recognized, can lead the observer to correct deductions regarding their definitive source (fig. 27.3). Having visual access to this brand of explicit lameness is like "finding the treasure," because the true origin is visibly communicated in the horse's movement. Since most pathognomonic deficits can mimic other gait abnormalities upon initial (casual) glance, the observer should be both intimately familiar with their appearance as well as committed to their meticulous characterization during assessment.

27.3 Lameness Traits That Are *Pathognomonic*

Visual Interpretation	Description	Tentative Diagnosis
Forelimbs		
The toe of the foot pitches upward off the ground surface during stance.	An inability to flex the digit (foot) results in its hyperextension under weight-bearing load.	Rupture of the deep digital flexor tendon.
The fetlock sinks to the ground surface during stance.	Disruption of fixed ligament attachments along the back of the fetlock joint result in a lack of associated support.	Rupture of the suspensory apparatus.
Hind Limbs		
The horse inadvertently stumbles on the hind end.	Often described as "falling through a trap door," this deficit most often occurs at the trot in tight corners and during downward transitions (from trot to walk, for example). It is a less dramatic version of upward patellar fixation. The horse usually recovers within one or two strides and resumes normal work.	Delayed patellar release or proximal patellar hesitation.
Hind limb is locked in extension with the toe dragging behind.	An inability to flex the stifle and hock due to persistent engagement of the stay-apparatus results in "locked" extension of the hind limb.	Upward patellar fixation.
"Goose stepping" of one or both hind limbs.	Following a shortened cranial (forward) phase to the hind stride, the limb abruptly stops forward motion and moves backward to "slap" the ground surface upon contact.	Fibrotic myopathy of the hamstring musculature.
The horse flexes at the stifle while maintaining hock extension (which may be excessive), causing a visible dimpling of the Achilles tendon along the back of the limb above the hock.	The horse's stifle and hock joints are no longer "linked together" by the reciprocal apparatus of the hind limb, which requires an intact peroneus tendon to function.	Rupture of the peroneus tertius tendon.
The horse flexes at the hock while maintaining stifle extension during stance, resulting in a lowering of the point of the hock relative to the contralateral limb.	Disruption of the structural connection between the back of the femur and the point of the hock results in an inability to maintain hock extension.	Compromise of the common calcaneal (or Achilles) tendon, which is comprised of the gastrocnemius tendon and a portion of the superficial digital flexor tendon.
The toe of the foot pitches upward off the ground surface during stance.	An inability to flex the digit (foot) results in its hyperextension under weight-bearing load.	Rupture of the deep digital flexor tendon.
The fetlock sinks to the ground surface during stance.	Disruption of fixed ligament attachments along the back of the fetlock joint results in a lack of associated support.	Rupture of the suspensory apparatus.

We should emphasize the fact that pathognomonic gait deficits are not always primary and may exist concurrently with or as a result of other pathology(ies). Consequently, they should be interpreted only to signify the reason behind altered movement rather than the primary cause of the horse's lameness. Remember, lameness *diagnosis* is the veterinarian's responsibility.

Gait deficits associated with intermittent upward patellar fixation, "shivers," stringhalt, and fibrotic myopathy of the hamstring musculature are individually distinctive but can closely parallel one another from a visual standpoint. It would behoove the observer to learn the subtle differences between these maladies (fig. 27.4 and **VL 27a–d**).

27.4 Distinguishing the Appearance of Fibrotic Myopathy, Shivers, Stringhalt, and Upward Patellar Fixation

Pathology	Fibrotic Myopathy	Shivers	Stringhalt	Upward Patellar Fixation
General Appearance	Shortened cranial (forward) phase to stride; lengthened caudal (backward) phase	Episodic and sustained hyperflexion with concurrent abduction (holding the affected limb out and away from the body)	Excessive spasmodic hyperflexion of one or both hind limbs during movement	Fixed pelvic limb extension followed by sudden abrupt hyperflexion
Cause of Gait Abnormality	Functional shortening of the cranial (forward) phase of the stride due to mechanical restriction of fibrotic/scarred semi-tendinosus/semimem-branosus (hamstring)	Underlying neuropathy and/or episodic muscle cramping resulting in prolonged flexion/abduction of affected limb	Neuropathy resulting in lateral digital extensor muscle hyperactivity	Failure of the medial patellar ligament to disengage from the medial trochlea of the femur, thereby disallowing normal flexion of the hind limb

	musculature along the back of limb			
Gaits Most Affected	Walking	Standing, backing	Walking, turning, backing	Walking, ambulating downhill
Relative Frequency of Gait Deficit	Every stride	Sporadic	Every stride	Sporadic
Also Look For	Goose-stepping	Concurrent tail elevation, coexisting lameness, stressful environment	Laryngeal hemiplegia (“roaring”), forelimb stumbling	Visible or audible “pop” to limb at initiation of flexion
You Probably Won’t See	Hyperflexion of affected limb(s)	Visible or audible “pop” to limb at initiation of flexion	Visible or audible “pop” to limb at initiation of flexion	
Signature Trait(s)	Goose-stepping (the foot moves backward just before impact and slaps onto the ground surface abruptly)	Horse holds up affected limb for an extended period of time	Horse immediately returns affected limb back to the ground from the flexed position	Horse may drag affected hind limb behind the body while the patella is “locked” and the hind limb is fixed in extension; an obvious “snap” or “pop” of the hind limb (most easily seen at the point of the hock) occurs at the moment the medial patellar ligament disengages and the limb flexes from the extended position
Video Link	**VL 27a** *Scan/Click to view video.* www.getsound.com/tutorials/27a	**VL 27b** *Scan/Click to view video.* www.getsound.com/tutorials/27b	**VL 27c** *Scan/Click to view video.* www.getsound.com/tutorials/27c	**VL 27d** *Scan/Click to view video.* www.getsound.com/tutorials/27d

28
Confirming the Primary Component(s) of Lameness

Accurately decoding lameness may feel like an overwhelming endeavor at this point. Fortunately, we are not required to interpret all of our observations simultaneously. The proper evaluation protocol enables the observer to address one visual impression at a time. We only make sense of our findings after all the information has been accumulated and independently assessed.

The importance of distinguishing primary and secondary deficits was reviewed in chapter 7 (p. 36). This exercise can be accomplished anywhere throughout the evaluation process, although having predetermined the nature and features of each component makes the task considerably less challenging for the observer.

It is often helpful to presume (sometimes incorrectly) that there is only *one* primary element to each lameness and that all other components are secondary. Multifactorial lameness should be considered only if physiologic connections between numerous components cannot be rationally contrived. In all cases of complicated lameness, the potential expression of artificial (or referred) asymmetry should be acknowledged.

Weight-bearing hind-limb lameness typically generates referred weight-bearing deficits in the ipsilateral forelimb (on the same side of the horse). Accordingly, coexisting weight-bearing deficits expressed on opposite sides of the horse (comprising both limbs of a single diagonal pair) are usually physiologically unrelated.

Non weight-bearing hind-limb lameness frequently generates referred non weight-bearing deficits in the contralateral forelimb (on the opposite side of the horse). If non weight-bearing hind-limb lameness is a consequence of biomechanical interference of the stay-apparatus, however, it can generate a non weight-bearing "thoracic skip" associated with the ipsilateral forelimb (on same side of horse).

In all cases we should proceed with caution: Do not devote too much time and energy to interpreting forelimb asymmetry until you have confirmed that related gait deficits are authentic (not an artificial product of what is occurring behind).

Manifestations of Referred Lameness

As effective observers, we should constantly be seeking manifestation of referred gait deficits during the course of our examination. Their expression might engender the following visual impressions:

- Weight-bearing lameness in a hind limb can produce referred weight-bearing lameness in the forelimb along the same side of the horse. For example, a horse with the right hind (RH) weight-bearing lameness will often choose to transfer weight to the left front (LF) limb (which is the other limb of the same diagonal pair) in an attempt to reduce load. Increased load-bearing of the LF limb generates the (artificial) appearance of weight-bearing lameness in the right front (RF) limb (**VL 28a**). In this setting, the shifting of weight is the primary driving force. *Since the referral is from hind to fore, the secondary component is often more severe than its primary counterpart.*

VL 28a
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- Weight-bearing lameness in a forelimb can produce a referred weight-bearing lameness in the hind limb on the opposite side of the horse. For example, a horse with a RF weight-bearing lameness will often choose to transfer weight to the other diagonal pair (comprising the RH and LF limbs), thereby giving the impression that the left hind (LH) limb is lame (**VL 28b**). *The referred hind limb component is always considerably less obvious (less severe) than the primary (forelimb) lameness.*

VL 28b
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- Pain-mediated combination lameness in a hind limb displaying a significant non weight-bearing component can produce a distinctive lameness in the forelimb on the same side of the horse. The artificial fore-limb lameness appears to be weight bearing in that the head and shoulders drop (into a hole) when the horse lands on the opposing forelimb (which shares the diagonal pair with the affected hind limb), but in this case the head and shoulders move *forward* in addition to moving downward; this yields the impression as though the horse is being pushed or shoved from behind (**VL 28c**). *In some cases the referred component is more severe than its primary counterpart.*

VL 28c
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- Non weight-bearing biomechanical (non pain-mediated) lameness in a hind limb can produce a non weight-bearing lameness in the forelimb on the opposite side, as the horse attempts to match the timing and length of the stride between both limbs within a single diagonal pair (**VL 28d**). This deficit is generally evident in both the walk and trot. *In some cases the referred component is more obvious than its primary counterpart.*

VL 28d
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- Non weight-bearing lameness in a forelimb can produce a non weight-bearing lameness in the hind limb on the opposite side, as the horse attempts to match the timing and length of the stride between both limbs within a single diagonal pair (**VL 28e**). *In this case, asymmetry associated with the forelimb is always greater than that observed in the corresponding hind limb.*

VL 28e
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- Proximal patellar hesitation (PPH) or delayed patellar release in one hind limb can result in an artificial "skip" affecting the forelimb on the same side of the horse. PPH prevents the horse from flexing the hind limb from the extended position at the appropriate moment (see chapter 16, p. 91). The limb, therefore, remains in contact with the ground surface for an extended period of time. To maintain balance, the other limb of the diagonal pair (the forelimb on the opposite side of the horse) also remains grounded for a prolonged period of time. In the meantime, the horse adjusts body weight to enable the second diagonal pair to delay landing until the first diagonal pair is ready to initiate flight. This type of body adjustment manifests as an obvious, non weight-bearing lameness associated with the second diagonal's forelimb: The horse tosses the head and neck upwardly during the flight phase of the stride. To the average onlooker, it often appears as though the horse is trying to pick up the canter in front while continuing to trot behind (**VL 28f**). This action has been casually described as a "thoracic skip."

VL 28f
Scan/Click to view video.
www.getsound.com/tutorials/28f

- Severe bilateral forelimb lameness can result in odd movement and placement of the hind limbs and feet, respectively. In some cases, referred gait abnormalities resemble neurological deficits. Hind stride is markedly shortened as the horse constantly adjusts and readjusts for disproportionate weight distribution between the fore and hind limbs (**VL 28g**).

VL 28g

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First, use the horse's overall body adjustments to isolate the affected region or limb(s). Then use the nature of the horse's lameness to determine which part of the limb is most likely affected. Finally, use specific features of the gait to generate a list of likely sources.

Remember to keep the process as simple as possible. First, determine if one side of the front end of the horse is dipping excessively. Then determine if one side of the hind end of the horse is sinking and/or rotating. These exercises will tell you which limb—or limbs—is lame. Then assess movement, orientation, and carriage of the median anatomy (head, neck, and back) to decide if there are any related issues. Next ask yourself if the comfortable side is dropping into a hole, if the uncomfortable side is toting a brick, or if both activities are occurring simultaneously. This will help equip you with the means to establish the nature of the horse's asymmetry(ies). Subsequently, assessing the perceived depth of the imaginary hole and/or the perceived weight of the theoretical brick will permit you to estimate the degree (or severity) of each lameness component. Once you've accomplished these tasks, it's time to put the pieces of the puzzle together.

Let's navigate the process step by step. All deficits are identified via explicit evaluation of each end of the horse and each end's diagonal pair of limbs. Using figure 28.1, we will evaluate the front limbs first, looking at the left and right sides independently from one another.

A couple of notes regarding biomechanical interference of the hind stay-apparatus:

• Horses with delayed patellar release that tend to exhibit greater asymmetry at the trot (as opposed to the canter) often have considerable lower hock pain (distal tarsitis) as an inciting cause of the stifle interference. Otherwise, they are usually worse at the canter.

• Horses with primary severe forelimb weight-bearing lameness may develop secondary intermittent upward patellar fixation (IUPF) as a consequence of excessive hind-limb extension, which occurs as the horse attempts to underload the front end.

28.1 Step-by-Step Lameness Assessment of the Horse

A. Assess Left Front (LF) Limb Soundness

Determine Location and Nature of Lameness

1. Is there a LF limb weight-bearing lameness?

Look at the RF limb: Is it falling into an imaginary hole?

Left front weight-bearing lameness looks as if the horse is stepping into a hole with the right front foot.

NO There is not a weight-bearing lameness in the LF limb.

YES There is a weight-bearing lameness in the LF limb.

Determine Severity of Lameness

How deep is the hole?

The deeper the imaginary RF hole, the more severe the LF lameness.

Establish the Primary Source of Lameness

Is there a concurrent LH limb weight-bearing lameness?

Look at the RH limb: Is it falling into an imaginary hole?

Left hind weight-bearing lameness looks as if the horse is stepping into a hole with the right hind foot.

NO

YES There is a concurrent weight-bearing lameness in the LH limb. Consider the possibility that the LF lameness is referred from the LH (as a result of shifting weight onto the RF limb).

Is there a concurrent weight-bearing lameness in the RH limb?

Right hind weight-bearing lameness looks as if the horse is stepping into a hole with the left hind foot.

NO LF lameness is likely primary with no referred component.

YES Consider the possibility that the RH lameness is referred from the LF (as a result of shifting weight to the opposite diagonal pair), especially if LF lameness is considerably more severe than RH lameness.

Determine Location and Nature of Lameness

2. Is there a LF limb non weight-bearing lameness?

Look at the LF limb: Is it dragging an imaginary brick?

Left front non weight-bearing lameness looks as if the horse is dragging a brick with the left front pastern.

NO There is not a non weight-bearing lameness in the LF limb.

YES There is a non weight-bearing lameness in the LF limb.

Determine Severity of Lameness

How heavy is the brick?

The heavier the imaginary LF brick, the more severe the LF lameness.

Establish the Primary Source of Lameness

Is there a concurrent RH limb non weight-bearing lameness?

Look at the RH limb: Is it dragging an imaginary brick?

Right hind non weight-bearing lameness looks as if the horse is dragging a brick with the right hind pastern.

NO

YES There is a concurrent non weight-bearing lameness in the RH limb. Consider the possibility that the LF lameness is referred from the RH or vice versa (to maintain diagonal synchrony).

Is there a concurrent biomechanical interference (delayed patellar release) associated with the left stifle?

NO LF lameness is likely primary.

YES Consider the possibility that the LF lameness represents a "thoracic skip" referred from the LH.

Determine Location and Nature of Lameness

3. Is there a LF limb combination lameness?

Look at the LF and RF limbs: Is the LF dragging an imaginary brick and the RF falling into an imaginary hole?

Left front combination lameness looks as if the horse is dragging a brick with the left front pastern and stepping into a hole with the right front foot.

NO There is not a combination lameness in the LF limb.

YES There is a combination lameness in the LF limb.

Determine Severity of Lameness

How deep is the hole?

The deeper the imaginary RF hole, the more severe the weight-bearing component.

How heavy is the brick?

The heavier the imaginary LF brick, the more severe the non weight-bearing component.

Establish the Primary Source of Lameness

LF combination lameness is likely primary, since it would be unusual for referred lameness to manifest in this way.

B. Assess Right Front (RF) Limb Soundness

Determine Location and Nature of Lameness

1. Is there a RF limb weight-bearing lameness?

Look at the LF limb: Is it falling into an imaginary hole?

Right front weight-bearing lameness looks as if the horse is stepping into a hole with the left front foot.

NO There is not a weight-bearing lameness in the RF limb.

YES There is a weight-bearing lameness in the RF limb.

Determine Severity of Lameness

How deep is the hole? — The deeper the imaginary LF hole, the more severe the RF lameness.

Establish the Primary Source of Lameness

Is there a concurrent RH limb weight-bearing lameness?

Look at the LH limb: Is it falling into an imaginary hole?

Right hind weight-bearing lameness looks as if the horse is stepping into a hole with the left hind foot.

NO

YES There is a concurrent weight-bearing lameness in the RH limb. Consider the possibility that the RF lameness is referred from the RH (as a result of shifting weight onto the LF limb).

Is there a concurrent weight-bearing lameness in the LH limb?

Left hind weight-bearing lameness looks as if the horse is stepping into a hole with the right hind foot.

NO RF lameness is likely primary with no referred component.

YES Consider the possibility that the LH lameness is referred from the RF (as a result of shifting weight to the opposite diagonal pair), especially if RF lameness is considerably more severe than LH lameness.

Determine Location and Nature of Lameness

2. Is there a RF limb non weight-bearing lameness?

Look at the RF limb: Is it dragging an imaginary brick?

Right front non weight-bearing lameness looks as if the horse is dragging a brick with the right front pastern.

NO There is not a non weight-bearing lameness in the RF limb.

YES There is a non weight-bearing lameness in the RF limb.

Determine Severity of Lameness

How heavy is the brick? — The heavier the imaginary RF brick, the more severe the RF lameness.

Establish the Primary Source of Lameness

Is there a concurrent LH limb non weight-bearing lameness?

Look at the LH limb: Is it dragging an imaginary brick?

Left hind non weight-bearing lameness looks as if the horse is dragging a brick with the left hind pastern.

NO

YES There is a concurrent non weight-bearing lameness in the LH limb. Consider the possibility that the RF lameness is referred from the LH or vice versa (to maintain diagonal synchrony).

Is there a concurrent biomechanical interference (delayed patellar release) associated with the right stifle?

NO RF lameness is likely primary.

YES Consider the possibility that the RF lameness represents a "thoracic skip" referred from the RH.

Determine Location and Nature of Lameness

3. Is there a RF limb combination lameness?

Look at the LF and RF limbs: Is the LF falling into an imaginary hole and the RF dragging an imaginary brick?

Right front combination lameness looks as if the horse is dragging a brick with the right front pastern and stepping into a hole with the left front foot.

NO There is not a combination lameness in the RF limb.

YES There is a combination lameness in the RF limb.

Determine Severity of Lameness

How deep is the hole? — The deeper the imaginary LF hole, the more severe the weight-bearing component.

How heavy is the brick? — The heavier the imaginary RF brick, the more severe the non weight-bearing component.

Establish the Primary Source of Lameness

RF combination lameness is likely primary, since it would be unusual for referred lameness to manifest in this way.

C. Assess Left Hind (LH) Limb Soundness

Determine Location and Nature of Lameness

1. Is there a LH limb weight-bearing lameness?

Look at the RH limb: Is it falling into an imaginary hole?

Left hind weight-bearing lameness looks as if the horse is stepping into a hole with the right hind foot.

NO There is not a weight-bearing lameness in the LH limb.

YES There is a weight-bearing lameness in the LH limb.

Determine Severity of Lameness

How deep is the hole? — The deeper the imaginary RH hole, the more severe the LH lameness.

Establish the Primary Source of Lameness

Is there a concurrent LF limb weight-bearing lameness?

Look at the RF limb: Is it falling into an imaginary hole?

Left front weight-bearing lameness looks as if the horse is stepping into a hole with the right front foot.

NO

YES There is a concurrent weight-bearing lameness in the LF limb. Consider the possibility that the LF lameness is referred from the LH (as a result of shifting weight onto the RF limb).

Is there a concurrent weight-bearing lameness in the RF limb?

Right front weight-bearing lameness looks as if the horse is stepping into a hole with the left front foot.

NO LH lameness is likely primary.

YES Consider the possibility that the LH lameness is referred from the RF (as a result of shifting weight to the opposite diagonal pair), especially if RF lameness is considerably more severe than LH lameness.

Determine Location and Nature of Lameness

2. Is there a LH limb non weight-bearing lameness?

Look at the LH limb: Is it dragging an imaginary brick?

Left hind non weight-bearing lameness looks as if the horse is dragging a brick with the left hind pastern.

NO There is not a non weight-bearing lameness in the LH limb.

YES There is a non weight-bearing lameness in the LH limb.

Determine Severity of Lameness

How heavy is the brick? — The heavier the imaginary LH brick, the more severe the LH lameness.

Establish the Primary Source of Lameness

Is there a concurrent RF limb non weight-bearing lameness?

Look at the RF limb: Is it dragging an imaginary brick?

Right front non weight-bearing lameness looks as if the horse is dragging a brick with the right front pastern.

NO

YES There is a concurrent non weight-bearing lameness in the RF limb. Consider the possibility that the RF lameness is referred from the LH or vice versa (to maintain diagonal synchrony).

Is there a concurrent non weight-bearing "skip" associated with the LF limb?

Left front "skipping" looks as if the horse is attempting to pick up the right-lead canter in front while trotting behind.

NO LH lameness is likely primary with no referred component.

YES Consider the possibility that the LF lameness represents a "thoracic skip" referred from LH biomechanical interference.

Determine Location and Nature of Lameness

3. Is there a LH limb combination lameness?

Look at the LH and RH limbs: Is the LH dragging an imaginary brick and the RH falling into an imaginary hole?

Left hind combination lameness looks as if the horse is dragging a brick with the left hind pastern and stepping into a hole with the right hind foot.

NO There is not a combination lameness in the LH limb.

YES There is a combination lameness in the LH limb.

Determine Severity of Lameness

How deep is the hole? — The deeper the imaginary RH hole, the more severe the weight-bearing component.

How heavy is the brick? — The heavier the imaginary LH brick, the more severe the non weight-bearing component.

Establish the Primary Source of Lameness

Is there a concurrent LF limb lameness? Does the horse appear as though it is being "pushed into" a hole under the RF foot?

Left hind limb combination lameness often generates the appearance that the right front limb is being "pushed into" a hole (from behind).

NO There is no referred component to the LH lameness.

YES There is a concurrent lameness in the LF limb. Consider the possibility that the LF lameness is referred from the LH (as a result of shifting weight and accommodating stride length).

D. Assess Right Hind (RH) Limb Soundness

Determine Location and Nature of Lameness

1. Is there a RH limb weight-bearing lameness?

Look at the LH limb: Is it falling into an imaginary hole?

Right hind weight-bearing lameness looks as if the horse is stepping into a hole with the left hind foot.

NO There is not a weight-bearing lameness in the RH limb.

YES There is a weight-bearing lameness in the RH limb.

Determine Severity of Lameness

How deep is the hole?

The deeper the imaginary LH hole, the more severe the RH lameness.

Establish the Primary Source of Lameness

Is there a concurrent RF limb weight-bearing lameness?

Look at the LF limb: Is it falling into an imaginary hole?

Right front weight-bearing lameness looks as if the horse is stepping into a hole with the left front foot.

NO

YES There is a concurrent weight-bearing lameness in the RF limb. Consider the possibility that the RF lameness is referred from the RH (as a result of shifting weight onto the LF limb).

Is there a concurrent weight-bearing lameness in the LF limb?

Left front weight-bearing lameness looks as if the horse is stepping into a hole with the right front foot.

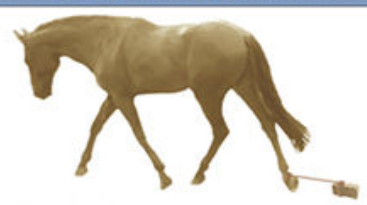
NO RH lameness is likely primary.

YES Consider the possibility that the RH lameness is referred from the LF (as a result of shifting weight to the opposite diagonal pair), especially if LF lameness is considerably more severe than RH lameness.

Determine Location and Nature of Lameness

2. Is there a RH limb non weight-bearing lameness?

Look at the RH limb: Is it dragging an imaginary brick?



Right hind non weight-bearing lameness looks as if the horse is dragging a brick with the right hind pastern.

NO There is not a non weight-bearing lameness in the RH limb.

YES There is a non weight-bearing lameness in the RH limb.

Determine Severity of Lameness

How heavy is the brick?

The heavier the imaginary RH brick, the more severe the RH lameness.

Establish the Primary Source of Lameness

Is there a concurrent LF limb non weight-bearing lameness?

Look at the LF limb: Is it dragging an imaginary brick?

Left front non weight-bearing lameness looks as if the horse is dragging a brick with the left front pastern.

NO

YES There is a concurrent non weight-bearing lameness in the LF limb. Consider the possibility that the LF lameness is referred from the RH or vice versa (to maintain diagonal synchrony).

Is there a concurrent non weight-bearing "skip" associated with the RF limb?

Right front "skipping" looks as if the horse is attempting to pick up the left-lead canter in front while trotting behind.

NO RH lameness is likely primary with no referred component.

YES Consider the possibility that the RF lameness represents a "thoracic skip" referred from RH biomechanical interference.

Determine Location and Nature of Lameness

3. Is there a RH limb combination lameness?

Look at the LH and RH limbs: Is the LH falling into an imaginary hole and the RH dragging an imaginary brick?

Right hind combination lameness looks as if the horse is dragging a brick with the right hind pastern and stepping into a hole with the left hind foot.

NO There is not a combination lameness in the RH limb.

YES There is a combination lameness in the RH limb.

Determine Severity of Lameness

How deep is the hole?

The deeper the imaginary LH hole, the more severe the weight-bearing component.

How heavy is the brick?

The heavier the imaginary RH brick, the more severe the non weight-bearing component.

Establish the Primary Source of Lameness

Is there a concurrent RF limb lameness? Does the horse appear as though it is being "pushed into" a hole under the LF foot?

Right hind limb combination lameness often generates the appearance that the left front limb is being "pushed into" a hole (from behind).

NO There is no referred component to the RH lameness.

YES There is a concurrent lameness in the RF limb. Consider the possibility that the RF lameness is referred from the RH (as a result of shifting weight and accommodating stride length).

28.2 Lameness Traits Summarized

	Weight-Bearing Deficits	Non Weight-Bearing Deficits	Combination Deficits
Left Front	 Left front weight-bearing lameness looks as if the horse is stepping into a hole with the right front foot.	 Left front non weight-bearing lameness looks as if the horse is dragging a brick with the left front pastern.	 Left front combination lameness looks as if the horse is dragging a brick with the left front pastern and stepping into a hole with the right front foot.
Right Front			

	 Right front weight-bearing lameness looks as if the horse is stepping into a hole with the left front foot.	 Right front non weight-bearing lameness looks as if the horse is dragging a brick with the right front pastern.	 Right front combination lameness looks as if the horse is dragging a brick with the right front pastern and stepping into a hole with the left front foot.
Left Hind	 Left hind weight-bearing lameness looks as if the horse is stepping into a hole with the right hind foot.	 Left hind non weight-bearing lameness looks as if the horse is dragging a brick with the left hind pastern.	 Left hind combination lameness looks as if the horse is dragging a brick with the left hind pastern and stepping into a hole with the right hind foot.
Right Hind	 Right hind weight-bearing lameness looks as if the horse is stepping into a hole with the left hind foot.	 Right hind non weight-bearing lameness looks as if the horse is dragging a brick with the right hind pastern.	 Right hind combination lameness looks as if the horse is dragging a brick with the right hind pastern and stepping into a hole with the left hind foot.

SECTION VII

The Gait Signature

Once the horse's deficits have been individually identified, the observer can build a gait profile (or "lameness ID") for the animal based on the visual data that was retrieved during the assessment process. The collection and characterization of various aberrations denotes a gait signature, which is basically an itemized description of the horse's movement at any given time. In the sound horse, the gait signature can function as a benchmark for future visual comparison(s).

29
Designation of the Horse's Gait Signature

Now it's time to make sense of our observations. For the budding observer, this is most easily accomplished via organized documentation of impressions amassed during visual assessment (fig. 29.1).

Of course, individual observers can amend these notes or create their own designations based on how each perceives lameness. In the end, most of us should reach similar conclusions with regard to our observations and their potential clinical significance.

29.1 Sample Classification of Gait Characteristics

Classification	Limbs Affected	Laterality of Lameness	Nature of Lameness	Gaits Most Affected	Surface Influence	Directional Influence	Unique Traits	# Deficits Observed
A	Thoracic	Unilateral	WB	At Rest, Walk, Trot	Worse on Hard	Worse to Inside	Significant head excursion, asymmetrically lands or bears weight on foot	31
B	Thoracic	Bilateral	WB	Walk, Trot	Worse on Hard	Worse to Inside	Significant head excursion, premature cranial phase to stride, cranial phase of stride is longer than caudal phase, lands toe-first, tripping, pointing	13
C	Thoracic	Bilateral	WB	At Rest, Walk, Trot	Worse on Hard	Worse to Inside	Premature cranial phase to stride, cranial phase of stride is longer than caudal phase, lands heel-first, pointing, treading	4
D	Thoracic	Unilateral or Bilateral	WB	Trot	Worse on Hard	Worse to Outside	---	33
E	Thoracic	Unilateral	WB and NWB	Trot	Worse on Soft	---	Shortened stride length, foot lands toe-first	8
F	Thoracic	Unilateral or Bilateral	WB and NWB	Trot	---	Worse to Outside	Delayed cranial phase to stride, shortened stride length	13
G	Thoracic	Unilateral or Bilateral	NWB	Walk, Trot	Worse on Soft	Worse to Outside	Delayed cranial phase to stride, shortened stride length, hypometric	9
H	Thoracic	Unilateral	WB and NWB	Walk, Trot	---	---	Caudal phase of stride is longer than cranial phase	1
I	Thoracic	Unilateral	WB and NWB	Walk	---	---	Dropped elbow, limb is held underneath body	1
J	Thoracic	Unilateral	NWB	Trot	Worse on Soft	Worse to Outside	Significant head swing, delayed cranial phase to stride, shortened stride length, hypometric	4
K	Thoracic	Unilateral	NWB	Trot	---	Worse to Outside	Head swing, delayed cranial phase to stride, shortened stride length, hypometric, holds neck lower than normal	3
L	Pelvic	Unilateral	WB	Walk, Trot	Worse on Hard	---	---	1
M	Pelvic	Unilateral	WB	Walk, Trot	Worse on Hard	Worse to Inside	Sticks toe into footing	1
N	Pelvic	Bilateral	WB	At Rest, Walk, Trot	Worse on Hard	---	Pelvic limbs camped underneath body, cranial phase of stride is longer than caudal phase, treading	1
O	Pelvic	Unilateral	WB and NWB	Walk, Trot	Worse on Soft	Worse to Outside	Sticks toe into footing	14
P	Pelvic	Unilateral or Bilateral	WB	Walk, Trot	---	Worse to Inside	Limb adduction during cranial phase of stride followed by sudden abduction, lateral rotation of the tarsi under weight-bearing load, excessive varus deformation (bowing) of tarsi under weight-bearing load	120
Q	Pelvic	Unilateral	WB and NWB	Walk, Trot, Canter	Worse on Soft	---	Shortened stride length, hypometric, sticks toe into footing	11
R	Pelvic	Unilateral	NWB	Trot, Canter	Worse on Soft	Worse to Outside	Delayed cranial phase to stride, shortened stride length, hypo-metric, sticks toe into footing	36
S	Pelvic	Bilateral	NWB	Canter, Canter Depart,	---	Worse to Outside	Delayed cranial phase to stride, shortened stride length, hypometric, sticks toe into footing	16

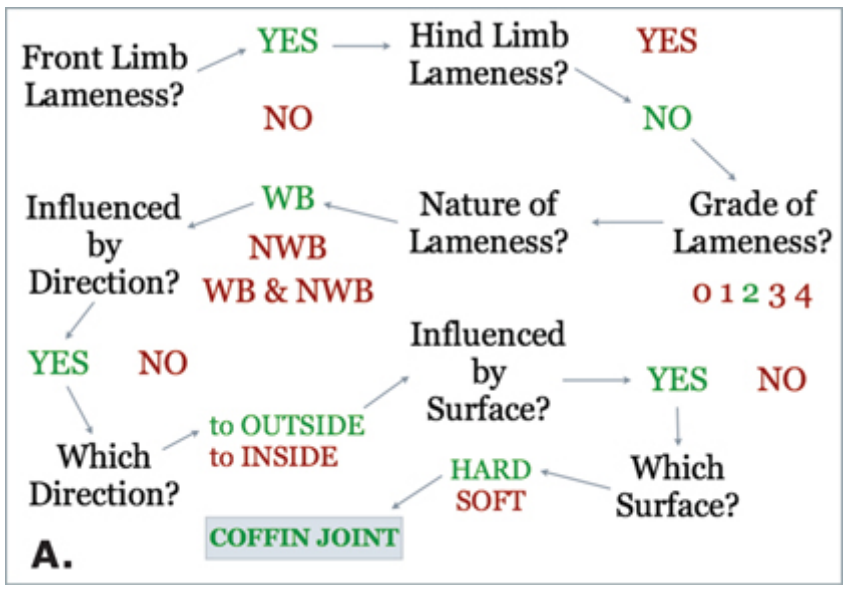
				Downward Transitions				
T	Pelvic	Bilateral	NWB	Canter, Canter Depart, Downward Transitions	---	---	Fixed extension of the hind limb, involuntary and sudden hyperflexion occurs at initiation of cranial phase of stride	16
U	Pelvic	Unilateral	WB	Walk, Trot, Canter	---	---	External rotation of the limb (hock-in and toe-out), cranial phase of stride is longer than caudal phase	13
V	Pelvic	Unilateral	NWB	Walk	---	---	Stifle flexes during cranial phase of stride but hock remains in extension	1
W	Pelvic	Unilateral or Bilateral	NWB	At Rest, During Backing	---	---	Prolonged hyperflexion of the limb with concurrent tail raising	1
X	Pelvic	Unilateral	NWB	Walk	---	---	Shorten cranial phase to stride, goose step	3
Y	---	---	---	---	---	---	LAMENESS NOT DETECTED	1
							TOTAL DEFICITS OBSERVED	**355**

30
Correlating the Gait Signature with Likely Sources of Lameness

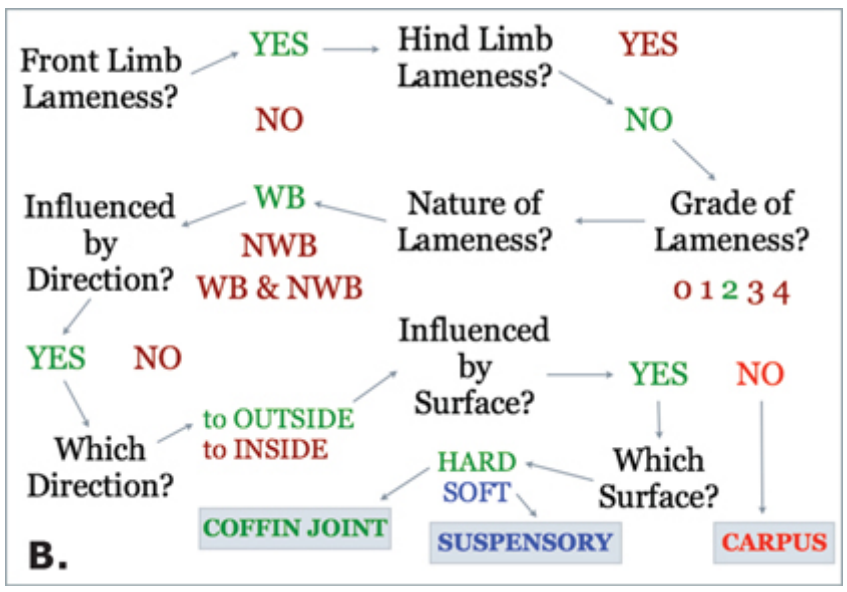
Despite the lack of specificity and objectivity associated with visual assessment, the examiner can still assign a number of visible aberrations in gait to consistent sources of lameness with some degree of precision. Classification and categorization of abnormal motion patterns can assist in the establishment of cursory relationships between visual impression and clinically significant pathology.

We utilize a similar *question and answer method* to that employed by the "20 Questions" game (see section VI, p. 157) when filtering our observations. We simply ask ourselves a series of visual questions relating to the horse's movement. Then we use a process of elimination to arrive at likely answers to these questions, which in our context are likely sources of lameness. Figure 30.1 A illustrates implementation of this technique during the visual assessment of a case study (**VL 30a**). This example highlights visible gait deficits that are symptomatic of forelimb coffin-joint pain.

30.1 Employing the Question and Answer Method to Expose Probable Causes of Lameness



A. You observe the horse in systematic fashion, querying yourself along the way. Your visual impressions allow you to answer one question at a time. In this sample case, your observations have led you to implicate coffin-joint disease as a likely cause of the horse's lameness. (WB = weight-bearing and NWB = non weight-bearing.)



B. Answering just one question (relating to the visual interpretation of surface influences) differently would lead to you considering alternate causes for the horse's lameness.

VL 30a
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Of course, if we had answered just a couple of questions differently our impressions might have directed us toward other potential sources of lameness (fig. 30.1 B).

In this sample study, the deciding factor between three potential sources of lameness was surface influence. We didn't discern any unique traits to the horse's gait, but in this instance it wasn't necessary.

As mentioned a number of times in this manual, the goal of visual assessment of the lame horse is *not* to procure a diagnosis of the problem. Rather, it is to facilitate and expedite the diagnostic process by showing the observer *where to look* for the problem. Appropriately, the significance of our observations can only be validated through ultimate veterinary diagnosis (fig. 30.2). In essence, having a diagnosis enables us to further define the implication of our impressions (retrospectively), which in turn improves their future diagnostic value.

30.2. Veterinary Validation of Visual Impressions

Veterinary diagnosis is required to validate your visual impression of gait abnormalities.

The clinical significance of the sample gait signatures listed in Figure 29.1 (see p. 212) can be judged based on the eventual diagnoses of a group of animals that displayed comparable traits (fig. 30.3).

30.3 Sample Correlation of Gait Deficits with Veterinary Diagnoses

Classification	# Deficits Observed	Remote (Regional)	On-Site Diagnosis	Remote Observations Validated	Remote Observations Invalidated	% Validated
A	31	Foot abscess Foot bruise P3 fracture	Navicular inflammation=1 Foot abscess=18 Foot bruise=4 P3 fracture=1 Laminitis=1 DIP joint=6	23	8	74.19
B	13	Thoracic navicular inflammation	Navicular inflammation=8 Foot bruise=1 Laminitis=1 DIP joint=4	8	5	61.54
C	4	Thoracic laminitis	Navicular inflammation=2 Laminitis=2	2	2	50
D	33	Medial foot abscess Medial foot bruise DIP joint	Medial foot abscess=5 Medial foot bruise=7 Medial hoof wall crack=2 Laceration/trauma of medial heel bulb=1 Medial heel quarter keratoma=1 DIP joint=17	33	0	100
E	8	Flexor tendons Suspensory ligament Palmar digital sheath	Flexor tendon(s)=4 Suspensory desmitis=2 Palmar digital tenosynovitis=2	8	0	100
F	13	Fetlock joint	Fetlock=9	9	4	69.23

			Radiocarpal joint=3 Flexor tendons=1			
G	9	Carpus	Fetlock=3 Radiocarpal joint=1 Midcarpal joint=2 Both radiocarpal/midcarpal joints=3	6	3	66.67
H	1	Elbow joint Radial nerve paresis	Elbow joint=1	1	0	100
I	1	Olecranon fracture Radial nerve paralysis	Radial nerve paresis=1	0	1	0
J	4	Bicipital bursitis	Bicipital bursitis=3 C5-6 arthrosis=1	3	1	75
K	3	Caudal cervical area	Bicipital bursitis=1 C5-6 arthrosis=1 C6-7 arthrosis=1	2	1	66.67
L	1	Foot abscess Foot bruise P3 fracture	Foot bruise=1	1	0	100
M	1	Heel abscess Heel bruise	Medial heel bulb bruise=1	1	0	100
N	1	Pelvic laminitis	Pelvic laminitis=1	1	0	100
O	14	Fetlock joint Flexor tendons Suspensory ligament Plantar digital sheath	Flexor tendon(s)=2 Palmar digital tenosynovitis=4 Femoropatellar joint=6 Femorotibial joint=2	6	8	42.86
P	120	Distal tarsal joints	Distal tarsitis=120	120	0	0
Q	11	Femorotibial joint	Femorotibial joint=6 Femoropatellar joint=5	6	5	54.55
R	36	Femoropatellar joint	Plantar digital tenosynovitis=2 Suspensory desmitis=2 Femoropatellar joint=24 Proximal patellar hesitation=8	24	12	66.67
S	16	Proximal patellar hesitation	Femoropatellar joint=5 Proximal patellar hesitation=11	11	5	68.75
T	16	Upward patellar fixation	Upward patellar fixation=16	16	0	100
U	13	Pelvis Coxofemoral joint Greater trochanteric bursa	Distal tarsitis=11 Coxofemoral joint=1 Greater trochanteric bursitis=1	2	11	15.38
V	1	Peroneus tertius rupture	Rupture of the peroneus tertius tendon=1	1	0	100
W	1	Shivers	Shivers=1	1	0	100
X	3	Pelvic fibrotic myopathy	Semitendinosus fibrotic myopathy=2 Semimembranosus/Gracilis fibrotic myopathy=1	3	0	100
Y	1	LAMENESS NOT DETECTED	CI dorsal osteitis=1	0	1	0
TOTAL	355			288/355	67/355	81.13

Nowadays, young veterinarians may be less inclined to spend a tremendous amount of energy decoding the visual expression of the horse's gait, especially when they have recently invested in expensive diagnostic imaging equipment. Neglecting any portion of the visual examination, however, can convolute the interpretation of findings acquired via other diagnostic modalities. Seasoned veterinarians will regularly apply their visual impressions to the diagnostic process, particularly in cases in which multiple forms of pathology emerge upon diagnostic imaging (e.g. radiography, ultrasonography, MRI). For instance, the clinical significances of ultrasonographic abnormality associated with the proximal suspensory ligament of the hind limb in a horse, and radiographic abnormalities associated with the lower tarsal (hock) joints in the same horse can be further defined via the visual assessment of the horse's gait: Proximal suspensory desmopathy customarily generates combination deficits that are accentuated when along the *outside* of a circle whereas distal tarsitis (lower hock pain) most often produces weight-bearing deficits that are exacerbated when along the *inside* of a turn or circle.

SECTION VIII

Applying Our Observations to the Diagnostic Process

The purpose of visual observation is not to diagnose the horse's lameness. Rather, it is merely intended to help us recognize asymmetry and to point us in a relevant diagnostic direction: "Where do we need to look for the problem?" As has been mentioned throughout the course of this book, knowing where to look for the problem makes it much easier to find. Simply recognizing lameness satisfies the horse owner's responsibility. The rest of the investigation is left to the veterinarian who, with increasing competence, will further "decode" the horse's movement visually so as to avoid over-relying on diagnostic imaging modalities in an attempt to generate a diagnosis.

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Visual Observation as Part of the Diagnostic Workup

The lack of specificity associated with processing visual impressions coupled with the fact that similar gait deficits exist for a variety of problems makes observation alone an impractical strategy for reaching or confirming a diagnosis of lameness, except in horses exhibiting a single pathognomonic abnormality (such as obvious goose-stepping in the hind limb). Differentiating between multiple problems within the foot, for example, is very difficult when based solely on visual impression(s). That said, distinguishing between problems in the foot and those above the fetlock joint can be relatively straightforward. Although relatively imprecise, this form of "regionalized" information can prove valuable in a number of circumstances:

• *Preemptive screening prior to hands-on veterinary examination.* Veterinarians who have some idea of the degree and nature of lameness are better prepared to perform forthcoming clinical evaluation. For example, a practitioner scheduled to visit a horse exhibiting acute (sudden) grade 4/5 right front weight-bearing lameness that was shod five days previously is likely going to be treating an abscess in the foot.

• *Differentiating between primary and secondary issues.* Exhaustive investigation and local treatment of secondary lameness are very common, marginally beneficial, and often unnecessary. We might detect clinical and ultrasonographic abnormalities associated with the horse's *longissimus* muscle along the thoracolumbar region, for example, but if it is secondary to chronic hind-limb lameness then the effect of any back treatment(s) will be relatively short-lived. As previously mentioned, secondary lameness often draws undue attention if it is more obvious than its underlying primary source.

• *Clarification of diagnostic imaging findings.* Nowadays, the implementation of one or more diagnostic imaging modalities (such as radiography, ultrasonography, or MRI) is an expected facet of the veterinary lameness workup. The improved image resolution afforded by modern equipment has enabled professionals to detect minuscule changes in the horse's structural anatomy. For the average veterinarian, the hardest part of the examination is often deciding which changes are clinically significant and which are not. Any intelligence gathered during visual assessment and clinical examination can help to direct the remainder of the workup appropriately. For example, the significance of radiographic changes associated with the left front pastern joint should be questioned if the horse's lameness stems from a different limb altogether (the primary lameness is elsewhere).

• *Follow-up veterinary assessment.* Regular observation allows veterinarians to track the horse's post-treatment progress more precisely over time. Appropriate response of primary issues to recommended therapy can be confirmed. Secondary issues can also be monitored; those that persist for a prolonged period may not spontaneously resolve pursuant to successful treatment of the primary inciter. If given enough time, secondary problems (like primary problems) can develop irreversible pathology that must be treated explicitly. In this context, secondary lameness has the potential to evolve into primary lameness and should, therefore, also be surveyed on a regular basis.

• *Sharing of observations between multiple individuals.* The horse's gait signature can function as a point of collaboration between horse enthusiasts. Denoting a lameness as "right front grade 3/5 weight-bearing lameness worse to the left" is considerably more illuminating than "right front lameness."

In addition to enhancing and clarifying the diagnostic process, visual assessment can also simplify it. Those of us who regularly and carefully observe our horses in motion will see fewer multifactorial cases, because we are more likely to depict gait abnormalities soon after their onset and before additional primary issues have had time to develop. We are also less likely to observe compensatory lameness for the same reason. Unless due to a common injury, it is relatively rare for two separate unrelated problems to occur simultaneously. In this context, regular observation actually simplifies the diagnostic process by making multifactorial and complicated lameness less probable.

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Building and Sharing Your Gait Signature Library

Accurately classifying and correlating gait deficits with potential sources requires that we document and archive our impressions for future reference.

Video Archiving

It is advantageous to acquire video footage of your subject(s) when possible. This not only permits more intent motion analysis (via the use of software when applicable), but also provides a cinematographic record of the animal's movement. This record can be shared amongst a group of individuals and/or stored for future reference.

Most veterinarians maintain radiographic, ultrasonographic, scintigraphic (etc.) images for documentation purposes. Equine surgeons will often include cinematographic clips of endoscopic and arthroscopic procedures in their patients' files. Video footage of the horse in motion, however, is a diagnostic image that has been largely overlooked up to this point.

Successful lameness management over the long term is facilitated through the establishment and maintenance of video records. Preemptive acquisition and storage of "sound" or "benchmark" footage is especially valuable to the observer who is currently assessing a horse for lameness.

Written Reports

A brief description of the horse's lameness including type, nature, degree, distinct deficits, and any unique responses to environmental manipulation should be included with the animal's evaluation record. The adept observer can envision the horse's movement simply by reading this form of report. For example, an observer currently assessing a horse that previously exhibited right front grade 3/5 weight-bearing lameness worse to the left will know immediately if the animal's current asymmetry is a result of the same or a different pathologic source. Even if the same pathology is implicated as the cause, any prevailing disparities in the degree of lameness and/or the quality of deficits will be obvious.

Include the Diagnosis

The diagnosis, once obtained by the attending veterinarian, provides us with the opportunity to translate visual impressions into meaningful clinical information. This form of translation is known as *clinical reasoning*. Once interpretative "rules" have been established for an observer, a more educated approach to future visual evaluation is possible. Procuring the diagnosis enables the observer to work "backward" to discern any visible gait deficits that were also displayed by other horses suffering from the same affliction(s). Over time, explicit patterns of movement can be assigned clinical relevance with increasing confidence, thereby aiding in the future recognition of pathology.

Sharing Records

Current technology allows for easy storage and seamless sharing of electronic records between individuals physically removed from the horse and each other. Pooling our video footage (with annotations) provides access to a large number of cases, thereby equipping each observer with the means to refine their subjective diagnostic accuracy.

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The Future of Visual Assessment and Gait Signature Characterization in the Diagnosis and Management of Equine Lameness

Most modern equine veterinary practices and teaching hospitals comprise specialized departments tailored to facilitate lameness diagnosis and treatment. Practitioners are invariably upgrading the diagnostic modalities and therapeutic methodologies utilized in equine sports medicine. While great advancements have been made in these fields, the professional approach is still somewhat *reactive* in nature. Many performance horses are diagnosed after the window of opportunity for effective treatment has already passed. This is because problems are often first noted long after their inception.

The implementation of *equine sports wellness programs* devised to actively *seek out* patient lameness will enable equine professionals to adopt a *proactive* approach to performance management. This strategy should, in turn, improve our horses' prognosis for future success on the racetrack, in the show ring, and on the trail. As was discussed in the early chapters of this book, early recognition is *integral* to the effectual management of equine soundness over the long term.

At the moment, the horse's primary caretaker is best situated to perceive lameness at or shortly after its onset. Expedited recognition will, therefore, necessitate local corroboration in one form or another:

- Enhancing the "visual intelligence" of the local caretaker.
- Accelerating communication between the local caretaker and veterinarian.

The latter objective could be accomplished in one of two ways:

1. Scheduling regular "soundness checkup" appointments with the veterinarian. This option may not be feasible for the average horse owner due to restrictions imposed by time, distance, and finances.

2. Streamlining video correspondence between the local caretaker and veterinarian. Current technology makes this option nearly effortless for both parties involved.

The future development of lameness recognition and visual characterization may likely entail a combination of improved client education, client dialogue integration on the veterinary practice model, digital dynamic tracking of the horse's movements, and digital analysis of the gait signature.

Phase I. Improved Client Education

Many practices and institutions have already implemented education curricula targeted at improving their clients' "eye for lameness." Regular administration of seminars, short courses, wet labs, online tutorials, hand-out distribution, and so on have improved professionals' chances of interfacing with lame horses before their treatment becomes less rewarding.

Phase II. Client Dialogue Integration into the Veterinary Practice Model

Recent advancements in telecommunication permitted seamless sharing of video footage between individuals in remote locations. Current technology allows the veterinarian to quickly and inexpensively perform the following:

- Pre-purchase screening assessment.
- Pre-appointment assessment.
- Post-appointment (recheck) evaluation.
- Regular (wellness) soundness inspection.

The dramatic increase in evaluation efficiency afforded by telemedical observation allows the equine veterinarian to provide an attentive, proactive approach to managing and preventing lameness. Progress can be assessed on a regular basis with very little investment of time and money on behalf of the practitioner. This translates into a better overall service to the client. Although telemedical assessment comprises only one facet of comprehensive lameness investigation, its implementation can indubitably enhance the diagnostic proficiency of the equine performance veterinarian.

Phase III. Digital Dynamic Tracking of the Horse's Movements

Motion sensing and analysis software designed to track and analyze the horse's movements in a spatial setting currently exists.[26] Products implementing this technology, however, have to be physically applied by a medical professional onsite.

The development of software aimed at tracking specific anatomic movements of the horse in a cinematographic setting is currently underway. By tracking specific features of the horse as it moves across the video screen, this tool will permit self-generation of one or more wave patterns. The character of each wave coupled with its relationship to coexisting waves can be used to create an *electronic* gait signature. Tangible, storable gait signatures favor more objective pattern characterization and grade quantification amongst a group of observers.

Since the latter technology is applied to video footage rather than live animals, this form of analysis can be performed from anywhere. Consequently, it can be implemented with greater frequency and ease as compared to contemporary modalities.

Phase IV. Digital Analysis of the Gait Signature

Once a digital gait signature is generated, it is logical to presume that software can be used to analyze its traits and deduce consistent and reproducible conclusions with regard to potential clinical implications. This technology would provide a means for the equine professional to translate visual pattern recognition into something that could be interpreted in a consistent and objective manner.

Deliberate and progressive documentation, refinement, and allocation of the patent details of reproducible gait patterns is essential to advancing the visual recognition and characterization aspects of equine lameness management. The advent of digital analysis, storage, and communication technologies makes this venture highly feasible in sport horse practice.

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About the Author

Photo by Kate Schultz

Dr. Bob Grisel began his equine veterinary training at the age of 13 when he worked for a racetrack veterinarian near his home in south Florida. Bob quickly developed an interest in solving the many problems that affect performance horses. He dedicated the next 30 years of his life to learning as much as he could about sport horse medicine and surgery.

During high school and college, Bob spent every moment that he wasn't in the classroom working within the equine veterinary field. He spent the majority of his time at racetracks in Florida and New York, as well as gaining surgical experience at two referral facilities in Colorado.

Bob graduated from the University of Florida's College of Veterinary Medicine with honors and received multiple awards for his performance in equine surgery while enrolled as a student. Bob then moved to southern California where he received extensive training in equine surgery and radiology under the tutelage of a world-renowned veterinary staff.

Upon completing a residency in equine surgery with an emphasis on arthroscopy, Bob was recruited by Oregon State's College of Veterinary Medicine. He and his family moved to Corvallis where Bob spent time on the large animal teaching staff at OSU. Bob's surgical interests focused on performance-related problems, and he developed and published many surgical techniques designed to better treat the equine athlete. Such techniques include ultrasound-guided arthrocentesis of the equine cervical facets and arthroscopic treatment of solitary osteochondromas in the horse.

Bob was offered an equine surgical position in the Atlanta area while working the 1996 Olympic Games. Shortly after relocating to Georgia, Bob built The Atlanta Equine Clinic, a state-of-the-art, full-service equine diagnostic, surgical, and emergency care facility located in Braselton. Bob performed surgery, diagnostic imaging, and lameness evaluations for the clinic, which served a diverse clientele.

Bob's interest in equine lameness and surgery continued to flourish at a rate commensurate with the increasing number of performance-related cases referred to his practice. In 2005, Bob left the hospital facility to devote 100 percent of his energy toward equine sports medicine.

Bob has since developed a reputation for his meticulous and accurate diagnostic ability, as well as for his effective treatment strategies for performance-related problems in the horse. He is regularly consulted on issues concerning lameness and prepurchase examinations worldwide.

Bob's recent publications illustrate the correlation between abnormal gait characteristics and specific sources of pain/lameness in the horse. In addition to his book and accompanying video channel, he offers "Lameness for the Layman" short courses to enhance the horse owner's awareness of equine lameness, as well as improve one's ability to identify likely causes.

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