General Articles

The effect of induced forelimb lameness on thoracolumbar kinematics during treadmill locomotion

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Summary

Reasons for performing study: Lameness has often been suggested to result in altered movement of the back, but there are no detailed studies describing such a relationship in quantitative terms.

Objectives: To quantify the effect of induced subtle forelimb lameness on thoracolumbar kinematics in the horse.

Methods: Kinematics of 6 riding horses was measured at walk and at trot on a treadmill before and after the induction of reversible forelimb lameness grade 2 (AAEP scale 1–5).

Ground reaction forces (GRF) for individual limbs were calculated from kinematics.

Results: The horses significantly unloaded the painful limb by 11.5% at trot, while unloading at walk was not significant. The overall flexion-extension range of back motion decreased on average by 0.2° at walk and increased by 3.3° at trot (P<0.05). Changes in angular motion patterns of vertebral joints were noted only at trot, with an increase in flexion of 0.9° at T10 (i.e. angle between T6, T10 and T13) during the stance phase of the sound diagonal and an increase in extension of the thoracolumbar area during stance of the lame diagonal (0.7° at T13, 0.8° at T17, 0.5° at L1, 0.4° at L3 and 0.3° at L5) (P<0.05). Lameness further caused a lateral bending of the cranial thoracic vertebral column towards the lame side (1.3° at T10 and 0.9° at T13) (P<0.05) during stance of the lame diagonal.

Conclusions: Both range of motion and vertebral angular motion patterns are affected by subtle forelimb lameness. At walk, the effect is minimal, at trot the horses increased the vertebral range of motion and changed the pattern of thoracolumbar motion in the sagittal and horizontal planes, presumably in an attempt to move the centre of gravity away from the lame side and reduce the force on the affected limb.

Potential relevance: Subtle forelimb lameness affects thoracolumbar kinematics. Future studies should aim at elucidating whether the altered movement patterns lead to back and/or neck dysfunction in the case of chronic lameness.

Introduction

The present-day equine practitioner is confronted with increasing numbers of cases presented for poor performance, subtle gait irregularities or alleged back problems. Although it is obvious that the axial skeleton is the link between the extremities, there is controversy as to the relationship between back problems and lameness. In a population of horses presented for orthopaedic problems, 26% had concurrent lameness and back pain upon palpation (Landman et al. 2004). Dyson (2005) reported that, in the majority of horses with primary thoracolumbar or sacroiliac pain, overt lameness was not a feature, but many horses showed restricted hindlimb propulsion, poor hindlimb engagement and a low-grade toe drag. These are, however, qualitative studies in cases based on clinical judgement and with dissimilar criteria. Besides, thoracolumbar abnormalities secondary to lameness have not been fully described. Experimental, quantitative lameness-studies on whole body dynamics have been conducted (Buchner et al. 1995, 1996a,b; Vorstenbosch et al. 1997; Keegan et al. 2000), but these focused more on head and trunk movements than on specific thoracolumbar kinematics. Pécot et al. (1998) demonstrated a small influence of induced lameness on dorsoventral mobility, but relatively little detail was provided because only 4 markers were used to analyse back mobility.

Recent developments in analysing 3D thoracolumbar kinematics based on the work by Faber et al. (1999, 2000) and Johnston et al. (2002) have created the possibility of accurately analysing the effect of specific conditions or interventions on equine back kinematics. So far, this analysis has been used successfully to study the influence of physiological factors (Johnston et al. 2004), of the presence of clinical back pain (Wennerstrand et al. 2004) and of specific head and/or neck positions (Rhodin et al. 2005; Gómez Álvarez et al. 2006) on thoracolumbar kinematics. The present study aims to elucidate the effect of subtle forelimb lameness on back kinematics using the same analysis. The study was conducted as a first assessment of the relation between subclinical lameness and back and neck

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motion to improve the basic knowledge on secondary back problems. The hypothesis tested was that even a subtle lameness would result in a measurable change in thoracolumbar kinematics. For this purpose, the kinematics of the vertebral column and limbs in horses at walk and trot were determined on a treadmill, before and after the induction of fully reversible, subtle forelimb lameness.

Materials and methods

Horses

Kinematics of the back was measured in 6 sound Dutch Warmblood horses without lameness or other abnormalities, age 11.7 ± 4.9 years, height at the withers 163 ± 4.8 cm and body mass 577 ± 37.1 kg, while they were walking (1.6 m/s) and trotting (4.0 m/s) on a treadmill. The horses had been trained previously and were well accustomed to the treadmill. The experimental protocol was approved by the Animal Experimentation Committee of Utrecht University.

Lameness induction

Reversible lameness was induced in the left forelimb with a modified shoe featuring a nut welded to the inner side of the toe region. A bolt in the nut could be tightened to exert pressure on the sole, thus provoking pain. A more extensive description of the technique can be found elsewhere (Merkens and Schamhardt 1988). The lameness provoked was grade 2 of the AAEP scale (lameness difficult to observe at a walk or trot in a straight line; consistently apparent under some circumstances, such as weight carrying, circling, inclines, hard surface: Stashak 2002).

Quantification of lameness

The method used to quantify the lameness made use of the fact that during a supporting-limb lameness the horse tries to reduce the load of the painful limb (Buchner et al. 1996b). Therefore, loads on individual limbs were calculated from kinematics according to a recently developed method (McGuigan and Wilson 2003; Bobbert et al. 2007). The method involves the calculation of the total ground reaction force (GRF) from kinematics (Bobbert and Santamaria 2005), followed by the determination of the distribution of this force over individual limbs in those phases of the stride cycle where only 2 limbs are in contact with the ground. It has been shown that changes in peak individual limb reaction forces over time can be calculated using this method with a standard error of measurement of 0.2 N/kg. At walk, the GRF were calculated from the distal limb length assuming that the distal limbs operate as linear springs, of which the force-length relationships were determined using calculated individual limb forces at trot (Bobbert et al. 2007).

Data collection

Measurements were performed using the infrared-based ProReflex automated gait analysis system1, operating at 100 Hz. Spherical infrared light reflective markers with a diameter of 19 mm were glued to the skin over the spinous processes of thoracic vertebrae 6, 10, 13 and 17 (T6, T10, T13, T17), the lumbar vertebrae 1, 3 and 5 (L1, L3, L5), and the 3rd sacral vertebra (S3). Markers were also placed on the coxal tubers and to the lateral sides of the hooves. Markers were located on the limbs, head and neck (Bobbert and Santamaria 2005). Six infrared cameras situated at both sides of the treadmill recorded the marker locations while the horses were standing square and at walk and trot before, during and after the induced lameness. The actual recordings were performed during 10 s after 1 min locomotion on the treadmill. The treadmill was stopped for 1 min between the 3 consecutive measurement sessions (before, during and after induction of the lameness) in order to tighten the bolt in the shoe or to remove it.

Data analysis

Qualisys Track Manager Software1 was used to capture and process data. A standard right-handed orthogonal Cartesian coordinate system was used to describe the motion of the vertebral column. Motion was described as flexion-extension (in the sagittal plane), lateral bending (in the horizontal plane), and axial rotation of the sacral bone (in the transversal plane). All the vertebral movements were calculated using Backkin1 and presented as angular motion patterns (AMP) during the stride cycle. The range of motion (ROM) was calculated for each AMP and defined as the difference between maximal and minimal values of the AMP. Data captured in the square standing horse before and after the lameness induction were used to determine the zero value in the AMPs in each horse. The vertebral angles were defined as the angle between 3 adjacent marked vertebrae (e.g. the angle at T10 is the angle between the line from T6 to T10 and the line from T10 to T13). The calculated angles are shown in Figure 1. The overall flexion-extension range of motion was the average of the ranges of motion of all the vertebral angles in the sagittal plane. The beginning of each stride cycle was taken to be the initial ground contact of the left hindlimb. The correlation coefficient between the vertebral angular motion patterns was calculated to quantify the intravertebral pattern symmetry (Faber et al. 2000). The neck angle was calculated as the angle between the markers on T6 and atlas and the horizontal plane. Stride length was calculated from the marker on the left hindlimb. Protraction-retraction angle was calculated for the 4 limbs using the markers on the hooves and T6 for the forelimbs, and the hooves and S3 for the hindlimbs.

The distribution of values for kinematic variables and calculated forces was tested for normality. If normally distributed, further analysis was carried out using ANOVA for repeated measures and a Bonferroni post hoc test. The overall range of motion was analysed for variance deviations, with the different vertebrae of individual animals being treated as repeated-measures. If data were not normally distributed, a Wilcoxon signed rank test was used. The level of significance was set at P<0.05. All data were expressed as mean ± s.d. unless otherwise stated.
Results

Quantification of lameness

The lame limb was significantly unloaded only at trot. The peak vertical ground reaction force on the lame limb significantly decreased from 13.1 ± 1.5 to 11.6 ± 1.4 N/kg (P<0.05). At walk, the peak vertical GRF on the lame limb was 7.3 ± 1.0 N/kg before and 7.1 ± 0.9 N/kg during lameness.

Stride length and protraction-retraction angle

There were no significant changes in the stride length or in the protraction-retraction angle of the 4 limbs in either of the gaits (Table 1).

Vertebral range of motion

At walk, the overall flexion-extension ROM of the vertebral column was significantly reduced from 6.2 to 6.0° in the lame condition (Fig 2). However, when testing the range of motion of individual vertebral angles, the range of motion in the lame

| TABLE 1: Range of motion (ROM) values (mean ± s.d., °), neck angles (°), stride length (m) and protraction-retraction angles (°) at walk and trot in horses with induced subtle forelimb lameness |
|----------------------------------|------------------|------------------|------------------|
|                                   | Walk             | Trot             |
| Motion                           | Sound            | Lame             | Sound            | Lame            |
| Flexion-extension T10            | 5.1 ± 1.0*       | 4.8 ± 0.9*       | 3.4 ± 0.6*       | 4.0 ± 0.8*      |
|                                 | T13              | 6.3 ± 0.6        | 6.1 ± 0.9        | 2.3 ± 0.7*      | 2.9 ± 0.6*      |
|                                 | T17              | 6.8 ± 1.0        | 6.6 ± 1.4        | 2.3 ± 0.4       | 2.6 ± 0.4       |
|                                 | L1               | 6.6 ± 1.4        | 6.4 ± 1.7        | 2.8 ± 1.0       | 2.9 ± 0.7       |
|                                 | L3               | 6.4 ± 2.0        | 6.1 ± 2.0        | 2.9 ± 0.7       | 3.0 ± 0.7       |
|                                 | L5               | 5.9 ± 2.0*       | 5.7 ± 2.1*       | 2.9 ± 0.7       | 2.9 ± 0.9       |
| Overall variation                | 6.2 ± 1.3*       | 6.0 ± 1.5*       | 2.8 ± 0.7        | 3.1 ± 0.7*      |
| Lateral bending T10              | 8.9 ± 1.9        | 8.9 ± 2.0        | 7.2 ± 1.2*       | 6.7 ± 1.6*      |
|                                 | T13              | 5.0 ± 1.0        | 4.6 ± 1.2        | 4.3 ± 1.3       | 4.1 ± 1.3       |
|                                 | T17              | 3.2 ± 0.9        | 3.1 ± 1.0        | 3.3 ± 0.9       | 3.4 ± 0.9       |
|                                 | L1               | 4.0 ± 1.4        | 4.4 ± 0.7        | 3.1 ± 0.9       | 3.1 ± 0.8       |
|                                 | L3               | 5.3 ± 1.6        | 6.0 ± 1.6        | 3.9 ± 1.1       | 3.9 ± 0.9       |
|                                 | L5               | 6.7 ± 1.9*       | 7.3 ± 1.7*       | 4.7 ± 0.9       | 4.6 ± 0.9       |
| Axial rotation S3                | 9.6 ± 1.3        | 9.4 ± 1.7        | 6.2 ± 0.7*       | 5.6 ± 0.9*      |
| Neck angle                       | 94.4 ± 2.6       | 90.6 ± 2.4       | 103.1 ± 1.0*     | 95.1 ± 1.3*     |
|                                 | 78.6             | 76.4             | 84.5             | 81.4             |

*Statistically significant differences between sound and lame condition.

Fig 2: Flexion-extension range of motion (ROM) values (means in degrees) of every vertebral angle at walk (a) and trot (b) in horses before (sound) and during (lame) subtle forelimb lameness induction. *Statistically significant differences between sound and lame condition.

Fig 3: Flexion-extension angular motion pattern (AMP) at T10 and L1 from a horse at trot before (sound) and during (lame) induction of subtle forelimb lameness. Values in square standing position have been used to determine the zero reference value in the AMPs.
condition was significantly smaller, only at T10, L1 and L5. In the lame condition there was a significant increase in lateral bending range of motion at L5 only and no change in axial rotation of the sacral bone (Table 1).

At trot, the overall vertebral flexion-extension ROM increased significantly from 2.8° to 3.1° during lameness (Fig 2). This increase was individually significant at T10 and T13. Besides, there was a significant decrease in the lateral bending range of motion at T10 and in the axial rotation range of motion of the sacral bone (Table 1).

**Vertebral angular motion patterns**

Changes in patterns of vertebral angles were observed at trot. There was a significant increase of 0.9° in flexion at T10 during the stance phase of the sound diagonal. During the entire stance phase of the lame diagonal there was a significantly increased extension of 0.7° at T13 and 0.8° at T17, whereas increased extension of 0.5° at L1, 0.4° at L3 and 0.3° at L5 were seen only at mid-stance, i.e. when the loading of the lame limb is maximal (Fig 3).

There was a significant increase in bending towards the left (which was the lame side) of 1.3° at T10 and of 0.9° at T13 at mid-stance of the lame diagonal. There were no changes in lateral bending in the lumbar region or in the axial rotation of the sacral bone.

**Intravertebral pattern symmetry**

There was a significant decrease in the symmetry of the lateral bending intravertebral pattern at trot during lameness, which was 96% at T10 and 95% at T13, compared with 97% of symmetry in the control measurements for both vertebral angles.

**Neck angle**

The neck had, on average over the entire stride cycle, a lower position only at trot. This is indicated by a reduction of 7.9% in the neck angle (Table 1).

**Discussion**

The results of this study support the hypothesis that subtle lameness results in a measurable change in thoracolumbar kinematics. It was a deliberate choice to induce a very subtle lameness. A severe lameness would have disrupted the entire chain of motion of the various body segments and would, therefore, inevitably have affected back motion as well. Theoretically, a subtle lameness could be absorbed in the proximal parts of the limbs and would then not be transmitted to the trunk and the axial skeleton. A subtle lameness is, by definition, difficult to perceive and grade by the human eye. For this reason a quantitative approach was chosen to define the lameness, based on the fact that a horse with a supporting lameness will always, to some extent, try to remove load from the affected limb (Buchner et al. 1996b). Consequently, knowing the loading of the individual limbs, it is possible to prove or disprove the existence of a supporting lameness. In this study, an objective method, based on the calculations of the ground reaction forces from kinematic data (McGuigan and Wilson 2003; Bobbert et al. 2007) was used. Through this approach, it was possible to demonstrate the existence of a subtle lameness at trot, shown by a reduction of the peak forelimb vertical GRF by 11.5%. Such a reduction was also observed by Weishaupt et al. (2006), who measured horses featuring a subtle forelimb lameness directly with an instrumented treadmill. In that study the peak forelimb vertical GRF decreased by only 4%, but speed was lower (3.5 m/s). In the present study, the subtle lameness did not have a significant influence on the linear and temporal stride variables, which is in agreement with earlier studies on induced lameness (Buchner et al. 1995; Weishaupt et al. 2006).

The flexion-extension range of motion of the whole thoracolumbar vertebral column was significantly increased at trot in the lame condition. This increase was most evident in the cranial thoracic area. It is known that an increased vertical range of motion of the head is an indicator of forelimb lameness (Keegan et al. 2000). By modification of the motion pattern of the head, the horse reduces the load on the painful limb (Buchner et al. 1996a; Vorstenbosch et al. 1997; Keegan et al. 2000). Given the connection of the head to the thoracolumbar vertebral column through the neck, the increased range of motion of the cranial thoracic vertebral column does not come as a surprise. At walk, the overall range of motion was reduced rather than increased. This difference in sign between the adaptation at walk and the adaptation at trot may be explained by the fact that a subtle lameness provokes evident changes in head motion patterns only at trot and not at walk due to the much larger ground reaction forces at trot, implying that there is no need for compensatory movements of the head, neck and back at walk. The slight reduction in flexion-extension range of motion at walk may reflect an overall increase in stiffness of the back as a response to very mild pain sensed by the horse, which may affect in this way the relatively high-amplitude free-swinging motion of the back that is characteristic of the walk in the completely sound horse. Such a reduction of the flexion-extension of the vertebral column could possibly lead to chronic stiffness of the back, chronic back pain and/or persistent rigidity.

At trot, the increased flexion at T10 in the lame condition during the stance phase of the sound diagonal is in line with the lowering of the neck (and head) during the stance phase of the sound limb (Vorstenbosch et al. 1997). As a consequence of this low position of the neck (and head), an increased flexion of the thoracic part of the trunk will be induced (Gómez Álvarez et al. 2006). Furthermore, during the lame diagonal stance phase, the rest of the back (T13, T17, L1, L3 and L5) was more extended while the cranial thoracic area (T10 and T13) was bending laterally towards the lame side. As a compensatory mechanism in lameness, the vertical force of the lame limb shifts to the hindlimbs in the lame diagonal and to the sound forelimb during the sound diagonal (Weishaupt et al. 2006). In addition, an upward movement and a lower peak vertical acceleration of the head help to unload the limb in the vertical plane during the lame stance phase (Buchner et al. 1996a). These compensatory mechanisms can therefore be supposed to induce extension of the vertebral column and a shift of the centre of gravity towards the sound side and towards the hind quarters in the horizontal plane (Marks 1999; Buchner et al. 2001). A shift away from the lame side in the horizontal plane can be carried out only through a lateral bending of the vertebral column with the concave side at the lame side and the convex side at the sound side, i.e. a bending towards the lame side. Such an action could be modulated...
through a contraction of the longissimus lumborum muscle; electromyography studies of this muscle have shown that it may act to limit the lateral bending of the trunk (Tokuriki et al. 1997). The longissimus dorsi muscle may play a role too, as it is mainly responsible for stabilisation of the vertebral column in a response to dynamic forces (Licka et al. 2004).

Some changes in angular motion patterns at individual vertebrae were not statistically significant in this study, but these findings were consistently observed in consecutive vertebral segments. When taken as a whole, there was a significant change in thoracolumbar motion in those cases. It should be emphasised that the changes were provoked by an intentionally very slight lameness, hardly perceptible to the eye, and that only acute effects were measured. If a horse suffers from a chronic ailment of the limb, as is invariably the case in clinical cases, long-term adaptation processes might ultimately lead to chronic back problems, recurrent acute episodes of soreness, or just permanent minor spinal muscle pain due to asymmetrical loading of the spine. Such conditions might affect the performance of horses and provoke biomechanical compensations, which could lead to pathologies in other areas.

It is concluded that subtle forelimb lameness affects both the range of motion and the vertebral angular motion patterns to a limited, but statistically significant extent. Not surprisingly, the effect is best detectable in the cranial thoracic region. When the sound diagonal is loaded, horses tend to flex the cranial thoracic back, which follows the movement of the neck and head downwards. When the lame diagonal is loaded, they extend the rest of the back, shifting the mass to the hind quarters away from the painful limb. They will further increase lateral bending towards the lame diagonal when it is loaded, bringing the centre of gravity more towards the sound side. The changes are relatively minor in extent, but might affect muscular tension and vertebral function when present for a prolonged period in cases of chronic lameness. The observations therefore lend credibility to the alleged implication of subclinical lameness in the pathogenesis of vertebral dysfunction in horses. However, further research is necessary to demonstrate this relationship unequivocally and to understand the long-term adaptation processes.

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References


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