Chapter 4

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

Under revision

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Summary

Reasons for performing the study: Hindlimb lameness has often been suggested to cause altered motion of the back, but there are no detailed studies describing such a relationship.

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

Methods: Kinematics of 6 riding horses was measured during walk and trot on a treadmill before, during and after the application of pressure on the sole of the left hindlimb using a well-established sole presure model. Reflective markers were located at anatomical landmarks on the limbs, back, head and neck for kinematic recordings. Ground reaction forces (GRF) in individual limbs were calculated from kinematics to detect changes in loading of the limbs.

Results: When pressure on the sole of the hindlimb was present, the horses were judged as lame (grade 2 on the AAEP scale 1-5) by an experienced clinician. No significant unloading of this limb was found in the group of horses (unloading was observed in 4 and it was not detectable in the other 2), but statistically significant effects on back kinematics were detected. The overall flexion-extension (FE) range of motion (ROM) of the vertebral column was increased at walk, especially in the thoracic segments. Axial rotation (AR) ROM of the pelvis was also increased. At trot, the FE ROM was decreased only in the segment L3-L5-S3. During the stance phase of the lame limb, the segment T6-T10-T13 was more flexed and the neck was lowered at both gaits; the thoracolumbar segments were more extended at walk and trot. There were no significant changes in the stride length or protraction-retraction angles in any of the limbs.

Conclusions: Subtle hindlimb lameness provoked slight but detectable changes in thoracolumbar kinematics. The subtle lameness induced in this study resulted in hyperextension and increased ROM of the thoracolumbar back, but also in decreased ROM of the lumbosacral segment and rotational motion changes of the pelvis.

Potential relevance: Even subtle lameness can result in changes in back kinematics, which emphasises the intricate link between limb function and thoracolumbar motion. It may be surmised that, when chronically present, subtle lameness induces back dysfunction.

Keywords: Back kinematics, riding horses, induced hindlimb lameness.

Introduction

Horses modify their gait mechanics to compensate for any injury or source of pain. Lameness is one of the most common symptoms of a locomotor disorder affecting mechanics of the entire body. Horses try to cope with lameness by several mechanisms all aiming to unload the painful limb. Compensatory mechanisms in limb loading have been described by Weishaupt *et al*. (2004), who showed a reduction of the total vertical impulse per stride, a reduction of the impulse in the lame diagonal stance phase, shifting of the impulse within the lame diagonal to the forelimb and in the sound diagonal to the hindlimb, and a decrease of the loading rate by the increase of the stance duration. In order to allow for these changes in limb loading, the motion of other parts of the body need to be changed, and these changes can to a certain extent become detectable during clinical examinations. Kinematical studies have shown that in hindlimb lameness at trot the croup is elevated at first contact of the lame limb, and is lowered during weight-bearing to descend further during the stance phase of the sound limb (Buchner *et al*. 1996a; Cadiot and Almy 1924). The head and neck are lowered during the stance phase of the lame hindlimb (Cadiot and Almy 1924; Denoix and Audigie 2001; Uhlir *et al*. 1997), and this motion pattern changes from the normal biphasic sinusoidal pattern to a curve with only a single elevation at the beginning of the sound diagonal (Denoix and Audigie 2001), likewise the trunk pattern (Buchner *et al*. 1996a). Adaptations in head and trunk movements occur in both forelimb and hindlimb lameness (Buchner *et al*. 1996a; Uhlir *et al*. 1997), together with changes in the angular motion patterns of the limbs and spatial and temporal stride parameters (Buchner *et al*. 1995; 1996b).

Given the central position of the thoracolumbar spine in the body, lameness will forcibly often affect the motion pattern of the vertebral spine, which may be manifested as a reduction in the horse's performance. Although there is no doubt that a close relationship exists between back problems and lameness, there is great need in equine practice to improve the understanding of this relationship. Landman *et al*. (2004) reported that the prevalence of lameness in horses with back problems was 74%, while the prevalence of back problems in horses with lameness was 32%. Data on lameness-induced changes in thoracolumbar motion are scarce. Pourcelot *et al*. (1998) reported in a descriptive study in a single horse that the thoracolumbar back showed less extension during the lame diagonal stance phase, but increased extension during the sound diagonal stance phase at trot. There were no data on the walk in that study.

Clinically, moderately lame horses demonstrate altered movement of the back, head and neck. In the case of hindlimb lameness we would further expect an increase in axial rotation of the pelvis, following ascending and descending motion of the croup. The effect of a very subtle lameness on spinal kinematics is more difficult to predict. With a low-grade subtle lameness the changes in back movement, if present, are presumably much smaller and most probably difficult to detect with the naked eye. Nevertheless, even small changes in back motion, when chronically present, could result in an injury or vertebral disorder. Therefore, detailed studies are necessary to fully understand the effects on back kinematics of both forelimb and hindlimb lameness. In a previous study, we showed that subtle forelimb lameness provokes changes in the thoracolumbar motion pattern and increases the range of motion in the vertical and horizontal planes at trot (Gómez Álvarez *et al*. 2007a). It is conceivable that hindlimb lameness will have a similarly effect on back motion too. However, the character of any effects might be different, given the anatomical differences between fore and hindlimbs, in particular with respect to their anatomical connection to the body.

The aim of this study was to investigate the effect of artificially induced subtle hindlimb lameness on back motion. Quantification of this effect may improve our understanding of back problems secondary to lameness. Our hypothesis was that even a subtle lameness would result in a measurable change in thoracolumbar kinematics. To test this hypothesis, we analysed the kinematics of 6 riding horses walking and trotting on a treadmill before and during the induction of reversible subtle hindlimb lameness.

Materials and Methods

Horses and general experimental design

Back and limbs kinematics were measured in six sound Dutch Warmblood horses, 11.7 ± 4.9 years of age, with a height at the withers of 163 ± 4.8 cm, and a body mass of 577 ± 37.1 kg, while walking (1.6 m/s) and trotting (4.0 m/s) on a treadmill before, during and after the induction of lameness. The horses had been trained previously on the treadmill in order to get them accustomed to treadmill locomotion. The Experimental Animals Commission of Utrecht University had approved the experimental protocol.

Lameness induction
Reversible lameness was induced in the left hindlimb 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 transient pain. A more extensive description of the technique can be found elsewhere (Merkens and Schamhardt 1988). The lameness provoked was grade 2 on 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)).

Evaluation of lameness
During a supporting limb lameness the horse tries to reduce the load of the painful limb (Buchner *et al*. 1996b). Loads of individual limbs were calculated from kinematics as proposed elsewhere (Bobbert *et al*. 2007; McGuigan and Wilson 2003). Briefly, distal limb length of the forelimb was measured using markers on the hoof and elbow joint, and of the hindlimb using markers on the hoof and stifle joint. 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 two 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. The GRFs were calculated at walk from distal limb length assuming that the distal limbs operate as linear springs. The force-length relationships that emerged from these calculations were used to calculate individual limb GRF 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 19 mm diameter markers 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). Extra markers were placed on the coxal tuberosities, the lateral sides of the hooves; on the limbs for load calculations (Bobbert and Santamaria 2005), and on the atlas for the neck angle calculation. Six infrared cameras situated on both sides of the treadmill recorded the horses while standing square and while walking and trotting on the treadmill before, during and after lameness induction. The recordings were performed during 10 seconds after the first minute of locomotion on the treadmill. After capturing data in the non-lame reference condition the treadmill was stopped for 1 minute to allow

for tightening of the bolt, inducing lameness, and again after the lame condition to allow for removal of the bolt.

Data analysis

Qualisys Track Manager Software[1] was used to capture and process the data and Matlab®[2] for further analyses. A standard right-handed orthogonal Cartesian coordinate system was used to describe the motion of the vertebral column. Angular motion patterns (AMP) were described as flexion-extension (in the sagittal plane), lateral bending (in the medio-lateral plane), and pelvic axial rotation (S3 with respect to the position of the markers on the coxal tuberosities looked from behind of the horse). All AMP were calculated using Backkin®[1] as means and standard deviations during several averaged stride cycles. The changes in the AMP were described according to the phase of the stride cycle when these occurred. Data captured in the square standing horse before and after the lameness induction were used to determine the reference (zero) value of the AMPs in each horse. The range of motion (ROM) was determined from each AMP by taking the difference between the maximal and minimal AMP values. Each vertebral angle was defined as the angle between three adjacent marked vertebrae (e.g., the angle at T10 is the angle between the line from T10 to T6 and the line from T10 to T13). The beginning of each stride cycle was defined as the instant of initial ground contact of the left hindlimb. The correlation coefficient between the vertebral angular motion patterns was calculated to quantify the intra-vertebral pattern symmetry (Faber *et al*. 2000). The neck angle with respect to the horizontal plane was calculated using the markers on T6 and atlas. Stride length was calculated from the hoof marker on the left hindlimb. Protraction-retraction angle was calculated for each of the forelimbs using the markers on the hooves and T6, and for each of the hindlimbs using the markers on the hooves and S3.

The distribution of values for kinematic variables and calculated forces was tested for normality. If normally distributed, analysis was carried out using ANOVA for repeated measures and a Bonferroni correction. 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 for all tests was set at $p<0.05$.

Results

Evaluation of lameness

During the pain induction, each of the horses was judged to be lame by an experienced clinician. The mean peak vertical ground reaction force on the painful hindlimb was 9.3±1.3 N/kg at trot and 4.3±0.5 N/kg at walk before lameness induction, and 9.0.±0.9N/kg at trot and 4.4±0.4 N/kg at walk during lameness, which was not a statistically significant difference. Of 6 horses, 4 showed reduced peak load on the lame limb at trot and 5 at walk, 2 (walk: one) did not show changes in the measured peak load on the lame limb. These horses increased however the peak load on the non-lame hindlimb.

Stride length and protraction retraction angle

Both protraction-retraction mean angles and stride length remained unchanged at both gaits (Table 1).

Vertebral ranges of motion

At walk, the induced lameness provoked an overall increase of the flexion-extension ROM of the vertebral column from 6.2 to 6.6 degrees (mean). A significant increase was also found at individual thoracic segments: at T10, T13 and T17. Further, the axial rotation range of motion of the pelvis was increased. At trot, the overall ROM did not differ from the sound condition, only the flexion-extension ROM at L5 was decreased (Fig. 4.1a, b) (Table 1).

Vertebral angular motion patterns

Several changes ($p<0.05$) in the mean angular motion patterns of the vertebrae were found at walk and trot after lameness induction (Fig. 4.2). Flexion at T10 was increased by 0.8 degrees (mean of all horses) during the stance phase of the lame limb at trot and by 0.3 degrees at walk. An increase in extension was observed in the thoracolumbar area also at both gaits: at walk, there was an increase in the extension at T13 (0.5 degrees), T17 (0.7 degrees) and L1 (0.7 degrees) at the middle of the stance phase of the left (lame) hindlimb and again at the middle of the stance phase of the right (sound) hindlimb (0.8, 0.8 and 0.6 degrees, respectively), while extension at L3 and L5 was 0.6 degrees greater during the whole stance phase of the lame hindlimb (Fig. 4.2a, b). At trot, extension at T13 and T17 was increased by 0.2 and 0.3 degrees respectively, during most of the sound diagonal. No changes were observed in the lumbar segment at trot.

Table 4.1. Range of motion (ROM) values (mean ± SD, degrees) for selected individual vertebrae, neck angles (degrees), stride length (meters) and protraction-retraction angles (degrees) at walk and trot in six horses with induced subtle hindlimb lameness.

Motion		Walk		Trot	
		Sound	lame	sound	lame
Flexion-extension	T10	5.1±1.0*	5.9±1.1*	3.4±0.6	3.6±0.8
	T13	6.3±0.6*	7.0±1.1*	2.3±0.7	2.5±0.7
	T17	6.8±1.0*	7.4±1.3*	2.3±0.4	2.4±0.4
	L1	6.6±1.4	7.0±1.6	2.8±1.0	2.8±0.6
	L3	6.4±2.0	6.4±1.7	2.9±0.7	2.9±0.6
	L5	5.9±2.0	5.9±1.6	2.9±0.7*	2.6±0.8*
	Overall variation	6.2±1.3*	6.6±1.4*	2.8±0.7	2.8±0.7
Lateral bending	T10	8.9±1.9	9.3±1.6	7.2±1.2	7.5±2.2
	T13	5.0±1.0	5.3±0.9	4.3±1.3	4.3±1.2
	T17	3.2±0.9	3.3±1.2	3.3±0.9	3.3±0.9
	L1	4.0±1.4	3.9±1.5	3.1±0.9	3.2±0.8
	L3	5.3±1.6	4.9±1.6	3.9±1.1	3.8±1.1
	L5	6.7±1.9	6.4±1.3	4.7±0.9	4.5±1.1
Pelvic axial rotation	S3	9.6±1.3*	11.4±1.7*	6.2±0.7*	6.0±0.9*
Neck angle		94.4±2.6	89.9±2.3	103.1±1.0*	89.8±1.4*
Stride length		1.8±0.2	1.9±0.1	2.8±0.1	2.8±0.1
Protraction-retraction angles	Right hindlimb max protraction max retraction ROM	 14.3 -26.2 40.5	 14.5 -25.8 40.3	 12.5 -25.9 38.4	 12.5 -25.9 38.4
	Left hindlimb max protraction max retraction ROM	 15.6 -23.9 39.5	 11.2 -25.1 39.3	 11.4 -26.7 38.1	 11.8 -25.5 37.3
	Right forelimb max protraction max retraction ROM	 16.7 -23.1 39.8	 17.6 -22.4 40.0	 16.9 -23.6 40.4	 17.6 -22.9 40.5
	Left forelimb max protraction max retraction ROM	 16.8 -22.5 39.3	 16.3 -22.2 38.5	 17.8 -24.9 42.7	 19.6 -23.3 42.9

* Statistically significant differences between sound and lame condition.

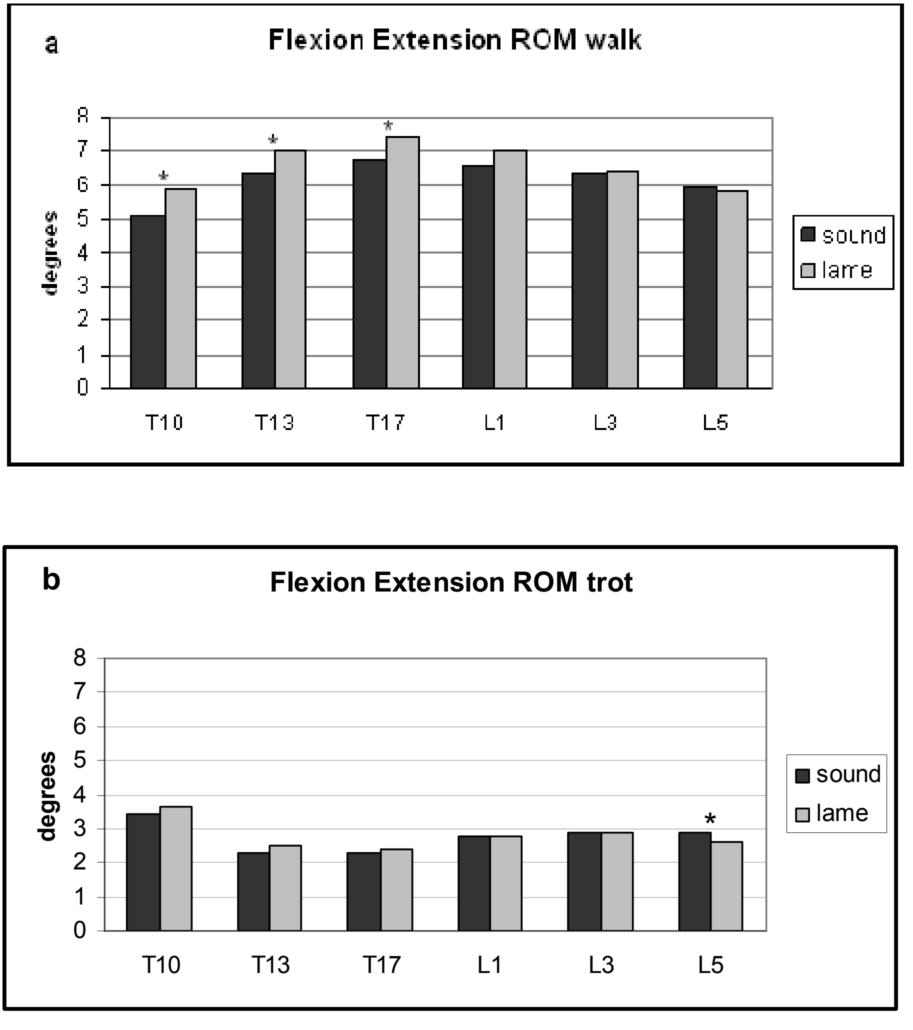

Figure 4.1. Flexion-extension range of motion (ROM) for each vertebral angle in 6 horses before (sound) and during (lame) subtle lameness caused by induced pain in the hindlimb, at a) walk and b) trot. Significant differences between sound and lame condition are indicated with *

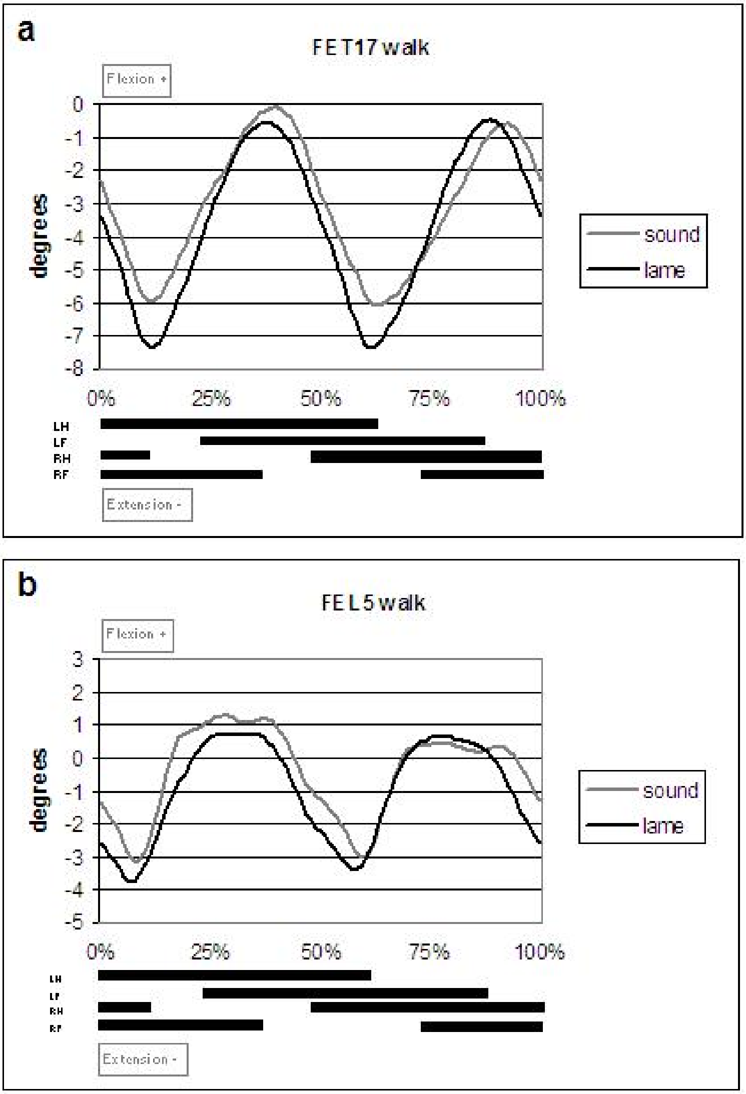

Figure 4.2. Flexion-extension angular motion patterns (AMP) of one horse at walk before (sound) and during (lame) subtle lameness caused by induced pain in the hindlimb: a) angle at T17 (T13-T17-L1) and b) angle at L5 (L3-L5-S3). The black bars at the bottom of the graph indicate the stance phases of the limbs.

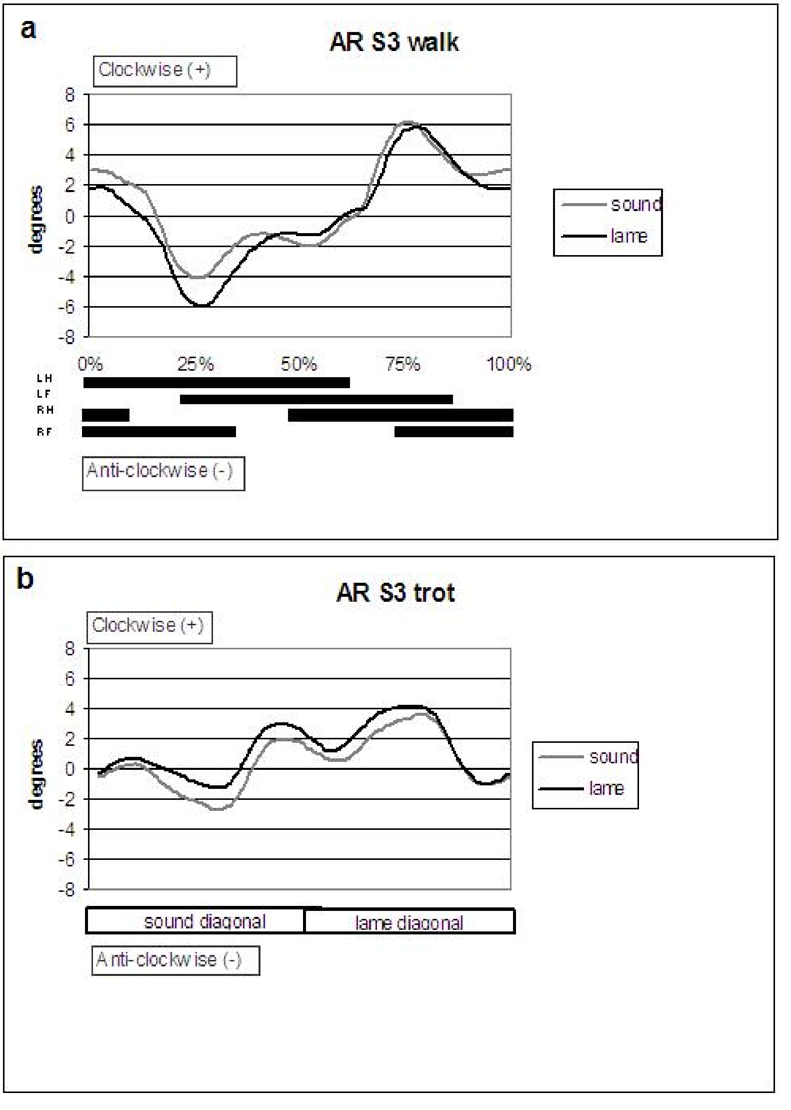

Figure 4.3. Axial rotation of the pelvis of one horse at a) walk and b) trot; before (sound) and during (lame) subtle lameness caused by induced pain in the hindlimb. Clockwise and anticlockwise directions are seen from behind of the horse. The black bars at the bottom of the graph indicate the stance phases of the limbs.

At walk, lateral bending was increased to the left (lame side) at T13 by 0.3 degrees during the end of the stance phase and the beginning of the swing phase of the lame hindlimb.

Changes in pelvic motion during walk were opposite to those during trot. During the stance phase of the lame hindlimb at walk, anticlockwise (looked from behind of the horse) rotation of the pelvis was increased by 1.2 degrees (mean); while at trot it was rotated 0.6 degrees (mean) clockwise during both diagonals (Fig. 4.3a, b).

Intravertebral pattern symmetry

The mean intravertebral pattern symmetry was unchanged by the induced lameness in most of the vertebral angles. However, it diminished at T13 for lateral bending during trot (from 97% to 94%; $P<0.05$). No changes in intravertebral pattern symmetry were observed at walk.

Neck angle

At trot, the neck was lowered during the entire stride in the lame condition. The neck angle changed on average 13.3 degrees.

Discussion

The hypothesis that subtle hindlimb lameness would result in a measurable change in thoracolumbar kinematics was supported by the outcome of this study. The induction of this subtle lameness did not result in all horses showing measurable changes in limb loading, though judged to be clinically lame. A consistent sign of lameness shown by the horses of this study was the lowering of the neck, especially at the lame diagonal at trot. This phenomenon has been described earlier (Denoix and Audigie 2001; Vorstenbosch *et al*. 1997). Stride length was not affected by the lameness, which is in agreement with other studies of induced subtle lameness (Buchner *et al*. 1995; Weishaupt *et al*. 2004). Also, protraction-retraction angle was not changed due to lameness. These somewhat inconclusive findings concur with the AAEP definition of this degree of lameness (lameness difficult to observe at a walk or trot in a straight line (Stashak 2002)), and confirm that lameness was indeed subtle. Nevertheless, detectable and systematic changes occurred in kinematics of the back at both gaits. In fact, the two horses that did not show changes in limb loading showed more apparent changes in their vertebral motion. It may be that some horses respond to a subtle lameness by changing the loading of the lame limb, while others do not change limb kinetics but instead change back kinematics and pelvic motion. The latter mechanism may affect gait relatively slightly, but does not necessarily lead to more appropriate locomotion.

Most of the changes we found in vertebral column kinematics are in agreement with the literature. Our finding of a greater averaged flexion at T10 combined with less elevation of the neck and head during the lame stance phase, is in accordance with the observation that lowering the neck helps to flex the cranial back (Gómez Álvarez *et al*. 2006). Our finding of a more extended thoracolumbar back at both gaits during the stance phase of the sound hindlimb agrees with the results of the lameness study of Pourcelot *et al*. (1998). In contrast to that study we did not find reduced extension during the lame diagonal stance phase, but this may well be due to the fact that lameness was less severe in our study, causing the associated kinematical changes to be less pronounced. The increased extension in the lumbar segment observed in the present study at walk is a new finding. Thus far, there were no reports on the effects of subtle lameness on back kinematics at walk. The increased extension may be a sign of overall stiffening of the hind quarters as a reaction to the induced pain in the sole. These changes in the motion of the lumbar segment were not seen at trot, probably because trot is an altogether different gait than walk with only two limbs on the ground simultaneously and with considerably less motion of the back (Faber *et al*. 2002) due to increased muscle activity (Robert *et al*. 2002).

The changes in range of vertical motion were different between gaits; the range increased at walk but did not change at trot. At trot, lowering the neck may to a certain extent help to avoid loading the painful limb. At walk, a similar effect may possibly be achieved by increasing the back ROM in the vertical plane. These changes occurred mainly in the thoracic area and might perhaps be interpreted as a compensatory mechanism to the tension in the lumbar area which was more extended than normal, as pointed out above. In the horse, an increased lumbar extension may go along with increased vertical ROM in the back, as has been shown earlier (Gómez Álvarez *et al*. 2006).

The changes in thoracolumbar kinematics provoked by subtle hindlimb lameness differ from those induced by forelimb lameness (Gómez Álvarez *et al*. 2007a). It is conceivable that hindlimb lameness produces more tension in the back than forelimb lameness because of the direct bony connection of back and hindlimb through the pelvis. In the fore quarters no such bony connection exists and in forelimb lameness the load on the painful limb can more easily be reduced by changing the head and neck motion, without a severe impact on thoracolumbar kinematics. The tight anatomical connection of the hindlimbs to the pelvis (and thus to the vertebral column) can explain the decrease of FE ROM at L5 at trot and the changes in the pelvic range of motion and the pattern of pelvic rotation at both gaits. At walk AR ROM of the pelvis was increased and it was rotated on average more to the lame side during the lame stance phase. At trot this AR ROM was not changed, but it was rotated to the sound side during the entire stride. Buchner *et al*. (1996a) found a reduction of the vertical displacement of the pelvis at the lame side combined with increased rotational movements. The clockwise rotation of the pelvis as found in the present study at trot is in agreement with this, as it is a rotation towards the sound side. However, vertebral reaction to lameness at walk seems to follow another mechanism and is partly effectuated through a change in lateral bending which concurs with an increase in AR of the pelvis to the same side.

In conclusion, induced subtle hindlimb lameness results in changes in thoracolumbar kinematics without necessarily producing detectable changes in the kinematics of the limbs or in ground reaction forces. Hindlimb lameness resulted in subtle back increase in extension, a slightly increased range of motion of the thoracolumbar back, a slightly decreased range of motion of the lumbosacral segment and rotational motion changes of the pelvis.

This study investigated acute effects of lameness, and long-term effects may be different. It may be presumed that also in the chronic situation compensatory changes in back kinematics will occur, which, if present for a prolonged period, might contribute to the pathogenesis of chronic back dysfunction.

Manufacturers' addresses

1 Qualisys Medical AB, Gothenburg, Sweden.
2 Matlab® (The MathWorks, Inc. Natick, Massachusetts)

Acknowledgements

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