A Look at Lameness Through the Eyes of Functional Anatomy (and Biomechanics)

J.-M. Denoix, DVM, PhD, HDR, Founder ISELP, LAIA-ECVDI, DACVSMR, DECVSMR, INRAE

The purpose of this paper is to show how the clinical manifestations of lame horses are correlated to functional anatomy and internal biomechanical data. A lot of information can be obtained from visual analysis of lame horses under standardized situations combining different gaits, ground surfaces and exercises. These data are not only useful for diagnostic purposes but are key to evaluating the significance of imaging findings and to establish an adequate rehabilitation program avoiding uncomfortable situations for the horse. In practice, analysis of video-recordings (at normal speed or slow motion) is recommended to confirm, complete, or extend analysis of clinical manifestations. More sophisticated methods have been used or are in development to give further objective data especially at the trot, but as the vast majority of functionally normal horses present gait asymmetries, there is a need for establishing the criteria to discriminate between physiological lateralization and lameness. Author’s address: CIRALE-NEV-EnvA, Goustranville, F-14430, France; e-mail: jean-marie.denoix@vet-alfort.fr. © 2021 AAEP.

1. Introduction

The diagnosis of lameness has been considered for a long time in many textbooks and papers, and symptoms as well as causes of musculoskeletal injuries have been extensively discussed and debated.1–11 Evaluation of the horse at the trot combined with diagnostic analgesia has been widely used, investigated, and recommended and is generally considered as the gold standard approach for establishing the origin of pain.12–14 Nowadays, the extraordinary development of imaging techniques and technologies has permitted the diagnosis and documentation of an unlimited number of causes of lameness. Constant improvement and increased availability of these techniques for field practice have enabled the practitioner to reach a more precise identification and documentation of lesions and abnormalities with the conviction or the hope for a better management of the affected horse. The drawback of having access to a variety of more sensitive techniques is the risk of overinterpreting imaging findings and overestimating their significance; it is also to bypass the essential step that consists of establishing the correlation between the clinical presentation and the lesion(s). The purpose of the pre-sentation is to demonstrate how a horse alters its gait to decrease the pain induced by each type of condition. The basic concept presented in this paper is that gait alterations (lameness) are a live expression and the reflection of functional anatomy and biomechanics, as the horse changes its gaits to reduce the biomechanical stresses on painful anatomical structure(s). Typical alterations of the gait pattern at the walk, trot, and canter evaluated in different situations and exercises are presented in this paper to show how lame horses illustrate the distribution of biomechanical stresses on musculoskeletal lesions and to demonstrate that, from a diagnostic point of view, the type of lameness is more important than the grade. The format of this paper consists of following each step of the dynamic
examination and to analyze gait and movement alterations of different types of lameness in the light of functional anatomy and biomechanics. A number of studies have been done to compensate the limitations of the subjective evaluation of lameness. Nevertheless, an interesting debate arose a few years ago about the use of quantitative gait analysis and the significance of the objective technological data obtained. Another objective of this paper is to demonstrate that there is still room for a visual blocking of proximal regions is not possible. But functional (and physical) asymmetry needs to be identified as it can predispose to secondary problems such as tendon disease on the limb carrying more weight. Differentiation between lameness and asymmetry or laterality is especially crucial when performing a single examination such as in a pre-purchase examination. It is sometimes difficult to conclude in this context and reexamination of the horse at its intended level of use is indicated to reinforce the decision. Origins of physical and functional asymmetry include the consequences of developmental conditions, sequelae of old trauma, and morphofunctional asymmetry of the nervous system command and control. Laterality in horses has been related to morphological, behavioral, occupational, neurological, or gender factors. Depending on the origin of the lameness, a differential diagnosis has sometimes been suggested to distinguish pain manifestations from mechanical lameness and neurological problems. However, in many horses two of these components are combined and are integrated in the diagnostic approach. Musculoskeletal lameness and neurological conditions are sometimes considered as separate conditions and inter-observer agreement of neurological manifestations is poor. But, especially in the caudal cervical and lumbosacral areas, intervertebral arthropathies induce pathological and functional alterations of the corresponding spinal nerves (or segments) and clinical manifestations are a mixture of ostearticular and neurological symptoms.

Lameness or Laterality?

In most cases, lameness is a clinical expression of pain, with the horse redistributing the load between limbs (kinetic aspect) and this results in asymmetrical gaits or movements (kinematic aspect). However, is asymmetry a lameness? Having examined sound and lame horses for more than 30 years, live and subsequently on video recordings, I still don’t know what a sound horse is. I have read numerous textbooks and an endless number of papers on different types and grades of lameness based on the severity of asymmetrical load distribution or manifestations, but I can hardly identify a physically and functionally symmetrical horse. Most of (if not all) the horses show some degree of asymmetry when examined at different gaits or at work. Asymmetry at the walk or at the trot is common and can be objectivated using gait analysis systems. Is it possible to discriminate a low grade of lameness from asymmetry in horses? Contrary to the majority of lameness, asymmetry is not pain related. Asymmetry is not improved with rest, it does not worsen with the level of exercise or when the athletic demand increases; it can be compatible with adequate performance and it sometimes improves with exercise. Therefore, recognition of asymmetry versus lameness is easier when doing longitudinal rechecks of horses over prolonged periods (up to several years). Clinically, there is no change after performing adequate diagnostic analgesia (although this procedure is not entirely sensitive as complete blocking of proximal regions is not possible). But functional (and physical) asymmetry needs to be identified as it can predispose to secondary problems such as tendon disease on the limb carrying more weight. Differentiation between lameness and asymmetry or laterality is especially crucial when performing a single examination such as in a pre-purchase examination. It is sometimes difficult to conclude in this context and reexamination of
Stance phase lameness conditions (or supporting limb or weight bearing lameness) are the most common ones and have been widely presented and discussed. The horse uses the inertia of its body mass to increase deceleration, bear more weight, and achieve greater propulsion from the sounder limb. On the lame limb, the horse reduces shock absorption (shock absorption lameness: decreased cranial phase of the stride), decreases load (mid-stance or weight bearing lameness), and/or reduces propulsion (propulsion lameness or push off lameness).

Swing phase lameness conditions (swinging limb lameness) are often underestimated. They show up during the pulling forward part (e.g., shoulder syndrome including scapulohumeral arthropathies and bicipital apparatus injuries), mid-swing (e.g., decrease flexion because of tendonitis of the extensor carpi radialis, fetlock partial ankylosis; or increase flexion angles: rupture of the extensor carpi radialis), and protraction (e.g., reduced protraction: shoulder syndrome, nerve and muscle paresis; increased swing of protraction: deficit of the digital flexor muscles).

Mixed lameness conditions are common. Even if the clearest manifestations happen during either the stance or the swing phases, many single causes of lameness may alter both (from prodotrochlear syndrome to shoulder problems; Fig. 2).

The primary or baseline lameness is not always the most obvious and the primary cause of a lot of flexor tendon or suspensory apparatus injuries must be searched for an adequate management of the horse.

Secondary lameness is also called complementary or compensatory lameness. Pain in one limb induces redistribution of the load on the other limbs. As a typical example, a superficial digital flexor (SDF) tendon injury on a forelimb can be the consequence of pain on the opposite forelimb or the opposite hindlimb. But, considering load redistribution between...
forelimbs and hindlimbs, compensatory lameness should not be confused with secondary lameness due to asymmetry. This paper focuses on qualitative data. When mentioned, the severity of the lameness is expressed using the AAEP grading scale, although this scale is mainly based on stance phase alterations and other grading systems have been used. For more clarity, the gait characteristics of the lame or lamest limb (referred to as the ‘lame limb’) are considered. In multiple limb lameness, the combination of single limb characteristics may vary extensively depending on the number of sites of pain or mechanical defects. Flexion tests and diagnostic analgesia are not considered in this paper. Horse management during examination in hand, on the lunge, or when the horse is ridden or driven is important for both horse and human safety but is beyond the scope of the paper.

2. Hard (or Firm) Surface

Examination at the Walk

A common limitation of the clinical assessment of lameness is to restrict the examination at the trot and to rapidly move on to diagnostic analgesia. Examination at the walk can provide valuable information and is easily accessible as the movements are slow (pedagogical didactic value). Besides, in very painful horses, this should be the only situation used to evaluate the lameness. As the walk is a slow gait with no suspension phase, there is less load impact and less maximum load at mid-stance phase, but there is an increased stance phase duration. Therefore, the clinical manifestations of a horse at the walk are more related to movement (kinematics) than to forces (kinetics).

Straight Line

Examination of the horse at the walk on a straight line on a hard and horizontal surface is probably the best situation to analyze the limb conformation, fetlock support, and foot landing. Looking at the horse from the front, with the horse coming toward the observer, is more reliable than the standing examination for assessment of limb conformation such as varus/valgus deformities of the carpus and fetlock. The cranial view is also useful for assessment of the foot landing and balance. Even moderate angular deformities have direct consequences on the load distribution within the joints and their identification is an essential contribution to the management and even the prevention of associated or secondary injuries. Besides, as alternate muscle contraction highlights left to right asymmetry, identification of myopenia (muscle atrophy) is easier and more reliable at the walk than during the static examination, especially on the hindlimbs.

Forelimbs. Stance phase (weight bearing) lameness conditions. To reduce the load absorption and maximum load on the affected limb, horses with very painful forelimb lameness demonstrate wide head oscillations (with higher velocity of the head dropping on the sounder limb) and decreased protraction during the swing phase (Fig. 3). Therefore, the affected limb shows a decreased fetlock extension during the stance phase and an increased fetlock extension on the sounder limb is observed. A hindlimb interference can be seen with increased protraction of the opposite hindlimb in an attempt to reduce the load on the affected forelimb. Horses with severe pain of the suspensory apparatus (e.g., proximal sesamoid bone fracture; Fig. 3) present a reduction of the cranial phase and an extended caudal phase of the stride. This is correlated to biomechanical data showing a higher tension of the suspensory apparatus and SDF tendon during the first part of the stance phase at the walk (Fig. 4). Several studies show that at the walk the peak of load on the deep digital flexor (DDF) apparatus (DDF tendon and its accessory ligament [AL-DF tendon]) takes place at the end of the stance phase. This explains why horses with severe pain in the DDF apparatus or in the podotrochlear apparatus (navicular syndrome) show a reduction of the caudal phase of the stride and...
a wider cranial phase (Fig. 5). The same manifestations can be seen with injuries of the DDF tendon in contact with a radial osteochondroma or a fracture of the olecranon providing attachment to the ulnar head of the DDF muscle.58 A foot abscess at the dorsal aspect of the sole may also induce shortening of the caudal phase of the stride as pressure increases over the toe area at the end of the stance phase.74 A decrease in fetlock suspension on the lame limb may be present in cases of failure of the suspensory apparatus or SDF tendon rupture or elongation. But because of the limited load during the stance phase at the walk, most of the horses with suspensory apparatus desmopathies or proximal suspensory ligament desmopathies/enthesopathies are asymptomatic at this gait. In some horses, the reduction of fetlock extension may be the only dynamic manifestation of suspensory disease at the walk (Fig. 6). Extension of the distal interphalangeal joint at the end of the stance phase with sudden elevation of the toe is pathognomonic of DDF tendon elongation or rupture. Shoulder instability is often clearer at slow gaits compared to faster gaits. Suprascapular nerve paralysis (sweeney syndrome) typically induces a varus of the shoulder joint worse at the end of the stance phase. Indeed, the lateral stability is no longer achieved by the infraspinalus muscle body eccentric and isometric contractions. This manifestation is accompanied by a reduction of the forelimb protraction (cranial part of the swing phase) as a result of a combined paresis or paralysis of the supraspinatus muscle making it unable to extend the shoulder joint during the swing phase.

**Swing phase lamenesses.** Two typical examples of swing phase lameness can be observed in clinical cases. Some young horses with lesions of the shoulder joint (osteochondrosis, osteochondrodysplasia, fracture of the tuberculum supraglenoidalis) show a marked decrease of protraction, wide head oscillations with a higher velocity during elevation of the head, synchronous of protraction of the affected limb. Pain during the swing phase is caused by the concentric contraction of the supraspinatus muscle extending the shoulder joint as a Type 1 lever arm; the pressure over the articular surfaces of the humeral head and glenoid cavity of the scapula are therefore increased (Fig. 7). This lameness also has a stance phase component characterized by lowering of the head on the sounder limb, reduced fetlock
extension, lengthening of the caudal part of the stride on the lamer limb, and hindlimb interference. Horses with injuries to the bicipital apparatus (biceps brachii, sulcus intertubercularis, bicipital bursa) also display a shortening of protraction and of the cranial phase of the stride. Horses with brachial plexus nerve root impingement caused by caudal cervical spine arthropathies may demonstrate moderate to severe reduction of the cranial phase of the stride due to paresis of the muscles achieving the protraction (Fig. 8). In most of these secondary neurological cases (with primary cervical arthropathies), there is less alteration of both head movements and fetlock suspension and reduction of the cranial phase of the stride is less visible at faster gaits. Developmental or acquired arthropathies of the caudal cervical articular process joints (C5-T1), responsible for brachial plexus nerve roots pathological and functional alterations are part of what can be called the equine cervicothoracobrachial (CTB) syndrome (Figs. 8–10). This syndrome overlaps what is described in human medicine for a long time as the cervicobrachial syndrome and the thoracic outlet syndrome, although horses lack a clavicle. Other lesions causing the same typical dyskinesia of protraction of the forelimb in horses include caudal cervical intervertebral disc lesions, arthropathy of the first costovertebral joint and congenital abnormalities of the first rib such as agenesis, hypoplasia, dystrophia, and synostosis with often incomplete and dystrophic fibrous union of the first two ribs. All these abnormalities or injuries are in close anatomical relation with the brachial plexus components, either with its nerve roots (ventral rami of C6 to T1 cervical spinal nerves at the level of the intervertebral foramen) or the brachial plexus itself, passing between the scalenus medius and the scalenus ventralis muscles and then crossing over and lying on the first rib. Resulting alteration of the nerve conduction is responsible for paresis of the muscles achieving the protraction. They include the supraspinatus muscle innervated by the suprascapular nerve (coming from the cervical nerves C6 and C7), the cleidobrachialis muscle innervated by the axillary nerve (coming from C7 and C8), and the biceps brachii and brachialis muscles innervated by the musculocutaneous nerve (coming from C6, C7, and C8) (Fig. 9). Involvement of the radial nerve roots (C7, C8, and T1) or pathway may induce forelimb protraction and of the cranial phase of the stride. Horses with brachial plexus nerve root impingement caused by caudal cervical spine arthropathies may demonstrate moderate to severe reduction of the cranial phase of the stride due to paresis of the muscles achieving the protraction (Fig. 8). In most of these secondary neurological cases (with primary cervical arthropathies), there is less alteration of both head movements and fetlock suspension and reduction of the cranial phase of the stride is less visible at faster gaits. Developmental or acquired arthropathies of the caudal cervical articular process joints (C5-T1), responsible for brachial plexus nerve roots pathological and functional alterations are part of what can be called the equine cervicothoracobrachial (CTB) syndrome (Figs. 8–10). This syndrome overlaps what is described in human medicine for a long time as the cervicobrachial syndrome and the thoracic outlet syndrome, although horses lack a clavicle. Other lesions causing the same typical dyskinesia of protraction of the forelimb in horses include caudal cervical intervertebral disc lesions, arthropathy of the first costovertebral joint and congenital abnormalities of the first rib such as agenesis, hypoplasia, dystrophia, and synostosis with often incomplete and dystrophic fibrous union of the first two ribs. All these abnormalities or injuries are in close anatomical relation with the brachial plexus components, either with its nerve roots (ventral rami of C6 to T1 cervical spinal nerves at the level of the intervertebral foramen) or the brachial plexus itself, passing between the scalenus medius and the scalenus ventralis muscles and then crossing over and lying on the first rib. Resulting alteration of the nerve conduction is responsible for paresis of the muscles achieving the protraction. They include the supraspinatus muscle innervated by the suprascapular nerve (coming from the cervical nerves C6 and C7), the cleidobrachialis muscle innervated by the axillary nerve (coming from C7 and C8), and the biceps brachii and brachialis muscles innervated by the musculocutaneous nerve (coming from C6, C7, and C8) (Fig. 9). Involvement of the radial nerve roots (C7, C8, and T1) or pathway may induce forelimb protraction and of the cranial phase of the stride. Horses with brachial plexus nerve root impingement caused by caudal cervical spine arthropathies may demonstrate moderate to severe reduction of the cranial phase of the stride due to paresis of the muscles achieving the protraction (Fig. 8). In most of these secondary neurological cases (with primary cervical arthropathies), there is less alteration of both head movements and fetlock suspension and reduction of the cranial phase of the stride is less visible at faster gaits. Developmental or acquired arthropathies of the caudal cervical articular process joints (C5-T1), responsible for brachial plexus nerve roots pathological and functional alterations are part of what can be called the equine cervicothoracobrachial (CTB) syndrome (Figs. 8–10). This syndrome overlaps what is described in human medicine for a long time as the cervicobrachial syndrome and the thoracic outlet syndrome, although horses lack a clavicle. Other lesions causing the same typical dyskinesia of protraction of the forelimb in horses include caudal cervical intervertebral disc lesions, arthropathy of the first costovertebral joint and congenital abnormalities of the first rib such as agenesis, hypoplasia, dystrophia, and synostosis with often incomplete and dystrophic fibrous union of the first two ribs. All these abnormalities or injuries are in close anatomical relation with the brachial plexus components, either with its nerve roots (ventral rami of C6 to T1 cervical spinal nerves at the level of the intervertebral foramen) or the brachial plexus itself, passing between the scalenus medius and the scalenus ventralis muscles and then crossing over and lying on the first rib. Resulting alteration of the nerve conduction is responsible for paresis of the muscles achieving the protraction. They include the supraspinatus muscle innervated by the suprascapular nerve (coming from the cervical nerves C6 and C7), the cleidobrachialis muscle innervated by the axillary nerve (coming from C7 and C8), and the biceps brachii and brachialis muscles innervated by the musculocutaneous nerve (coming from C6, C7, and C8) (Fig. 9). Involvement of the radial nerve roots (C7, C8, and T1) or pathway may induce forelimb
paresis (muscle weakness) further predisposing the horse to faulty steps, stumbling or even falling. These manifestations are often intermittent but because of the danger for the rider a special

attention should be given to the quality of forelimb protraction, which may be affected unilaterally or bilaterally. Swing phase lamenesses are rarely seen alone. With shoulder joint or bicipital apparatus injuries, there is often a stance phase component of the pain when load is put on the affected limb. For horses presenting a CTB syndrome, there is often an elongated caudal part of the stride compensating the shortened cranial phase (Fig. 8).

Hindlimbs. Weight bearing (stance phase) lamenesses. Looking at the hind fetlock suspension during the stance phase at the walk is essential as a decrease in suspension can be compensated with activation of the flexor muscles and tendons at faster gaits. The most common causes of this defect include degenerative suspensory apparatus lesions and sequela of juvenile tendon laxity. A unilateral or bilateral defect of fetlock suspension with a low pastern axis is seen in degenerative suspensory disease (in any location between its proximal insertion and its branches). It must be noted that a horse that is lame because of pain at the origin of the suspensory ligament can present with a defect of fetlock suspension despite a load reduction on the affected limb. These clinical manifestations are the result of the functional alteration created by the anatomopathological degeneration of the suspensory apparatus. A decrease in fetlock suspension can also be seen in horses presenting with SDF tendonitis or laceration, or proximal SDF tendon degenerative enthesopathy at the level of the supracondylar fossa of the femur. Contrary to horses with prominent degenerative suspensory disease, horses with painful proximal suspensory enthesopathies show a decreased fetlock extension. Many other conditions can be responsible for the reduction of fetlock extension during the stance phase at the walk. The differential diagnosis includes foot abscesses, fetlock problems (Fig. 11), severe suspensory apparatus pain, severe distal tarsus pain,
femorotibial joint pain, stress fractures, and others. Most of hindlimb lameness conditions induce a reduction of the cranial phase of the stride (clearer at the trot), but reduction of the caudal phase with lateral rotation of the limb can be seen in some conditions such as coxofemoral joint injuries (fracture of the acetabulum or of the femoral neck). This manifestation is correlated to the tension on the capitis and accessory ligaments of the hip joint that increases with medial rotation of the femur and decreases with lateral rotation. It is often associated with deviation of the pelvis and hind quarters, with the horse walking sideways. Fractures of the tuber coxae (and some fractures of the tuber ischiadicum) may induce a reduction of the caudal phase of the stride. The ventral displacement of the affected tuber coxae induces a shortening of the tensor fascia latae and a limitation to hip extension. Looking at the horse laterally at the walk is the best situation to assess the hindlimb sagittal conformation and identify the “straight hock-low fetlock” syndrome. Although this condition can be the result of congenital or juvenile tendon laxity, it is often seen in horses presenting with progressive degenerative suspensory ligament disease. It is sometimes considered erroneously that horses with straight hocks are predisposed to suspensory disease. Biomechanically, the pathophysiology of this syndrome follows this sequence: a degenerative suspensory disease induces a fetlock suspension defect; hyperextension of the fetlock increases tension on the SDF tendon, which pulls the tuber calcanei cranially and induces secondarily hyperextension of the hock. Therefore, a straight hock conformation is not a predisposing factor to suspensory disease but is just the result of an existing elongation of the suspensory ligament (Fig. 12). On the other hand, a defect of hock extension can be seen in horses presenting with a luxation of the SDF tendon from its normal position over the tuber calcanei. Twisted hock syndrome is better identified at the walk when the horse is examined from the rear. This condition shows up during the caudal phase of the stride and has been seen in horses with femorotibial problems. A potential cause is an asymmetric radius of curvature of the medial and lateral femoral condyles, inducing a combination of lateral displacement and medial rotation of the tibia. This results in a varus of the hock at the end of the stance phase. The manifestation is often reduced at the trot or at faster gaits. A defect of hock extension combined with hyperextension of the stifle and dorsal subluxation of the fetlock has been seen in horses with sciatic nerve paresis. Paresis of the gastrocnemius muscle is responsible for hock extension defect and hyperextension of the stifle, both inducing tension of the SDF tendon responsible for fetlock flexion. A defect of stifle extension during the stance phase is typical of femoral nerve paralysis which induces a paresis or paralysis of the quadriceps femoris muscle. This muscle becomes unable to achieve its eccentric contraction during the first part of the stance phase and its concentric contraction to open the stifle during the propulsion. Increased extension of the stifle may be secondary to osteochondrosis of the lateral trochlear ridge, with the horse displacing the patella proximally to avoid pressure on the lesion or to reduce patellar instability over a dysplastic femoral trochlea.

Swing phase lamenesses. The typical example of a hindlimb swing phase lameness is the rupture of the peroneus tertius. The affected horse shows a pathognomonic lack of hock flexion despite a hyperflexion of the stifle; there is no or little incidence on the stance phase. This mechanical lameness is induced by rupture or elongation of the peroneus tertius, making it no longer able to achieve the passive solidarization between stifle flexion and hock flexion during the swing phase. It must be added that as the hock no longer flexes, the calcaneus lever arm does not act anymore on the SDF tendon to induce flexion of the fetlock joint which remains extended. Different conditions cause hyperflexion of the hindlimb joints in different parts of the swing phase. Hindlimb joint hyperflexion after breakover is seen in horses presenting foot abscesses over the toe area. When pressure is concentrated on the dorsal part of the foot at the end of the stance phase, pain is responsible for sudden active flexion and elevation of the foot arch at the beginning of the swing phase. In case of upward fixation of the patella, a sudden spastic high velocity hyperflexion of the hindlimb can be seen during the first part of the swing phase when the patella is removed from the tuberculum of the femoral trochlea after a sudden lateral rotation induced by contraction of the gluteofemoralis muscle. Sudden reduction of protraction at the end of the swing phase with caudal retraction of the distal limb just before foot landing is typical of fibrotic myopathy of the semitendinosus muscle (Fig. 13). Other muscles such as the semimembranosus and the gracilis muscles can be

Fig. 12. Six-year-old Standardbred trotter gelding who presented a recurrence of severe third interosseous muscle (TIOM, suspensory ligament) rupture on April 4, 2019. Before this event (May 3, 2019, left image) hock and fetlock joint angulation was within normal limits. For several weeks after the rupture, there was a dropping of the fetlock and hyperextension of the tarsus. After enough healing with extensive scar tissue (July 10, 2019), there was a reduction of fetlock and tarsus extension.
affected with this condition and present similar manifestations. Although often considered as a mechanical lameness, this typical gait can be seen in horses without muscle fibrosis. Stringhalt is another swing phase lameness showing up at the walk.\textsuperscript{84} Hyperflexion of all hindlimb joints during the swing phase and worse during the cranial part of it, has been attributed to a lack of proprioceptive adjustment and muscle coordination. This disease highlights the passive coordination between all the hindlimb joint angles which flex in unison (Fig. 14).\textsuperscript{85}

Mixed Lameness at the Walk. Horses affected with proximal or distal peroneus tertius enthesopathies may present a swing phase lameness and a weight bearing lameness. A lameness induced by a foot abscess may present a stance phase and a swing phase component (see above). Neurological problems such as sciatic nerve paralysis or paresis can cause a lack of fetlock extension during the stance and swing phases.

Alternating Circles on a Firm Surface

Forelimbs. Horses reproduce on the circles the manifestations described on a straight line. Nevertheless, examination on alternating circles is essential as sometimes this is the only or the most demonstrative situation for detection of obvious to subtle changes of common conditions. As the limbs at the walk on a short circle combine a long stance phase duration with collateralmotion and associated rotation\textsuperscript{86-90} most of the horses affected with a podotrochlear syndrome (navicular disease) present with a reduction of the caudal phase of the stride (propulsion) when the lame limb is on the inside of the circle (Fig. 15). This typical gait alteration is induced by asymmetrical stresses on the podotrochlear apparatus (distal sesamoid bone, DDF tendon, and associated structures) as a result of collateral motion and rotation. These extra-sagittal movements are higher on the distal limb on the inside of the circle which is more oblique than the outside limb over the examination surface. Moreover, they are exacerbated at the end of the stance phase (Fig. 16).\textsuperscript{86} Horses with bilateral podotrochlear syndrome demonstrate this reduction of the caudal phase of the stride on both turns in an alternate manner; this results in a shortened gait. The swing phase protraction follows a longer pathway on the outside limb and reduction of protraction is sometimes clearer when the affected limb is outside of a short circle compared to the straight line (with the same causes: shoulder syndrome, CTB syndrome). On both the inside and outside limbs, special attention should be given to intermittent manifestations of carpus instability or of toe dragging as subtle manifestation of paresis.

Hindlimbs. Examining the horse on alternating circles at the walk provides another opportunity to check for joint motion, fetlock suspension, and changes in the cranial or caudal phases of the stride. As described for the forelimbs, collateralmotion and
rotation are responsible for asymmetrical distribution of stresses and exacerbation of pain on affected structures of the distal limb. However, generally speaking, as there is less load on the hindlimbs, the manifestations of distal hindlimb lameness at the walk are less demonstrative than on forelimbs. For example, in horses presenting with podotrochlear syndrome in a hindlimb, reduction of the caudal phase of the stride on the turns is less clear than on forelimbs. Nevertheless, pain can be exacerbated in case of injuries involving the distal and proximal interphalangeal joints as well as the fetlock joint especially when the proximal sesamoid bones are affected. The walk on alternating circles is one of the most informative situations to detect neurological problems on the hindlimbs and especially ataxia, paresis and dysmetria. A shortening of the hindlimb stride length accentuated on the turns may be seen in horses with lumbosacroiliac pain or dysfunction.91–92

**Axial regions.** A normal horse at the walk on alternating circles demonstrates relaxation and alternating lateroflexion of the axial regions. In the vertebral column, lateroflexion on one side is spontaneously associated to rotation on the opposite side.93 These combined movements induce shearing of the interspinal ligaments and spinous process margins. Besides, the sacroiliac joint undergoes greater range of flexion-extension, lateroflexion, and rotation at the walk than at the trot.94 Neck, thoracolumbar, or pelvic pain may induce a short and stiff gait, a lack of lateral bending, and sometimes counter-curvature (counter-bending).

---

**Fig. 16.** Functional anatomy of the distal interphalangeal joint of the left forelimb when the foot position is asymmetrical on a left turn. Elevation of the lateral quarter induces medial rotation of the distal phalanx (P3), tension of the medial collateral ligament (MCL) and relaxation of the lateral collateral ligament (LCL). As the lateral part of the condyle of the middle phalanx moves in a palmar direction, pressure is concentrated on the lateral part of the distal sesamoid bone (DSB) and the distal sesamoidean ligament (DSL) is stretched laterally. The lateral lobe of the deep digital flexor tendon (DDFT) receives pressure from the DSB and the medial lobe of this tendon is stretched.

**Fig. 17.** Ground reaction forces on a vertical (Fz-A) and horizontal craniocaudal (Fx-B) axes of a 600-Kg body weight steeple chaser at the walk (1.4m/s), trot (4.3m/s) and canter (leading limb, at 6, 8, and 10m/s for Canter 1, 2 and 3). The vertical and horizontal forces increase with the speed (From Robin D, Chateau H, Falala S, et al. 2008, with permission).

**Examination at the Trot on a Firm Surface**

**Trot on a Straight Line**

At the trot, the increased speed and the presence of a suspension phase increase the vertical and horizontal loading rate during the shock absorption phase and the vertical load at mid-stance compared to the walk (Fig. 17).60,95 Therefore, stance phase lameness is exacerbated and increased activation of the muscle groups at faster gaits makes some swing phase manifestations shade off. As the trot is an alternating symmetrical gait, most of the kinematic or kinetic devices developed to assess lameness use this gait as a reference.17–36,46 Gait analysis data have been compared with the visual clinical observation.20,34,38,40 Representation of the trajectories (vertical displacement over time) of the head, withers, and tuber sacrale versus time provides visual objective data on gait asymmetries (Fig. 18). These techniques have the potential to improve objective assessment of diagnostic analgesic techniques or rider technique and to help check horses after treatment or during a rehabilitation program. As the speed has an influence on the subjective and objective evaluation of asymmetry,
Trotting speed should be adapted and consistent on repeated occasions for diagnostic purposes and to check the horse after diagnostic analgesia or treatments.97,98

Forelimbs at the trot on a straight line. In most situations (except in perfectly bilateral lameness), to decrease the loading rate and load intensity on the lame limb the horse uses the inertia of the swing of the cervicocephalic pendulum (head and neck) and decreases the cranial phase of the stride. The result of this strategy is a reduction in fetlock extension. How is this strategy expressed clinically?

When examining a horse from the front (or the rear), alteration of the sinusoidal pattern of the head trajectory showing asymmetrical oscillations is the most obvious clinical manifestation of a forelimb lameness. These asymmetrical head and neck oscillations can also be seen from the side, but as head movements can disturb their regularity, this criterion can be difficult to assess, especially for low grade lameness. Altered sinusoidal pattern of the vertical displacement over time of the withers is often a more reliable manifestation of the horse’s strategy to put more weight on the sounder limb (Fig. 18).27 To decrease cumulation of vertical (z axis) and horizontal (x axis) loading rate after landing at the trot on a hard surface, the lame limb shortens the cranial phase of the stride. This is seen in many conditions including interphalangeal, fetlock, carpus, elbow, and shoulder conditions. In horses with pain in the suspensory apparatus, the increased loading rate combined with a peak of load in this apparatus during the cranial part of the stance phase67,71 induces a clear reduction of the cranial part of the stance phase at the trot compared to the walk. As mentioned above for shoulder and CTB syndromes, reduction of the cranial phase of the stride is often clearer at the walk. Even when a horse has demonstrated a reduction of the caudal phase of the stride at the walk (e.g., for podotrochlear syndrome), the cranial phase of the stride is more affected at the trot. It seems like the horse is more sensitive to the increased forces at the trot (kinetic effect) and less sensitive to the movement (kinematic effect). As mentioned above, at the walk, a slower movement and a longer stance phase duration without suspension phase increase the impact of distal interphalangeal joint extension on clinical manifestations. The decreased vertical load intensity and the reduction of the cranial phase of the stride induce a decreased fetlock extension on the lame limb compared to the opposite limb (Fig. 18).58,59 This reduction of fetlock extension is more pronounced in horses with pain in either the fetlock or the suspensory apparatus. In horses affected by a functional deficit of the suspensory apparatus or flexor tendons (rupture or elongation), the difference in fetlock extension during the stance phase is not proportional to the uneven loading of the limbs as expressed by the asymmetrical sinusoidal trajectory of the withers (or tuber sacrale for the hindlimbs). Some more proximal joint or periarticular injuries of the forelimb induce changes in movements of flexion-extension. This is especially seen for shoulder injuries (e.g., osteochondrosis, bicipital apparatus injuries) and carpal, carpal canal or extensor carpi radialis injuries.

Hindlimbs at the trot on a firm straight line. When examining the horse from the rear, redistribution of the load from the lame limb to the sounder limb is associated with an asymmetrical...
sinusoidal pattern of the tuber sacrale trajectory. Comparison of the left and right tuber coxae trajectories has also been used for diagnosing hindlimb lameness. Nevertheless, as demonstrated by most of the technical devices used for gait analysis, the tuber sacrale trajectory is a more reliable reflection of the horse’s strategy to put more weight on the sounder limb. This is expressed in an increase in downwards displacement of the tuber sacrale during the cranial part of the stance phase of the sounder limb which subsequently moves up the hindquarters (and tuber sacrale) using the inertia of the bodyweight to reduce the load during the stance phase of the lamer limb (Fig. 20). Discrimination between load absorption lameness and propulsion lameness can be done by considering the trajectory of the tuber sacrale. Alteration of the sinusoidal pattern of the head (cervicocephalic pendulum) movement can be seen with moderate to severe hindlimb lameness. Lowering of the neck is synchronous to the stance phase of the lamer hindlimb in an attempt to shift the load to the forelimb diagonally. For example, to reduce the load on a painful left hindlimb, the horse increases the weight on the right forelimb, the head exhibiting a lower position during the right diagonal stance phase. This has been confirmed using kinematic analysis of lame horses and of induced lameness on a treadmill. 

Examining the horse at the trot from the side facilitates identification of a reduction of the cranial phase of the stride. Just as on the forelimbs, the shortening of the engagement reduces the horizontal component of the ground reaction force and the duration (and therefore the impulse) of the load absorption part of the stance phase. As a consequence of the weight redistribution and shortening of the cranial phase of the stride, asymmetrical extension of the fetlocks can be seen from the side and the rear, with the horse exhibiting more extension of the fetlock in the sounder limb and less extension in the lamer limb (Fig. 20). However, in horses with alteration of fetlock suspension induced by elongation of the suspensory apparatus or the flexor tendons, fetlock extension can be exaggerated despite a reduction of load. In addition, asymmetrical flexion of the hocks during the stance phase can be observed with less flexion on the lamer limb due to the reduction of weight inducing less passive elongation of the SDF muscle and tendon as well as the superficial and deep aponeuroses of the gastrocnemius and lateral digital flexor muscles (Fig. 20 and 21). Dragging of the toe of the lame limb close to its mid-swing phase is a swing phase abnormality but not necessarily a swing phase lameness: it is the consequence of a lack of elevation of the croup by the lame limb during its propulsion phase, followed by a downwards displacement of the hindquarters during the stance phase of the sounder limb. 

Fig. 19. Eight-year-old Selle Français gelding jumper presenting a chronic desmopathy of the lateral branch of the suspensory ligament on the right forelimb. At trot on a straight line on hard ground, there is clearly less extension of the right fore fetlock than of the left one at mid-stance phase. There is also a reduced protraction of the left forelimb (left image) which anticipates the reduction of the cranial phase of the stride.

Fig. 20. Seven-year-old Standardbred trotter gelding presenting a fracture of the medial plantar process of the distal phalanx on the right hindlimb. At trot on a straight line on hard ground the croup is higher during mid-stance phase of the right hindlimb and lower during mid-stance phase of the left hindlimb. On the right hindlimb, there is less flexion of the hock and less extension of the fetlock than of the left one at mid-stance phase. As a consequence, during the mid-swing phase, the right toe is closer to the ground surface than the left one.

Fig. 21. Nine-year-old double pony gelding jumper presenting a bilateral distal intertarsal joint arthropathy with spontaneous ankylosis on the right hindlimb. At trot on a straight line on hard ground, the pony presented a left hindlimb lameness; at mid-stance phase, there was less fetlock extension and less flexion of the tarsus on the left hind limb than on the right one.
Axial regions. The trot is the most adequate gait for evaluation of the passive dorsoventral flexibility of the thoracolumbar spine.\textsuperscript{102-105} At the trot electro-myographic studies combined with kinematic analysis have demonstrated that the thoracolumbar spine is subject to passive flexibility induced by the alternating up and downwards movement of the abdominal visceral mass during the succession of support and suspension phases.\textsuperscript{100,105-108} Elevation of the visceral mass during the propulsion phase of one diagonal induces passive flexion of the thoracolumbar spine which is stabilized by the extensor epaxial (erector spinae and multifidus) muscles (Fig. 22). Dropping of the abdominal visceral mass at landing pulls down the thoracolumbar spine and its extension is limited by the flexor hypaxial (iliopsoas and abdominal wall) muscles. This passive flexibility of the thoracic, thoracolumbar, and lumbosacral areas is not synchronous and decreases with the speed (Fig. 22).\textsuperscript{105,106,108} It is also reduced in horses with back pain (F. Audigié, unpublished data) and during the stance phase of a lame diagonal.\textsuperscript{110,111} Its visual assessment helps to diagnose horses with pain or injuries of the thoracolumbar spine (kissing spines, articular process arthropathies, spondylosis) or of the lumbosacroiliac area.\textsuperscript{92,112,113}

A horse with back pain or caudal neck pain tries to reduce thoracolumbar flexion and extension movements by increasing the axial muscle tone. Clinically the horse demonstrates back (or neck) stiffness. It must be mentioned that horses with bilateral limb problems (especially forelimbs) reduce the elevation of the gait and therefore reduce visceral mass displacement and acceleration which consequently induces similarly a reduction of thoracolumbar flexibility. The differential diagnosis is then made by comparing the horse’s gait on soft ground which usually reduces distal limb manifestations and increases axial contribution to the gait (see below). Assessment of cervicothoracic flexibility is more difficult and less specific. It is more dependent on the forelimb locomotion.

Fig. 22. Kinematic analysis of the flexion-extension angles (T: thoracic angles, TL: thoracolumbar angle, LS lumbosacral angle) of the equine back at trot on straight line. A, Markers are placed over the sixth and twelfth thoracic vertebrae, over the second lumbar vertebra, tuber sacrale, and fifth sacral vertebra. B, The dorsal vertebral angles are not synchronous: the thoracic one extends and flexes before the thoracolumbar angle and the lumbosacral angle. C, The amount of motion of these vertebral angles decreases with the speed. (From Audigié et al. 1999 and Robert et al. 2002, with permission).

Trot on the Lunge in a Circle on Firm Ground

Horses with symmetrical movement on the straight line have been shown to present vertical head and pelvic asymmetry during lunging and this should not be interpreted as lameness.\textsuperscript{114} In this study, less than half of the horses showing symmetrical gait on a straight line had inverted and equivalent asymmetrical movements of the head and pelvis when examined on the lunge in opposite directions. This demonstrates that examination on the straight line has limitations to detect pain or biological or acquired laterality of the horse.

Forelimbs at the trot on circles on firm surfaces. The majority of forelimb lamenesses are worse when the affected limb is on the inside of the circle. As the horse leans inside to balance the centrifugal force,\textsuperscript{115} and as the feet are closer than the proximal parts of the limbs, the inside forelimb and hindlimb are more oblique to the ground surface than the outside limbs (Fig. 23). Therefore, collateromotion and associated rotation are higher on the inside limbs. These extrasagittal movements explain why pain on distal joints and in the podotrochlear apparatus including the distal part of the DDF tendon, is exacerbated when the affected limb is inside the circle (Fig. 16).\textsuperscript{87} Clinical manifestations of some conditions involving the proximal limb are less affected by collateromotion and rotation but may also, to a lesser extent, be worse when the lame limb is inside the circle. Less commonly, the horse may be lamer when the painful forelimb is outside the circle because of the increased load on the outside limb (e.g., suspensory apparatus diseases) or increased pressure at the medial aspect of this limb (Fig. 24). This is observed
with some fractures of the medial palmar process of the distal phalanx, subchondral bone pain in the medial part of the metacarpal condyle, medial splints (second or medial intermetacarpal syndesmopathy), medial third carpal or radiocarpal bones injuries, with the horse being sound or mildly lame at the walk for these last conditions. Propulsion lameness induced by a DDF tendonitis can occasionally be worse on the opposite circle (Fig. 25). As the suspensory apparatus is more affected by the loading rate and peak of load than by collateromotion and rotation, lameness induced by suspensory disease is usually clearer on a straight line and on the opposite circle. Horses presenting dyskinetic protraction induced by CTB syndrome or shoulder syndrome (scapulohumeral arthropathies or bicipital apparatus injuries) show a slower swing phase ending with a reduced cranial phase of the stride when the affected limb is placed outside the circle. Lameness severity can be similar on both circles in case of desmopathies of the lateral collateral ligaments of the interphalangeal and metacarpophalangeal joints, subchondral bone trauma of the medial part of the metacarpal condyle, as well as some elbow and shoulder joint conditions. For (subchondral) bone conditions, pain is generated by collateromotion and rotation when the limb is inside the circle and by concentration of the pressure at the medial aspect of the affected limb when it is outside the circle. In lateral collateral desmopathies and enthesopathies, pain is generated by increased tension on the ligament when the affected limb is outside the circle and by extrasagittal movements of the painful joint when this limb is inside the circle.

Hindlimbs at the trot on circles on a firm surface. A sound horse trotting on a circle puts more load on the outside hindlimb to counteract the centrifugal force. It is essential to

Fig. 24. Trajectories determined from inertial measurement units at the trot on a hard surface of the head, withers, and tuber sacrale of a 12-year-old jumper female presenting a subchondral bone trauma of the medial part of the metacarpal condyle on the left forelimb. On a straight line, the mare showed a very mild reduction of the load on the left forelimb (LF) and puts more load on the right hindlimb as a potential expression of diagonal compensation. On the left circle the trajectory of the withers is symmetrical but the head drops on the right forelimb (RF); after elevation of the croup by the inside LH, the tuber sacrale drops more on the right hindlimb. On the right circle, although there should be more load on the outside LF, the head and withers markedly drop during the RF stance phase (red line). The subchondral bone pain at the medial side of the fetlock is responsible for this stance phase lameness, exaggerated on the opposite circle. As the head drops during the RF diagonal stance phase, there is less load on the outside LH. The different curves represent the trajectories of successive strides.
compare the asymmetrical movements of the pelvis and fetlock extension of both hindlimbs on the left vs right circle to avoid overdiagnosis of lameness. This can be demonstrated establishing the asymmetrical vertical movement pattern of the tuber sacrale using inertial sensors (inertial measurement units) showing more downwards displacement of the tuber sacrale during the stance phase of the outside hindlimb.\textsuperscript{114,118–120}

Compared to the normal range of hindlimb asymmetry on circles, horses presenting interphalangeal or metatarsophalangeal arthropathies are lamer, with a reduction of the cranial phase of the stride when the affected hindlimb is inside the circle as limb obliquity increases collateral motion and associated rotation causing pain.\textsuperscript{87} As the load is higher on the outside limb,\textsuperscript{117} horses presenting with pain in the suspensory apparatus may be lamer when the affected limb is outside in a circle on a hard surface. In horses with severe pain, the downwards displacement of the tuber sacrale can be seen on the sounder inside hindlimb. Nevertheless, horses with desmopathy or enthesopathy of the medial branch of the suspensory ligament may stay lamer when the affected limb is inside the circle as lateromotion increases the stress of this branch. Other conditions involving the medial aspect of the hindlimb such as distal tarsus osteoarthritis or spongy (subchondral) bone cyst in the medial femoral condyle may be lamer when the affected limb is outside the circle, as pressure forces are increased medially.

**Axial regions on circles.** On the circle, the vertebral axis of the horse combines movements of flexion-extension movements with lateroflexion and rotation and the range of motion is greater compared to the straight line.\textsuperscript{93,100,118} Therefore, visual assessment of the amount of motion is facilitated especially when the horse decelerates from trot to walk. At the trot on the lunge in a circle, the sinusoidal pattern of the thoracolumbar oscillation is different for the two diagonal stance phases. As there is more load absorption on the outside hindlimb, there is more extension during the stance phase of this limb than during the stance phase of the inside hindlimb. These asymmetrical flexion-extension movements combined with lateroflexion and rotation can exacerbate thoracolumbar pain and create discomfort resulting in back stiffness and reduction of the stride length and suspension.

3. **Soft Surface on the Lunge**

When examining a horse in a circle on soft ground care should be taken by the operator holding the lunge as horses can buck from happiness on soft surfaces and may be dangerous, especially if they had a reduction of physical activity before the examination.

Examination at the Trot on the Lunge on Soft Ground

Comparing the gait abnormalities on soft and hard surface contributes significantly to the differential diagnosis of the cause of lameness and to the critical
assessment of the significance of imaging findings at the end of the examination. A soft surface is chosen to change the biomechanical stresses on the limbs; deep surfaces should be avoided for the routine diagnostic approach as the muscular demand for propulsion is increased and active efforts mask diagnostic information. Trotting speed and the radius of the circle may influence the grade of asymmetrical movements. Generally speaking, on soft ground surfaces the loading rate and loading intensity on the limbs decrease (Fig. 26) but propulsion forces and therefore the load on the tendons increase (Fig. 27). Moreover, on soft deformable surface, collateromotion and associated rotation are reduced as the foot penetrates more and the position of the center of pressure moves toward the center of the foot (Fig. 28). Therefore, the clinical manifestations of foot problems and distal or intermediate limb arthropathies improve more than those of proximal limb arthropathies or tendinopathies. Lameness induced by recent tendinopathies tend to be worse on a soft ground. Discussion on the properties of different types of surface is beyond the scope of this paper.

**Forelimbs**

When the affected limb is on the inside of the circle, horses presenting with chronic foot problems or distal arthropathies are improved on soft ground compared to the clinical manifestations on a hard surface. Improvement is less clear for intermediate arthropathies and mild or absent for proximal lameness. The lameness is rarely worse on soft ground, but this can be seen for recent and severe distal DDF tendon injuries. When the affected limb is on the outside of the circle, horses presenting recent injuries of the suspensory apparatus or flexor tendons (SDF and DDF tendons) are not improved or can be worse on soft ground. Several biomechanical factors are responsible for that including the higher load on the outside limb, the increased need for propulsion forces on soft ground, and the interphalangeal flexion reducing the DDF tendon contribution to the fetlock support. As seen in a circle on a hard ground, horses presenting dyskinetic protraction induced by CTB syndrome or shoulder syndrome show a slower protraction during the swing phase ending with a reduced cranial phase of the stride when the affected limb is placed outside the circle.

**Hindlimbs**

Many lame horses with distal or middle hindlimb injuries improve on soft ground compared to similar situations on hard ground as a result of the reduction in concussion, load absorption and extrasagittal movements, especially when the lamer limb is on the inside of the circle. Horses presenting pain in the suspensory apparatus typically present an exacerbation of the lameness when the affected limb is on the outside of the circle. As for forelimbs, this is explained by the higher load on the outside hindlimb, the increased propulsion forces on soft ground and the lower DDF tendon contribution to the fetlock support (Fig. 29). The same observation can be made with horses presenting lesions of the gastrocnemius or digital flexor (SDF, DDF) tendons. When the affected limb is on the outside of the circle, horses presenting with recent episodes of pain in the distal tarsus (bone spavin) or in the medial femorotibial joint are not or mildly improved as the reduction of loading rate on soft ground is canceled by the concentration of load at the medial aspect of the joint.

**Axial Regions**

In horses with pain or sensitivity in the distal joints, the suspension phase is reduced and passive vertebral axis flexibility decreases on the circle on a hard ground (see above). As these horses are more comfortable on a soft surface and display more active gaits, examination of the horse trotting on the lunge on soft ground facilitates assessment of the passive
thoracolumbar, lumbosacral as well as cervicothoracic flexibility.\textsuperscript{104,111} Criteria include the amount of motion, relaxation, and lateral bending on the circle (Fig. 30).\textsuperscript{112} They are altered in horses with back, neck, or lumbosacroiliac pain or stiffness (Fig. 31).

Examination at Canter on the Lunge on Soft Ground
In a study performed on 12 galloping horses on a straight line, no significant difference was observed in head and pelvis acceleration and rotation, limb timing, and stride duration measurement, before and after induction of lameness in the forelimb and hindlimb.\textsuperscript{37} Comparing left and right circles, asymmetry can be detected in forelimb and hindlimb movements as well as in axial positioning and mobilization. Canter provides another opportunity to assess the horse’s coordination as ataxia and hypermetria are sometimes revealed or clearer at this gait.

Axial Regions
At the canter, the thoracolumbar and lumbosacral regions are actively moved by extensor epaxial (erector...
spinae and multifidus muscles and flexor hypaxial (iliopsoas and abdominal wall) muscles. Active lumbosacral flexion and extension movements are wider and therefore easier to assess than thoracolumbar mobilization which remains proportionally limited. The canter on the lunge (except for racing trotters!) is an adequate situation to evaluate head and neck carriage and motion as well as neck and back coordination and lateral bending. Pain or pain adaptation may affect the relaxation of the gait, the stride length, the balance of the suspension phase, the positioning and mobilization of the axial regions and the quality of lateral bending on turns. Horses with neck or back pain tend to pull on the lunge or be counter-incurvated (Fig. 31). In horses with back pain, a lack of thoracolumbar lateroflexion (lateral bending, incurvation) predisposes the horse to display a disunited or rotary canter as the inside hindlimb is not placed adequately to achieve protraction.

Forelimbs and Hindlimbs

Biomechanical studies at the canter demonstrated that the vertical load was higher on the diagonal (trailing forelimb-leading hindlimb). There was more deceleration on the leading limbs and more propulsion on the trailing limbs. The canter highlights some defects in limb movement and especially protraction. Dyskinesia of the forelimbs induced by neurological deficits related to cervicothoracic arthropathies (part of the CTB syndrome) is usually more pronounced on the outside forelimb (Fig. 32). When the most affected limb is placed inside the circle, the horse may canter on the wrong sounder leading limb (leading limb outside) in a reiterated manner.
As mentioned above, a wrong placement of the hindlimbs (disunited canter) can be seen in horses with inadequate thoracolumbar lateroflexion. A protraction deficit induced by any injuries of the inside hindlimb can also result in a disunited canter. Looking at the horse performing downward transitions from canter to trot and from trot to walk is particularly informative on forelimb and hindlimb coordination and on how the horse is distributing the deceleration forces within the different limbs.

Small Jumps on the Lunge on Soft Ground
Examing jumpers doing small jumps on the lunge may provide additional information on forelimb and hindlimb lameness manifestations. Other objectives of this simple exercise include checking the owner or trainer’s concerns as well as anamnisis and getting information on the horse’s athletic capacities and style. Axial problems involving either the cervical or the thoracolumbar spine or the lumbosacroiliac junction need to be considered when the horse’s technique, power, and style are altered.

Abnormalities observed during jump approach and take-off include poor forelimb placement, propulsion and impulsion, lack of forequarter elevation, and poor neck mobilization or impulsion. These manifestations can be seen in horses with forelimb problems and horses with cervical or thoracolumbar injuries. A defect in hindlimb propulsion can be seen in jumpers with hindlimb, lumbosacroiliac or thoracolumbar injuries. A decrease in cervical and thoracolumbar mobility is the most obvious manifestation of back pain during the jump suspension or airborne phase. Unilateral or
bilateral deficit of forelimb or hindlimb flexion can be related to intrinsic limb injuries or to injuries of the corresponding vertebral segment and nerves. Eccentric contraction of the muscles of the proximal forelimb controlling the flexion of the shoulder and elbow joints, and eccentric contraction of the serrati and pectoral muscles as well as dorsal cervical muscles have an essential contribution to load absorption at landing after the jump (Fig. 34). 57

Neck stiffness at landing can be a manifestation of neck or thoracic pain (e.g., thoracic spondylosis). Extension and subsequent flexion of the lumbosacral area can be altered by lumbosacroiliac pain (Fig. 35). A leading preference at landing can be the manifestation of several conditions as explained by biomechanical data (Fig. 36). 137, 138 As the impulse (combining load and duration represented by the area under the curve) is larger on the leading limb, a horse with suspensory apparatus or fetlock pain prefers placing the painful limb as a trailing limb (contact limb) at reception (Fig. 37). Horses with reduction of protraction as a manifestation of CTB syndrome tend to prefer landing on the lead opposite to the lesion, the trailing limb being then on the more clinically affected side.

4. Examination of the Horse at Work

Ridden Sport or Pleasure Horses

Manifestations described above can be reduced or exacerbated, not only because of the saddle and rider interference, but also because of a different behavior of the horse at work. 139–142 Only key data are shortly presented in this paper.

Walk

Putting the saddle on and tightening the girth may reduce unilateral or bilateral protraction of the forelimbs. Several causes of this manifestation have been documented or suspected such as the CTB syndrome, withers, and thoracic injuries as well as costovertebral arthropathies as pressure over the sternum can reproduce the symptoms.

Trot on Alternating Diagonals Between Opposite Turns

Compared to the trot in hand and on the lunge, lameness at the trot on the ridden horse may be worsened because of the rider’s weight and technique or improved by appropriate rider’s management. 143, 144 Intensification has been seen with suspensory apparatus conditions in the forelimbs and hindlimbs. As the horse puts more load on the outside hindlimb on a circle (Fig. 29), 117 trotting on diagonals alternating left and right turns is recommended to evaluate the
inversion of the hindlimbs asymmetry on opposite turns or to get more data on lameness manifestations. The influence of the rider at the rising trot should be considered.\textsuperscript{138,145,146,147} During the sitting phase of the rider, the range of motion decreases between T12 and L2 and increases between T6-T12 and L2-L5.\textsuperscript{148} Besides, the roll of the pelvis is reduced and the load increases on the sitting diagonal (Fig. 38).\textsuperscript{144,148} When a forelimb limb presents a decreased fetlock suspension during the stance phase, sitting on the opposite diagonal may decreased fetlock extension asymmetry. Therefore, when a lame forelimb presents a decreased fetlock extension during the stance phase, sitting on the corresponding diagonal can attenuate kinematics manifestations of lameness; sitting on the sounder forelimb diagonal tend to increase forelimb extension asymmetry. On a right turn, compared to symmetrical sitting trot, at rising trot a left hindlimb lameness tends to be clearer as the rider adds weight on the right hindlimb. On the left turn, the rider sits on the right diagonal (right forelimb + left hindlimb) and a left hindlimb lameness tends to be reduced.\textsuperscript{146}

\textit{Canter}

Additional clinical and athletic information can be obtained at the canter including the horse’s intrinsic capacities and limitations and the rider’s influence.\textsuperscript{143} Equestrian aspects of assessment of the canter are beyond the scope of this paper.\textsuperscript{149} As for the canter in lunge, the quality of the gait, the mobilization and coordination of the limbs and joints, the positioning and mobilization of the axial regions are evaluated. Special attention should be paid when the horse is changing leads. When no obvious lameness is observed at the trot, difficulties or defenses during this exercise (to be assessed according to the level of education of the horse and rider) are usually related to proximal limb or axial problems such as the CTB syndrome for the forelimbs, thoracolumbar injuries or the lumbosacraliliac syndrome for the hindlimbs.

\textit{Jumping and Dressage Exercises}

Examination of the horse at work is routinely done to investigate athletic problems or to check anamnesis when lameness is described as happening in specific situations or exercises.\textsuperscript{140,143} This is sometimes required to evaluate diagnostic analgesic techniques in horses showing insufficient manifestations during the routine clinical examination in an attempt to get as objective an assessment of the block as possible. This can also be useful to evaluate the horse’s tolerance to different athletic exercises in order to adjust a rehabilitation program. Several studies have investigated the mechanics and kinematics of the horse while jumping.\textsuperscript{57,100,136,143,150} Influence of the height of the fence has been investigated using kinematic and kinetic analysis of the limbs of jumping horses.\textsuperscript{138,150–152}

In dressage horses, pain induced by collateromotion and rotation of the distal joints can alter the regularity of adduction and abduction movements during lateral work (e.g., half pass, shoulder in). Horses presenting a CTB syndrome tend to present a head nod on the sounder side (limb) to facilitate adduction during the swing phase of the more affected side. During a pirouette, as the horse concentrates the weight on the engaged inside hindlimb working as a pivot, the increased ground contact of the foot exacerbates the stresses induced by the medial rotation of this limb. Just as examples, medial rotation of the hind fetlock joint increases pressure on the lateral proximal sesamoid bone and tension on the deep layer of the medial collateral ligament. Medial rotation of the tarsus increases transverse shearing of the distal tarsal joints, tension of the calcanean fasciculus of the medial collateral ligament and stresses on the grooves of the tibial cochlea. Medial rotation of the femorotibial joint increase pressure on the cranial horn of the medial meniscus and tension of the cranial cruciate ligament. The tension of the capitis ligament and pressure on the articular surfaces of the coxofemoral joint are also increased with medial rotation of the corresponding hindlimb.

\textit{Thoroughbreds Ridden (at Canter) on the Track}

Examination of horses being ridden at the trot on the track is useful when only mild to moderate asymmetry or lameness can be seen at the walk and the trot in hand. Movement analysis using slow motion video at different gaits is particularly useful to detect or confirm visual observations (Fig. 39). Looking at the horse cantering on both left and right leads is essential to identify the horse’s preferred lead, which may be in relation to existing clinical or subclinical conditions or may predispose to secondary injuries induced by asymmetrical load on diagonals (Fig. 40).\textsuperscript{96,132,133,143}
As the canter is an asymmetrical gait, evaluation of the head, withers, and tuber sacrale trajectories does not provide clear clinical data on the redistribution of load between limbs in a lame horse. On the forelimbs, there is a higher fetlock extension on the trailing forelimb than on the leading one during the stance phase. Therefore, to assess clinically potential redistribution of load, it is essential to evaluate fetlock extension of the forelimbs or the hindlimbs at the left and right leading canter at the same speed (using slow motion video-recordings). When ridden at a canter, evaluation of the thoracolumbar mobility is limited but manifestations of back pain or discomfort may be exacerbated (Fig. 41). Visual assessment of the lumbosacral flexion and extension movements is easier and the amount of motion increases with the speed.

Harness Racing Trotters on the Track
Examination of racing Standardbred trotters in hand is often frustrating and observations are not always correlated to the complaint and anamnesis. Examination of the horse being driven on the track is an essential part of the evaluation of lameness and discipline related problems. Only the most common remarks are mentioned in this paper.

1. It is quite common to observe a lameness on one side when the horse is examined in hand and a lameness is on the opposite side on the track. The primary lameness seen at high speed induces an overload of the opposite limb at work. At slow speed when the stresses responsible for the manifestations of the primary lameness are lower, the secondary lameness shows up. Therefore, it is crucial to identify the primary cause of the athletic manifestations at training or high speed to direct the diagnostic approach and adequately manage the horse.

2. A frequent gait alteration at speed called trauquenard or hiking lameness can be seen with pain involving the forelimb or the hindlimb on the same side (Fig. 42). The forelimbs are at the trot and the hindlimbs seem...
to canter. The use of slow-motion video is essential to analyze the fast sequence of limb placement. In a horse presenting with a left forelimb lameness as in Fig. 42, the right hindlimb increases protraction and anticipates ground contact to reduce load on the painful left forelimb. As the right hindlimb lands before the left forelimb, the distance between the limbs of the left diagonal increases. As propulsion made by the right hind is more effective, landing of the left hindlimb is delayed inducing shortening of the right diagonal distance. The same features are observed in a horse presenting with a left hind lameness. Complementary analysis of this gait alteration can be performed using a video-camera placed over the helmet of the driver (Fig. 43).

3. A common manifestation on turns is to put more load on the inside hindlimb at medium and high speeds. Therefore, a hindlimb lameness is often worse when the affected
Fig. 44. Three-year-old harness Standardbred trotter male examined on the track at intermediate speed. The horse exhibits the aubin, cantering with the frontlimbs and trotting with the hindlimbs. This abnormal gait can be induced by different conditions from fetlock pain up to CTB syndrome and even by hindlimb pain. This particular horse exhibiting a defect of the right forelimb protraction had a fragmentation of the right C6-C7 articular processes with chronic synovitis of the corresponding joint.

limb is outside the turn. For example, when a trotter presents with a left hindlimb lameness on a straight line, quite systematically the lameness is worse on the right turn and the horse is improved on the left turn.

4. A deficit in forelimb protraction is responsible for an asymmetrical forelimb gait called aubin. The hindlimbs are at the trot and the forelimbs seem to canter (Fig. 44). The protraction deficit (swing phase lameness) is worse when the affected limb is on the outside of the turn. Different conditions can induce this gait abnormality ranging from distal limb injuries up to CTB syndrome.

5. Hindlimb asymmetry induces a corresponding forelimb asymmetry. Therefore, a complete clinical and imaging evaluation of the corresponding lateral (forelimb and hindlimb of the same side) is required.

6. The lumbosacral joint is highly specialized in flexion and extension. Rotation and, above all, lateroflexion are limited. Unlike in Thoroughbreds showing wide lumbosacral flexion-extension movements at fast speeds, in trotters protraction of one hindlimb is synchronous to propulsion of the opposite limb and rotation becomes the main movement of the lumbosacral junction. This explains why intertransverse lumbosacral arthropathy is a common condition in racing trotters.

5. Conclusions

Lame horses are live illustrations of functional anatomy and biomechanics. The trot is often considered as the reference gait for lameness evaluation and to assess diagnostic analgesia. Much information can be gained when the horse is examined at the walk, some of which (such as forelimb protraction or fetlock suspension) becoming less clear or even disappearing at faster gaits. Looking at the horse in different situations in hand and on the lunge, combining different surfaces and different exercises of training and even competing allows for a complete clinical and athletic assessment of the horse and understanding of its gait alterations. Identification of the best as well as the worst situations for the horse is not only essential from a diagnostic point of view but also provides adequate pieces of information to establish the best rehabilitation program for each individual patient. At present kinematic and kinetic technologies are being developed to provide objective data to evaluate lameness in horses; these devices are well adapted for assessment of a symmetrical gait such as the trot. In practice, evaluation of the different gaits and exercise conditions can be done using video-recordings in the field. Their analysis enables a global and detailed evaluation of the horse’s locomotion as every joint can be assessed independently. Moreover, based on these recordings, a more objective follow up of the horse’s locomotion can be done at several months or even yearly intervals allowing a more accurate assessment of the evolution of the disease process, of the efficacy of a treatment and/or of a rehabilitation program. No doubt that instrumental analysis of locomotion in horses will bring new data on the manifestations of pain, mechanical, and neurological disorders in the future. We must keep in mind that few horses display symmetrical gaits, so the new challenge is to determine what is an acceptable degree of asymmetry. As locomotion is the expression of an endless number of biological factors, the answer varies with individual horses and the diagnosis and prognosis of locomotor variations and troubles is not dependent only on figures, graphs, or statistical analysis but must consider the interindividual capacity of tolerance to pain as well as to the physical and functional asymmetry. The mixture of objective data combined with individual expression make this field of equine locomotion and its alterations such an exciting and fascinating discipline.

Acknowledgments

The Author wishes to acknowledge his colleagues of the INRAE-BPLC unit for providing scientific support to the clinical field and his colleagues working at CIRALE for their assistance in performing imaging procedures and contribution to the examination of our equine patients. A special thanks to the referring practitioners sending cases contributing to build the collective experience of our profession. The author of this paper has no actual or potential conflict of interest including any financial, personal, or other relationships with other people or organizations that could inappropriately influence, or be perceived to influence, his work.
Clinical activities and research of CIRALE-ENVA are supported by the Conseil Régional de Normandie, the Calvados Department, the FEDER and the Eperon Funds.

Declaration of Ethics
The Author has adhered to the Principles of Veterinary Medical Ethics of the AVMA.

Conflict of Interest
The Author has no conflicts of interest.

References
44. Adair S, Baus M, Belknap J, et al. Response to the letter to the editor: Do we have to redefine lameness in the era of...


