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Hindlimb response to tactile stimulation of the pastern and coronet

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Summary

Reasons for performing study: **Lightweight tactile stimulators attached to the hind pasterns increase the height of the hind hoof flight arc but details of the induced changes in swing phase kinematics and kinetics have not been investigated.**

Hypotheses: **Stimulators on the hind pasterns are associated with increased hindlimb joint flexions and increased positive work performed by the hip and tarsal musculature.**

Materials and methods: **Nine nonlame horses trotted 4 times with and without 55 g tactile stimulators loosely attached around the hind pasterns. Height of the flight arc and peak flexion angles of the hindlimb joints were measured and net positive and negative work performed across each joint during the swing phase were calculated using inverse dynamics analysis and compared across paired conditions.**

Results: **Speed and stride duration did not change but stimulators were associated with a reduction in hind stance duration. The flight arc was higher with stimulators due to increased flexions of the stifle, tarsal, metatarsophalangeal and distal interphalangeal joints. Positive work increased in the tarsal musculature, but not in the hip musculature, and negative work increased across the stifle, metatarsophalangeal and distal interphalangeal joints.**

Potential relevance: **The effects of tactile stimulation of the hind pasterns on joint motion and muscle activation may be used in physiotherapy and rehabilitation to restore or increase flexion of the hindlimb joints with the exception of the hip joint. The ability to stimulate concentric activity of the tarsal musculature may have therapeutic applications in conditions such as toe dragging.**

Introduction

Simple techniques to increase ranges of joint motion and facilitate muscle activation have potential applications in the restoration of locomotor function following injury or immobilisation (Goff and Stubbs 2007). A preliminary study showed that lightweight tactile stimulating devices attached loosely around the pasterns increased the height of the flight arc of the hooves in both the fore and hindlimbs, with the response being more exaggerated and persisting over a longer period of time in the hindlimbs (Clayton *et al.* 2008). The objective of this study was to describe in detail the effects of tactile stimulation of the hind pastern during trotting on limb kinematics and on net joint powers calculated using inverse dynamics analysis, which gives an indication of the muscle groups responsible for any changes in motion patterns.

The 3 joints in the proximal hindlimb, hip, stifle and tarsus, are each involved almost exclusively in either generation of mechanical energy (positive work) or absorption of mechanical energy (negative work) during the swing phase at trot (Clayton *et al.* 2002; Dutto *et al.* 2006). The musculature of the hip and tarsus acts concentrically to generate energy and drive the swing phase movements of the hindlimb. In early swing, positive work performed by the hip flexors protracts the hindlimb and positive work performed by the tarsal flexors raises the hind hoof clear of the ground. Later in the swing phase, the hip and tarsal extensors perform positive work to retract the limb and lower the hoof towards the ground. Movements of the stifle and tarsal joints are synchronised mechanically by the reciprocal apparatus, which also coordinates flexion of the metatarsophalangeal (MTP) joint. Extension of the MTP joint and movements of the DIP joint are driven inertially in the swing phase. The action of the musculature at the stifle, MTP and DIP joints is primarily eccentric: the extensor muscles do negative work to control joint flexion and the flexor muscles do negative work to control extension (Clayton *et al.* 2002; Dutto *et al.* 2006).

A large range of motion in flexion in the stifle and tarsal joints of the hindlimbs is considered a desirable characteristic in sport horses during both stance and swing phases (Back *et al.* 1994; Holmström *et al.* 1994; Morales *et al.* 1998), whereas lameness is often associated with changes in limb kinematics, including reduced ranges of joint motion (Buchner *et al.* 1996a; Clayton *et al.* 2000). When lameness resolves, the range of motion may not return to pre-injury values. For example, immobilisation of one fetlock of sound horses in a cast for 7 weeks resulted in a restricted range of joint motion that was not restored during 8 weeks of progressively increasing exercise (van Harreveld *et al.* 2002). In man, long-term changes in kinematic patterns that follow immobilisation or injury are due to altered mechanical properties of the articular and periarticular structures (Akeson *et al.* 1987; Michlovitz *et al.* 2004; Katalinic *et al.* 2009) or alterations in neuromotor control (e.g. Jerosch and Prymka 1996; Cowan *et al.* 2001) that may be present even in the absence of pain. Evidence of

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an underlying neuromotor control problem has been shown in human patients with back and knee pain. For example, individuals with patellofemoral pain syndrome have abnormal knee joint position sense or joint proprioception (Baker *et al.* 2002), altered electromyographic (EMG) firing patterns of *vastus medialis obliquus* and *vastus lateralis* that contribute to maltracking of the patella (Cowan *et al.* 2001; Owings *et al.* 2002), and reduced knee flexion in early stance due to the delay in onset of eccentric activity in *vastus medialis obliquus* (Crossley *et al.* 2004). In addition, strength is significantly decreased in the more proximal musculature, including hip external rotators and abductors (Ireland *et al.* 2003). It seems likely that similar neuromotor control problems, including altered joint proprioception and EMG firing patterns, may underlie and confound many gait abnormalities and disease processes in horses, such as intermittent upward fixation and chondromalacia of the patella (Latimer 2004) and back pain, including sacroiliac disease (Jeffcott and Haussler 2004; Goff *et al.* 2008). The equine hindlimb is actively stabilised during standing by activity of *vastus medialis* (Schuurman *et al.* 2003), but there is a lack of information describing EMG activity during locomotion, with or without dysfunction. Clinically stifle dysfunction may be associated with poor quadriceps tone, that may be a consequence of disuse atrophy from inactivity, injury or neurological disease (Walmsley 2003; Latimer 2004). As in man, there is also a very strong biomechanical interaction between the vertebral column and the limbs in horses (Gomez Alvarez *et al.* 2007a,b; 2008). For example, subtle lameness changes back kinematics (Gomez-Alvarez *et al.* 2007a; 2008) and induced back pain alters limb kinematics (Gomez Alvarez *et al.* 2007b) due to altered neuromotor control.

Therapeutic exercises targeted toward activation of specific muscles or muscle groups would be a useful addition to the techniques available to the equine veterinarian and physiotherapist. The study reported here is part of a broader research initiative to develop simple rehabilitation techniques that will facilitate restoration of normal locomotor function by activating and strengthening specific muscle groups. The experimental hypotheses were that tactile stimulation of the hind pastern during trotting will be associated with 1) increased swing phase flexions of the joints of the hindlimb and 2) increased positive work in the hip and tarsal musculature.

Fig 1: Stimulation device (mass 55 g) consisting of braided strap with 7 lightweight brass chains is shown attached loosely around the pastern with the chains hanging over the coronet onto the hoof wall.

Materials and methods

The study was approved under protocol 06/04-098-00 by the institutional animal ethics committee.

Horses

The subjects were 9 horses (4 mares and 5 geldings; height: 1.49 ± 0.07 m; mass: 428.8 ± 30.0 kg) that were assessed by an experienced observer to be nonlame at trot.

Tactile stimulation

Lightweight (55 g) tactile stimulators, consisting of a 1 cm wide braided nylon strap with 7 double strands of lightweight brass chain, 6 cm in length, were attached loosely around the hind pasterns (Fig 1). The chains brushed against the pastern and coronet when the hoof moved. The horses were accustomed to the sound and feel of the stimulators the day before data were collected.

Prior to data collection, 19 reflective markers were attached to the skin of the head, trunk and hindlimbs. Midline markers were attached to the forehead and overlying the spines of the tenth thoracic vertebra (T10) and sixth lumbar vertebra (L6). Markers were attached bilaterally over the lower part of the *tuber coxae*, cranioventral part of the greater trochanter, lateral femoral condyle, lateral talus and lateral condyle of the third metatarsus. In addition, 3 markers were attached to each hind hoof: 4 and 6 cm above the toe on the dorsal midline and mid-laterally 2 cm above the ground. Lateral radiographs of the hind hooves were taken with the hoof markers in place and with the horses standing on wooden blocks. During post processing, custom software was used to locate the centre of rotation of the distal interphalangeal (DIP) joint (Buchner *et al.* 1996b) and to determine hoof segment angulation based on the hoof marker positions.

Data collection

Kinematic data were collected using an automated motion analysis system[1] after calibration of the data collection volume (5 × 3 × 2 m) using a wand technique. The error in a linear measurement of 1.0 m within the calibrated volume was <0.8 mm. Data were collected under 2 conditions: with and without tactile stimulators attached to both hind pasterns. The 2 conditions were blocked within horses and tested in random order.

The first recording was a stationary file that was used to develop a marker template and to determine standing joint angles. Horses then trotted in hand along a rubberised runway with 4 consecutive trials of 30 m being recorded for each condition. This is a sufficient number of trials to establish representative kinematic data (Drevemo *et al.* 1980) but insufficient for the horses to habituate to the stimulators (Clayton *et al.* 2008). Immediately after each trial, the handler received feedback describing the average speed of the marker on T10 during that trial to facilitate consistency in speed across trials. When stimulators were used, they were attached to the pasterns after the horse had been positioned on the runway and was ready for the first trotting trial. The stimulators were removed before returning the horse to the stable. The horses rested in stables for 2 h between the 2 recording sessions.

Data analysis

Marker coordinate data were filtered using a Butterworth lowpass digital filter with cutoff frequency 12 Hz and corrections for skin displacement were applied to the markers on the *tuber coxae*, the greater trochanter and the lateral femoral condyle (van Weeren *et al.* 1992), then one stride from each of the 4 trials was analysed using proprietary[1] and custom software. General stride kinematics were described in terms of the horse's average speed and stride duration, hind stance and swing durations, and maximal height of the distal toe marker. Maximal protraction and retraction angles of the hindlimb were measured as the angles between the vertical and a line connecting the marker on the greater trochanter with the marker over the centre of rotation of the DIP joint. Protraction was assigned a positive value, retraction a negative value. Swing phase joint angles for the hip, stifle, tarsal, MTP and DIP joints were measured on the flexor aspect and expressed relative to the standing angle derived from the stationary file. The anatomical flexor side was caudal/plantar for the stifle, MTP and DIP joints and cranial/dorsal for the hip and tarsal joints. Relative to the standing angle, extension was assigned a positive value and flexion was assigned a negative value. Peak flexion and extension were determined as the minima and maxima, respectively, of the angle-time curves in the swing phase. Absolute joint angles were used in an inverse dynamics analysis to calculate net work done across each joint in the swing phase (Lanovaz *et al.* 1999). Failure to account for the mass of the stimulators could result in significant errors in the inverse dynamic calculations (Lanovaz *et al.* 2001). Therefore, a correction for stimulator mass was applied as described by Singleton *et al.* (2003).

TABLE 1: Mean ± s.d. of temporal and linear gait variables for horses trotting in hand without stimulators and with tactile stimulators on both hind pasterns. Asterisks indicate variables that differ significantly (P<0.05)

	No stimulators	Hindlimb stimulators
Speed (m/s)	3.06 ± 0.24	3.12 ± 0.22
Stride duration (s)	0.70 ± 0.04	0.70 ± 0.04
Stance duration (s)	0.29 ± 0.02*	0.27 ± 0.02*
Swing duration (s)	0.41 ± 0.03*	0.43 ± 0.04*
Hindlimb stance duration (% stride)	41.09 ± 2.37*	38.60 ± 2.95*
Hindlimb heel off (% stance)	79.93 ± 3.35*	81.88 ± 2.53*
Peak height of hind hoof (mm)	48.58 ± 10.83*	144.35 ± 68.20*

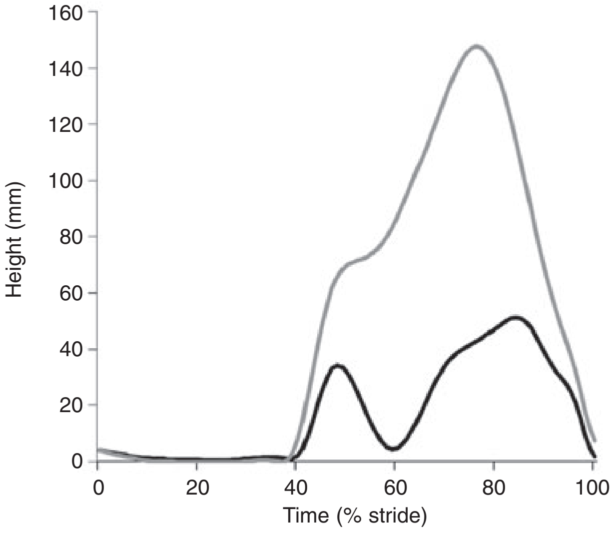

Fig 2: Graph of hind hoof height of one horse (mean of 4 trials) without stimulators (black line) and with stimulators on both hindlimbs (grey line).

Statistical analysis

For each condition, data for the 4 strides of the left and right hindlimbs were ensemble averaged to calculate descriptive statistics (mean, s.d.) for the measured variables[2]. Data were tested for normality of distribution using the Shapiro-Wilks test. Differences between the 2 conditions were sought using paired *t* tests for variables with a normal distribution and a Wilcoxon Signed Ranks Test for variables that did not show a normal distribution. A probability value of P<0.05 was used for all statistical tests.

Results

Speed and stride duration did not differ between conditions, but stance duration decreased and swing duration increased when stimulators were present. Peak height of the hind hooves during the swing phase increased approximately 3-fold when the horses wore stimulators (Table 1, Fig 2). The flight arc of the hoof without stimulators showed 2 peaks separated by a well-defined dip with maximal height being attained during the second peak. When stimulators were present, the hoof was raised more rapidly in early swing and the dip between the 2 peaks tended to disappear (Fig 2).

The increase in hoof height when wearing stimulators was associated with significant increases in peak flexion of the stifle, tarsal, MTP and DIP joints throughout the swing phase (Figs 3, 4, Table 2). Maximal flexion of the tarsal and stifle joints increased

TABLE 2: Mean ± s.d. of peak values of joint angles measured relative to standing angles in the joints of the hindlimbs in the swing phase of the stride for 9 horses trotting in hand without stimulators and with tactile stimulators on both hind pasterns. Negative values indicate flexion, positive values indicate extension relative to the standing angle. Asterisks indicate pairs of variables that differ significantly (P<0.05)

	No stimulators	Hindlimb stimulators
DIP min angle (deg)	-15.19 ± 8.79*	-19.10 ± 6.39*
DIP max angle (deg)	11.78 ± 8.56	8.33 ± 8.04
MTP min angle (deg)	-26.99 ± 7.48*	-40.47 ± 13.08*
MTP max angle (deg)	32.26 ± 9.24*	26.89 ± 9.88*
Tarsus min angle (deg)	-44.23 ± 3.13*	-59.24 ± 8.86*
Tarsus max angle (deg)	1.94 ± 1.83	2.16 ± 2.49
Stifle min angle (deg)	-36.42 ± 3.20*	-44.99 ± 4.64*
Stifle max angle (deg)	12.40 ± 2.42	12.58 ± 3.03
Hip min angle (deg)	-43.69 ± 3.94	-44.84 ± 4.23
Hip max angle (deg)	-24.97 ± 3.22	-24.88 ± 2.95
Maximal protraction (deg)	21.64 ± 2.42	21.49 ± 2.67
Maximal retraction (deg)	-19.79 ± 2.33	-20.19 ± 2.17

DIP: distal interphalangeal joint; MTP: metatarsophalangeal joint.

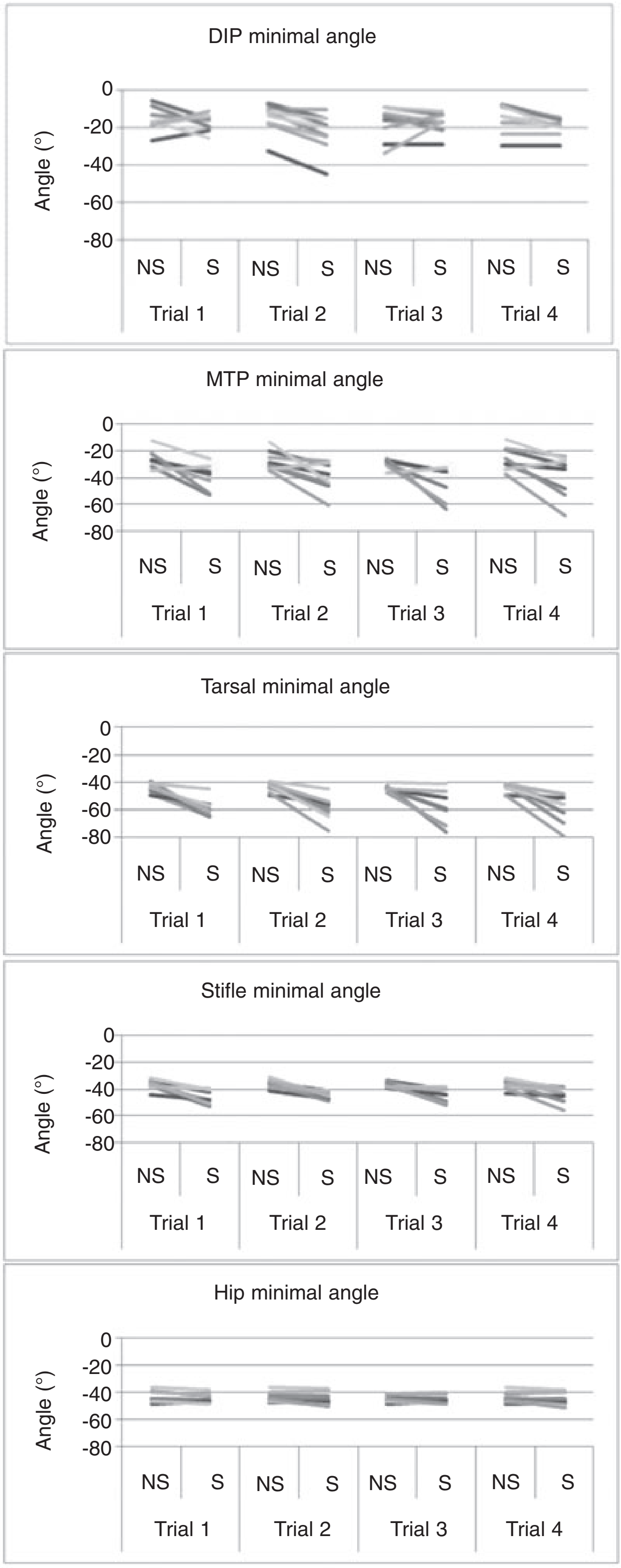

Fig 3: Minimal joint angles during the swing phase for the distal interphalangeal (DIP), metatarsophalangeal (MTP), tarsal, stifle and hip joints (top to bottom) during the swing phase. For each joint, values are shown for 4 consecutive trials (trials 1–4) without stimulators (NS) and with 55 g tactile stimulators attached to both hind pasterns (S). Data points are for 9 horses with lines connecting data points for the same horse across the 2 conditions. The same greyscale colour is used for each horse in every trial.

with stimulators in all horses for all matched trials (Fig 3), although some horses showed a larger response than others, which was responsible for the large variability between horses. There were no changes in the pattern of hip flexion (Fig 4) or in the angles of maximal hip flexion, limb protraction or limb retraction (Table 2). Work done by the hip musculature did not change. The early swing phase increases in joint flexions when wearing stimulators were associated with a large increase in positive work performed by the tarsal flexors, accompanied by larger and more prolonged bursts of negative work in the stifle and MTP extensor musculature that controlled flexion of these joints (Fig 4). In late swing, use of stimulators was associated with a larger burst of positive work in the tarsal extensors accompanied by increased negative work in the stifle flexors to control the rate of extension (Fig 4). At the DIP, net work done changed from positive without stimulators to negative with stimulators (Table 3) as a consequence of needing a larger burst of negative work in late swing to control the angle of the DIP joint at ground contact. In spite of the significant changes in limb kinematics and in work done across the joints of the hindlimb during the swing phase, limb kinematics at ground contact were not changed by the presence of stimulators.

Discussion

The first experimental hypothesis, which stated that the stimulators increased peak swing phase flexion angles, was supported for all joints except the hip, where there was no change in kinematics. The lack of a kinematic response at the hip joint is consistent with the absence of changes in limb protraction or retraction. The second experimental hypothesis, which stated that the use of tactile stimulators would be associated with increased positive work in the tarsal and hip flexors was supported for the tarsus but not for the hip. Therefore, the overall effect of pastern stimulation was to enhance the vertical excursion of the hoof via increased flexion of the joints distal to the hip. From a training standpoint, the type of action associated with use of the stimulators may be desirable in breeds competing in competitions that require animation, such as driving or gaited horses, but not in sport horses in which hip flexion is associated with a desirable increase in hindlimb protraction (Back *et al.* 1994).

Locomotion is driven by central pattern generators (CPGs) in the spinal cord that determine the frequency and coordination of limb movements by controlling flexion and extention of the joints, even in the absence of descending or afferent inputs (Grillner 1981). The resulting gait patterns are adapted in response to proprioceptive afferent feedback from receptors in muscles, ligaments, joint capsules, menisci and skin, as well as input from the special senses of sight, hearing and balance. This feedback contributes at the spinal level to arthrokinetic and muscular reflexes that are involved in neuromotor control of locomotion and dynamic stability (Jerosch and Prymka 1996). The CNS response to sensory feedback can be mediated via a simple reflex arc or through the action of the CPGs that adjust the timing and amplitude of muscular activities in the limbs (Duysens and van de Crommert 1998). As a result, gait is modulated to accommodate changes, such as the presence of tack or equipment on the limbs.

The present study focused on the swing phase because the hip, stifle, tarsal and MTP joints have a larger range of motion and, specifically, show more flexion during swing than stance (Back *et al.* 1995). A force plate was not used because ground reaction force (GRF) data are obtained, on average, for only about 50% of trials (Clayton and Schamhardt 2001). If the horses had performed sufficient trials to yield enough force data for analysis, they may have habituated to the stimulators, thus diluting the kinematic effects (Clayton *et al.* 2008). Therefore, GRF data were not recorded and data analysis was restricted to the swing phase in this study. However, the shortened stance duration when the horses were wearing stimulators suggests the generation of higher GRFs to maintain the necessary impulses, though force data would be required to confirm this. In cats, tactile stimulation on the dorsum of the foot during the stance phase stimulates the extensor musculature to retract the foot more rapidly and shorten stance (Duysens and Pearson 1976). Increased activation of the extensor musculature during stance could explain the reduced stance duration in our study and would fulfil the need for an increase in GRF to maintain the vertical and longitudinal impulses in the face of a shorter stance duration.

The tactile stimulators are thought to act via cutaneous mechanoreceptors. Cutaneous input is not required to establish a locomotor pattern in the hindlimbs but reflex modulation of limb muscle activation in response to cutaneous afferent stimulation has been proposed (Sherrington 1910). These reflexes, which differ from flexor withdrawal reflexes (Burke *et al.* 1991), are important in human subjects for posture (Day and Cole 2002) and gait (Zehr *et al.* 1997, 1998). The muscular response to cutaneous stimulation is phase dependent with the same stimulus triggering flexor or extensor muscles depending on limb position (Gauthier and Rossignol 1978; Rossignol *et al.* 1981), with the muscle of an antagonistic pair that is lengthened being more excitable due to stretching of its muscle spindles. In man, for example, cutaneous stimulation of the plantar surface of the foot triggers reflex modulation of the activity of the tarsal musculature (Fallon *et al.* 2005). With the limb extended in late stance, the tarsal flexors are more receptive to cutaneous stimulation but when the limb is flexed in mid swing the tarsal extensors are more receptive. Therefore, tibial nerve stimulation causes reflex facilitation of the ankle flexors at or just after the stance-swing transition, with the response changing to facilitation of the ankle extensors in late swing (Zehr *et al.* 1997). In the study described here, afferent input from cutaneous mechanoreceptors in the pastern region was via the tibial nerve. The increases in energy generation in the tarsal flexors in early stance and in the tarsal extensors in late stance are entirely consistent with the human responses described above. In the context of rehabilitative medicine, these findings offer the possibility of specific facilitation of the tarsal musculature using a simple tactile stimulator. This type of targeted response may play a crucial role in physiotherapy when muscle activation has been inhibited by pain or immobilisation and normal exercise is insufficient to reactivate the muscle.

In late stance, the tarsal joint extends more than the stifle joint (Clayton *et al.* 2002). The consequent stretching of *peroneus tertius* stores elastic energy that is released in early swing (Wentink 1978) and is responsible for the first elevation in the flight arc of the hindlimb. The second elevation of the flight arc is thought to be driven by muscular contraction of the cranial tibial and long digital extensor muscles that are active in the first half of swing (Jansen *et al.* 1992). We hypothesise that the primary effect of the stimulators is to enhance and precipitate the muscular contribution from the tarsal flexors, thereby increasing net work done across the tarsus. The outcome is a more rapid elevation of the distal limb that over-rides the mid-swing dip in the hoof flight arc, resulting in merging of the 2 flexion peaks. The increase in tarsal flexion is accompanied by increases in stifle and MTP flexion as a consequence of tension in the superficial digital flexor tendon (Wentink 1978). In addition, inertial forces associated with the upward acceleration of the metatarsal segment (Clayton *et al.* 2002) may contribute to increased flexion of the MTP and DIP joints between 20–40% swing.

The exaggerated flexor response and rapid hoof elevation when wearing the tactile stimulators may have an application in rehabilitation, for example in horses that drag their hind toes, provided there is no pathological reason for a restriction of joint motion that could be exacerbated by the flexor response. In some cases, a single use of the stimulators to facilitate muscular activity may be sufficient to restore a normal muscular coordination pattern. In other cases, repeated use of the stimulators may be required to train neuromotor control patterns and/or to strengthen the tarsal musculature. The force of muscular contraction is regulated by adjusting the number of motor units that are activated and/or their firing rate. Thus, the effect of tactile stimulators in recruiting a larger number of muscle fibres can be augmented by progressively increasing locomotor speed during a rehabilitation program. The effect of tactile stimulation of the hind pasterns has been shown to persist over a distance of at least 300 m when trotting in a straight line on a firm level surface (Clayton *et al.* 2008) but the effects of gait transitions, and different surfaces and terrains are not known. Furthermore, it is possible that, even after kinematic habituation to a technique such as the use of tactile stimulators, the afferent input from the stimulators may continue to provide a feedback effect causing a persistent, but less obvious, alteration of neuromotor control. Therefore a rehabilitation

TABLE 3: Mean and (s.d.) of net joint energies in the swing phase of the stride for the hind limbs of 9 horses trotting in hand without stimulators and with tactile stimulators on both hind pasterns. Asterisks indicate pairs of variables that differ significantly with and without stimulators (P<0.05)

	Positive work (J/kg)		Negative work (J/kg)		Total work (J/kg)	
	No stimulators	Stimulators	No stimulators	Stimulators	No stimulators	Stimulators
Hip	0.2412	0.2539	-0.0208	-0.0173	0.2204	0.2366
	(0.0434)	(0.0240)	(0.0151)	(0.0122)	(0.0521)	(0.0319)
Stifle	0.0151*	0.0093*	-0.1676*	-0.2024*	-0.1527*	-0.1933*
	(0.0066)	(0.0054)	(0.0363)	(0.0412)	(0.0376)	(0.0449)
Tarsus	0.0536*	0.0771*	-0.0023	-0.0028	0.0513*	0.0743*
	(0.0094)	(0.0204)	(0.0023)	(0.0024)	(0.0102)	(0.0203)
MTP	0.0015*	0.0007*	-0.0135*	-0.0160*	-0.0120*	-0.0153*
	(0.0015)	(0.0009)	(0.0033)	(0.0050)	(0.0035)	(0.0051)
DIP	0.0014*	0.0008*	-0.0011*	-0.0014*	0.0004*	-0.0007*
	(0.0012)	(0.0006)	(0.0007)	(0.0011)	(0.0008)	(0.0009)

DIP: distal interphalangeal joint; MTP: metatarsophalangeal joint.

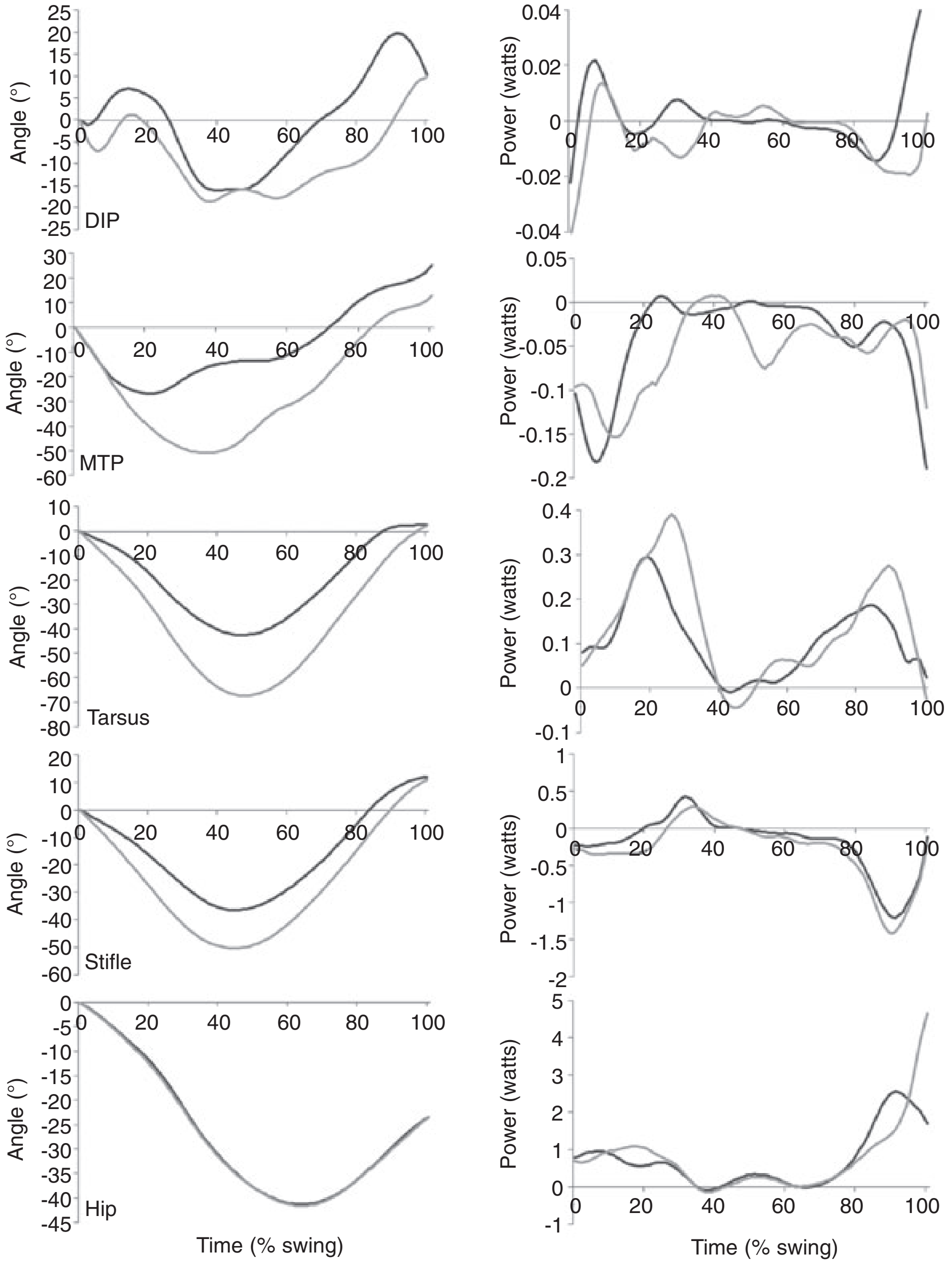

Fig 4: Mean joint angles standardised to angle at contact (left panel) and net joint powers (right panel) for one horse trotting without stimulators (black lines) and with 55 g tactile stimulators attached loosely around the hind pasterns (grey lines). From top to bottom the joints represented are: distal interphalangeal (DIP), metatarsophalangeal (MTP), tarsal, stifle and hip. Positive and negative net joint work are calculated as the areas beneath the positive and negative parts, respectively, of the power curve.

protocol might be based either on alternating periods of exercise with and without stimulators or on continuous use of stimulators during a training session. Future studies will be directed toward developing more specific rehabilitation protocols.

In conclusion, the application of lightweight tactile stimulation devices to the hind pasterns increased the height of the flight arc and changed its shape from biphasic to monophasic. This response is potentially useful for increasing swing phase flexions at the stifle, tarsal, fetlock and coffin joints but not at the hip joint, which determines the range of protraction and retraction. More specifically, tactile stimulation of the hind pastern may be used to recruit and strengthen the tarsal musculature, which drives the increases in swing phase flexions, and to stimulate eccentric action of the stifle musculature. These muscular facilitations may have therapeutic applications in restoring normal kinematic patterns after injury or immobilisation and in strengthening the musculature of the stifle and tarsal joints.

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Manufacturers' addresses

[1] Motion Analysis Corporation, Santa Rosa, California, USA.
[2] SPSS Inc., Chicago, Illinois, USA.

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