

The effect of bilateral palmar digital nerve analgesia on the compressive force experienced by the navicular bone in horses with navicular disease

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Summary

Horses with navicular disease have an increased load on the navicular bone in early stance. This has been suggested to be a response to pain in the heel region. Seven horses with clinical, radiographic and scintigraphic signs of navicular disease underwent forceplate and kinematic analysis before and after desensitisation of the heel region with a bilateral palmar digital nerve block. The compressive force exerted on the navicular bone during stance, and stride kinematics, were determined in each state. After regional analgesia of the palmar digital nerves (PDNB) the compressive force on the navicular bone was lower throughout stance. The mean \pm s.d. peak force at the beginning of stance was 7.05 ± 1.10 N/kg before, and 6.46 ± 1.15 N/kg after PDNB ($P = 0.01$) and at the end of stance the mean peak values were 5.00 ± 2.05 N/kg before, and 4.39 ± 1.65 N/kg after PDNB ($P = 0.05$). We explained this finding as indicating that the horse responds to heel pain (including pain in the navicular region) by contracting the deep digital flexor muscle to unload the heels. This increases the compressive load on the navicular bone, which may cause remodelling and, in some horses, damage to the overlying flexor cartilage, which is then painful and identified as navicular disease. This mechanism identifies navicular disease as a possible end point for a variety of heel related conditions.

Introduction

Navicular disease is a chronic intractable condition of riding horses. Its aetiology is poorly understood but the most popular theories involve either a vascular insufficiency (Colles 1979), a biomechanical susceptibility due to poor foot conformation and farriery (Wright and Douglas 1993), or a combination of the above (Pool *et al.* 1989). Horses with navicular disease demonstrate remodelling (Ostblom *et al.* 1982) and associated changes of the navicular bone (Wright *et al.* 1998) on radiographs and/or bone scans (Trout *et al.* 1991). This suggests that the bone has been subjected to an increased mechanical load (Rubin and Lanyon 1987), presumably as a result of the horse's conformation and/or its movement pattern (Stashak 1987; Wright 1993a). The

classic description of a horse with navicular disease is bilateral caudal foot lameness, and a movement pattern of toe first landing and a shortened cranial phase of the stride which is most apparent on hard ground (Stashak 1987; Turner 1989; Wyn Jones 1988). The gait is assumed to be a strategy to reduce pain in the navicular region, by reducing the loads experienced by the bone or by landing toe first to avoid painful heel concussion.

We have recently confirmed this toe first landing in horses with navicular disease and suggested that it is the result of a contraction of the deep digital flexor (DDF) muscle prior to foot contact and during early and mid-stance (Wilson *et al.* 2001). In that study, the compressive force exerted on the navicular bone by the deep digital flexor tendon (DDFT) in early stance was significantly higher in the diseased than in the control group. We proposed that this was the result of the horse attempting to unload the heels, as a response to pain in the navicular region, by contraction of the DDF muscle. This produced an increased moment at the DIP joint and an increased compressive force on the navicular bone from the DDFT. This increase in navicular bone loading, as a result of pain in the heel region, could then account for progression of the disease and a positive feedback loop as the horse makes further attempts to unload the heels.

If unloading of the heels is a specific response to heel pain, then desensitisation of the heels should reverse the effect. If, however, the force exerted on the navicular bone is not reduced after nerve block this would indicate either that the gait changes are long lasting or that they reflect some other change in limb biomechanics.

Injection of local anaesthetic around the medial and lateral palmar digital nerves, a palmar digital nerve block (PDNB), will block pain sensation and proprioception from the heel region of the foot including the navicular bone and associated structures (Stashak 1987). This will either dramatically improve the lameness of horse with navicular disease or make it switch to the other leg (due to the bilateral nature of the condition). Bilateral PDNB should result in an reduction in lameness and a more normal locomotion pattern, reversing, or at least greatly diminishing, the gait changes associated with pain in the navicular region. This analgesia of the heel region would also be expected to return the loading experienced by the navicular bone to the normal state.

We propose that horses with caudal foot pain alter their gait to unload their heels, but as a result this increases the peak

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compressive load exerted on the navicular bone, and that this effect will be reversed when the caudal region of the foot is desensitised with a PDNB. In this study, we set out to test the hypothesis that horses with navicular disease decrease the load on their navicular bones when the pain from that region is blocked by bilateral analgesia of the palmar digital nerves.

Materials and methods

Horses

Riding horses ($n = 7$, age 10–16 years, weight 425–590 kg), referred to The Royal Veterinary College Equine Referral Hospital for treatment of navicular disease were used in the study. All were riding horses: 1 Warmblood, 2 Thoroughbreds, 2 Thoroughbred crosses, 1 arab and 1 cob. All horses had radiographic (Wright 1993b) and/or scintigraphic (Keegan *et al.* 1996) signs of navicular disease, were mildly lame and in regular light exercise. Diagnosis of navicular disease had been confirmed using either intra-articular analgesia of the DIP joint or analgesia of the navicular bursa (Dyson and Kidd 1993). The horses' lameness scores were 1–3/10ths at trot in a straight line on a hard surface, and 2–5/10ths on left and right circles prior to nerve block. All horses were lame on the inside leg on both circles indicating that their lameness was bilateral. All horses had been through a 3 month course of corrective farriery to improve their foot balance and were assessed by an experienced farrier (C.P.) at the time of data collection as having good foot balance. They were all shod with good heel coverage using standard wide web shoes pulled back at the toe.

Assessment

Horses were assessed immediately before and after administration of a bilateral PDNB. Mepivacaine hydrochloride (Intra-Epicaine)¹ solution, 2 ml of 2%, were injected over the medial and lateral branches of the palmar digital nerves, at the level of the lateral cartilages, of both forelimbs. Desensitisation was confirmed when the horse failed to respond to a sharp point applied to the medial and lateral heels.

Each horse was trotted up in a straight line and lunged on both reins on a hard surface and scored for lameness on a scale of 1–10 by an experienced clinician before and after PDNB. Hemispherical retroreflective markers, 40 mm in diameter, were applied to the hoof wall approximately over the centre of rotation of the DIP joint, over the centre of rotation of the metacarpo-phalangeal (MCP) joint and over the proximal end of the 2nd (right forelimb) or 4th (left forelimb) metacarpal bones. Markers were applied on the lateral side of the left leg and the medial side of the right leg, using hot melt glue and hot glue gun². A further marker was applied to the left thorax of the horse in order to calculate its velocity. A latero-medial radiograph was taken of each forelimb.

Horses were then trotted in hand, from the right hand side, at a speed comfortable for the individual horse, over a forceplate runway within a 25 m long polythene tunnel covered in light proof material. The forceplate (9827BA)³ was set in concrete at the mid length of a 6 mm thick commercial conveyer belt matting runway. A 900 x 600 x 10 mm aluminium plate covered in the same rubber matting was bolted to the top of the forceplate. The forceplate signal was amplified by integral eight channel charge amplifiers, filtered through a low pass filter (6 db/octave from 50 Hz) and logged via a 12 bit AD converter

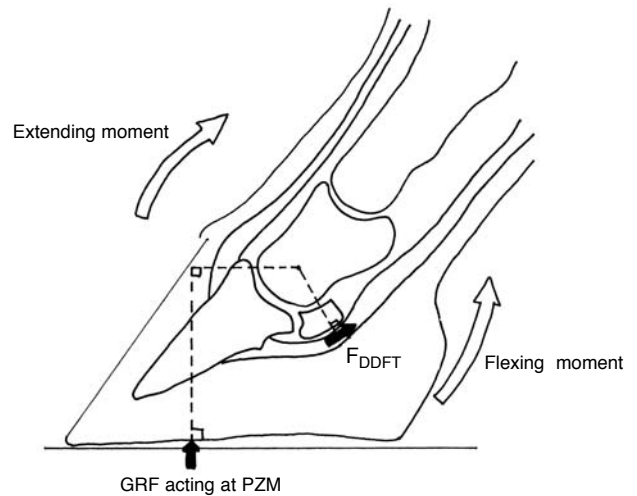


Fig 1: Diagram of the forces and moment arms acting around the distal interphalangeal joint. During the quasi static environment of stance, the ground reaction force (GRF) acts at the point of zero moment (PZM) and the extending moment that it produces is balanced by an equal and opposite flexing moment produced by the force in the deep digital flexor tendon (DDFT) and the moment arm created by the navicular bone.

at 500 samples/s into a personal computer using software written by the authors in LabView⁴. A video motion analysis system (ProReflex)⁵ was used to determine the position of the markers at a frame rate of 240 Hz. A minimum of 6, and up to 9 foot strikes were recorded for each forelimb before and after PDNB. Data were rejected if the horse was not judged to be moving freely at constant velocity or if the foot was placed on the edge of the forceplate.

Data analysis

Kinematics: The following kinematic parameters were calculated: velocity, stride length and frequency, cranial stance time, caudal stance time and total stance duration. As forces within the limb are a function of speed it was necessary to calculate speed of the horse in order to determine whether it was the same during forceplate assessment before and after PDNB. Mid stance was defined as the time when the marker on metacarpal II or IV was directly above the MCP joint marker. The time from foot contact (the point at which the vertical position of the foot marker became constant) to mid stance was defined as the cranial phase duration. The time from mid stance to heel off (when the vertical position of the foot marker began to increase again) was defined as caudal stance duration. The angle of the digit to the ground, defined as the angle that the line joining the foot and MCP joint markers made with the ground, was also calculated during stance.

Kinetics: It is possible, using the above data, to determine the compressive load imposed on the navicular bone during locomotion. During stance the force exerted down a horse's leg acts to extend the distal interphalangeal (DIP) joint. This joint extension is balanced by the pull of the DDFT (Bartel *et al.* 1978). The navicular bone increases the DDFT lever arm at the DIP joint (Fig 1) which results in compression of the navicular bone by the DDFT (Schryver *et al.* 1978; Willemsen *et al.* 1999).

It is possible to determine a theoretical balance point under a horse's foot where, for biomechanical calculations, the entire

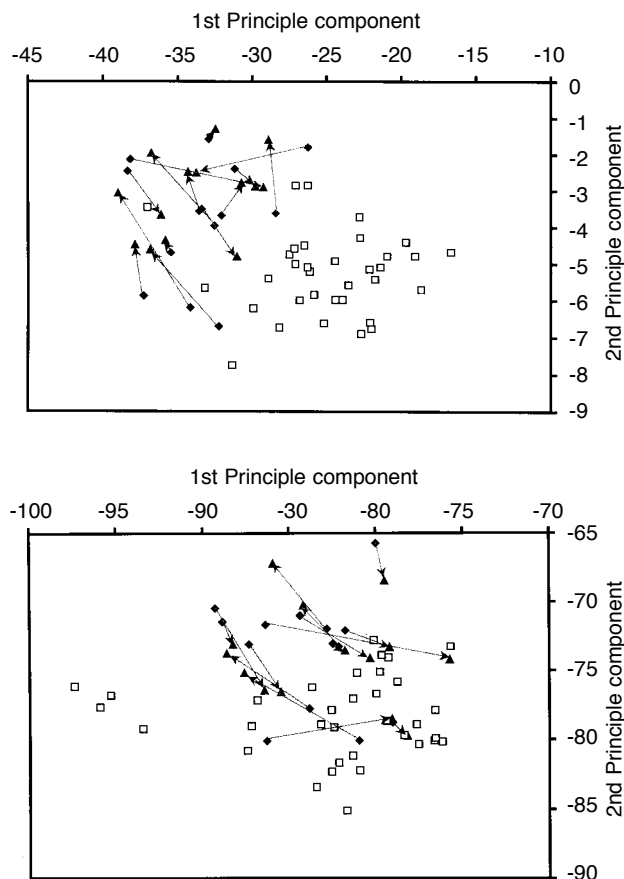


Fig 2: Plot representing the first and second principal components for a principal component analysis of the limb vertical force time curve at beginning (a) and end (b) of the stance phase for the forelimbs of 17 normal horses (\square) previously published by Williams *et al.* 1999 and the 7 horses with navicular disease used here before (\blacksquare) and after (\blacktriangle) bilateral PDNB. Each arrow links the values for a leg before and after nerve block.

weight transferred through the limb can be considered to act. This is the point of force or point of zero moment (PZM) (Nigg and Herzog 1999; Wilson *et al.* 1998). The distance of this from the centre of rotation of the DIP joint multiplied by the limb vertical force gives the torque or moment of the extending force on the DIP joint. The angular acceleration at the DIP joint is small during stance and it can be regarded as a quasi-static situation (Bartel *et al.* 1978; Schryver *et al.* 1978; Willemen *et al.* 1999) and the moments on the DIP joint can therefore be assumed to balance and the moment of the limb force is taken as equal but opposite to DDFT moment. The DDFT moment is the product of the force exerted by the DDFT and its moment arm (the offset of the tendon from the centre of rotation of the DIP joint caused by the navicular bone). The compressive force experienced by the navicular bone is given by the force in the DDFT and the cosine of half the angle between the parts of the DDFT proximal and distal to the navicular bone (Willemen 1997). The force experienced by the navicular bone can therefore be calculated during locomotion with data from a forceplate, motion analysis system and static radiographs (Wilson *et al.* 2001).

Standard formulae were used in a spreadsheet programme (Excel 97)⁶ to calculate the coordinates of the PZM, and a polynomial correction applied to improve the accuracy of the

TABLE 1: Group mean (\pm s.d.) values for speed (m/s), stance time (ms), stride length (m), stride frequency (Hz), cranial stance time (ms) (the time from foot contact to mid stance) and caudal stance time (ms) (the time from mid stance to heel off) before and after bilateral PDNB. Foot contact was defined as the time when the vertical position of the foot became constant; mid stance was when the marker on the proximal end of metacarpal II or IV was directly above the marker on the centre of rotation of the metacarpo-phalangeal joint; heel off was when the vertical foot position began to increase

	Before PDNB	After PDNB
Speed (m/s)	2.92 (0.24)	2.98 (0.19)
Stance time (ms)	329 (29)	321 (13)
Stride length (m)	2.136 (0.113)	2.180 (0.037)
Stride frequency (Hz)	1.36 (0.07)	1.22 (0.41)
Cranial stance time (ms)	175 (43)	159 (28)
Caudal stance time (ms)	153 (28)	161 (21)

determination of the PZM (Bobbert and Schamhardt 1990). The coordinates of each PZM record were expressed relative to the midline cranial extent of the toe using the kinematic data (Wilson *et al.* 2001). The data set was reduced to 100 time points evenly spaced over time by linear interpolation, and right leg mediolateral data inverted (Wilson *et al.* 1998). Limb vertical and cranio caudal force and the angle of the digit to the ground for each run were also interpolated as above.

The PZM, limb vertical force, the angle of the digit with the ground and measurements from the radiographs were used to calculate the compressive force exerted on the navicular bone during stance for each run. Mean curves (\pm s.e.) were produced for each horse forelimb; these were then averaged to produce curves for the group of horses before and after nerve block.

Statistical analysis

Navicular bone force data for each limb before and after PDNB were compared using a student's paired *t* test at 2 identifiable time points during stance: 1) The peak in the force time curve at the beginning of stance identifiable in horses with navicular disease. 2) The peak in the force time curve at the end of stance where the force peaks in normal horses.

The individual limb Fz time curves before and after nerve block were compared to those from a previously published group of seventeen normal horses (Williams *et al.* 1999) using principal component analysis of 4 (beginning) and 4 (end) of stance phase landmarks, identified from inflexions on the Fy curve (Dow *et al.* 1991).

Paired *t* tests were performed on the principal components for the navicular disease population before and after nerve block and an unpaired *t* test used for the comparison of normal and navicular disease horses. The kinematic parameters were compared for the before and after nerve block states using a Student's paired *t* test.

Results

Clinical assessment

After PDNB all horses were sound in a straight line and all but 2 were sound on the lunge on a hard surface. The remaining 2 horses, 1 and 5, remained 1/10th lame on the inside leg while on the lunge.

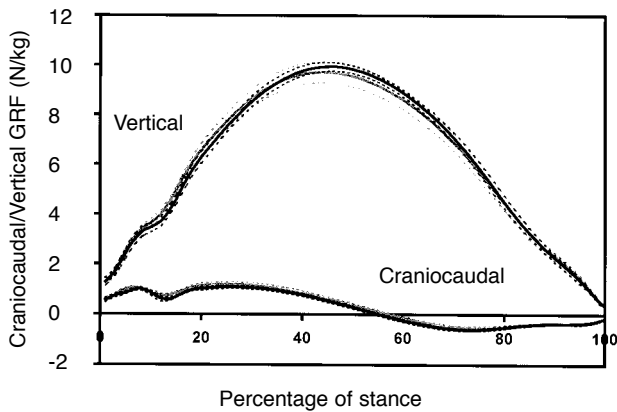


Fig 3: Plot of mean vertical and craniocaudal ground reaction force (GRF) against percentage of stance before (black) and after (grey) bilateral PDNB for a population of horses with navicular disease ($n = 14$). The dotted lines represent \pm one s.e. of the mean.

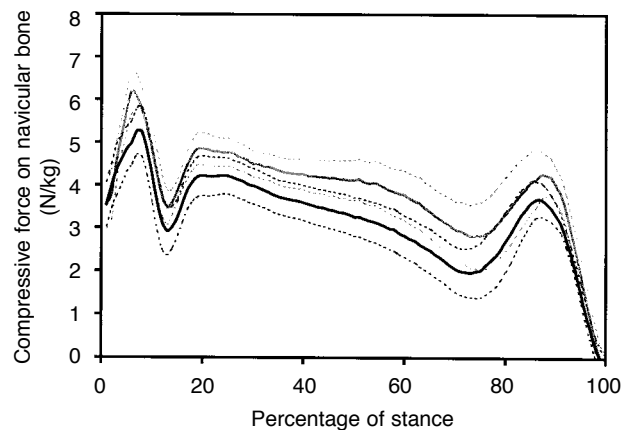


Fig 5 Plot of mean compressive force on the navicular bone for a population of horses with navicular disease before (black) and after (grey) bilateral PDNB ($n = 14$). The dotted lines represent \pm one s.e. of the mean.

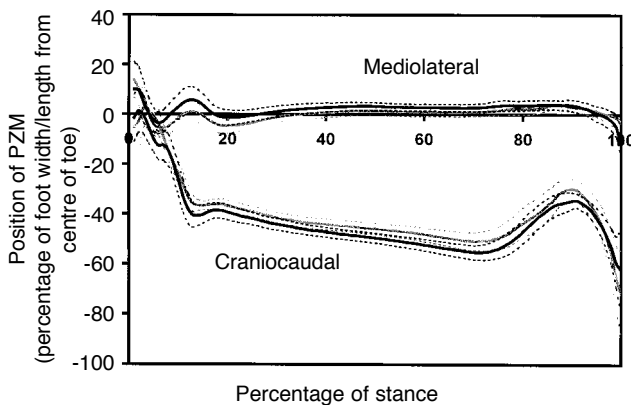


Fig 4: Plot of mean mediolateral and craniocaudal position of point of zero (PZM) moment against percentage of stance before (black) and after (grey) bilateral PDNB for a population of horses with navicular disease ($n = 14$). The dotted lines represent \pm one s.e. of the mean.

Kinematic analysis

There was no significant change in velocity, stance time, stride length or stride frequency after PDNB (Table 1). There was also no significant change in the absolute or relative duration of the cranial and caudal phases of stance.

Kinetic analysis

All horses became more symmetrical in terms of peak Fz after PDNB: the symmetry index (the ratio between values for the 2 forelimbs, ranging between 0 and 1: Williams *et al.* 1999) was 0.89 before PDNB and 0.97 after PDNB, $P = 0.01$

There were significant differences between the Fz time plots of the horses with navicular disease (in the unblocked state) and those for the group of normal horses at both the beginning and end of the stance phase. This comparison, undertaken using a principal component analysis (Williams *et al.* 1999), showed that at the beginning of the stance phase (Fig 2a) the 1st and 2nd principal component scores were both significantly different ($P < 0.001$), and at the end of the stance phase (Fig 2b) the 1st principal component score was not significantly different, but the 2nd was significantly different $P < 0.001$.

Principal component analysis revealed no apparent difference in the Fz time plots for the horses with navicular disease before and after PDNB (Figs 2a and b). Peak Fz did, however, occur slightly later after nerve block (Fig 3) 44.3% vs. 46.2% of stance $P < 0.001$. There was no significant change in the timing or magnitude of Fy (Fig 3). There was also no change in the mediolateral position of the PZM, but there was an apparent caudal displacement of the PZM throughout stance (Fig 4). This, however, was not significant.

The compressive force on the navicular bone during stance before and after PDNB is shown in Figure 5. This force is lower throughout stance after PDNB. Navicular bone force peaked early in the stance phase in 12 out of 14 legs both before and after PDNB. Horse 2 did not show a peak early in stance and was therefore removed from statistical analysis of the beginning of stance force peak. The mean \pm s.e. value for the early stance peak was 7.05 ± 1.10 N/kg at $6.75 \pm 1.66\%$ of stance before PDNB and 6.46 ± 1.15 N/kg at $7.08 \pm 4.33\%$ of stance after PDNB. The values for the peak compressive force on the navicular bone were significantly different $P = 0.01$, but the timing of this peak was not. All horses showed a peak at the end of stance around breakerover. The mean value of this peak was 5.00 ± 2.05 N/kg at $84.36 \pm 5.34\%$ of stance before PDNB and 4.39 ± 1.65 N/kg at $85.79 \pm 4.25\%$ of stance after PDNB. Again, the values for the mean force at this peak were significantly different $P = 0.05$, but the timing of the peak was not.

Discussion

The 7 horses in this study were variable in their history, breed, size and age. This along with the variable nature of the condition termed navicular disease probably created a spread of pathology and responsiveness to PDNB. This is evident in the observation that, while all horses improved after PDNB, 2 of the group were mildly lame on a circle suggesting that other structures within the foot, for instance the DIP joint, were painful. All horses did however fulfil the criteria applied as diagnostic of 'classic' navicular disease (Wright 1993a,b).

The navicular bone force time curve (Fig 5) was much flatter for both states in horses with navicular disease than the typical curve for normal horses presented by Willemen *et al.* (1999) and ourselves (Wilson *et al.* 2001). In sound horses the

bone force is low during early and mid stance, and then rises to a peak around 85% of stance (Schryver *et al.* 1978) which is the result of passive loading via the accessory ligament of the DDFT (Jansen *et al.* 1993). In the diseased horses the force rose to a pronounced force peak around 8% of stance. In 12 out of 14 legs the peak force recorded in early stance was actually greater than that recorded in late stance. The much higher forces on the navicular bone of diseased horses in early and mid stance are the result of higher forces in the DDFT (Wilson *et al.* 2001). These higher forces account for the unloading of the heels and are presumably the result of contraction of the DDF muscle, which could be confirmed by EMG analysis of the muscle during locomotion. The DDF muscle of the horse is highly pennate, with muscle fibres of mean length 17 mm in the humeral head (the largest head) (Hermanson and Cobb 1992), and appears to be capable of exerting the force required (Wilson *et al.* 2001).

Why the bone force curve and the vertical GRF time curve, assessed by principal component analysis, did not return to the shape reported for normal horses after nerve block is interesting. It may be that the DDF muscle fibres have shortened by removal of sarcomeres to accommodate its new shorter range of motion or that the enhanced DDF muscle stimulation was not completely reversed by short term nerve block. It would be interesting to examine some horses by forceplate, before and after palmar digital neurectomy, to determine the chronic response to loss of heel pain and to block (using local anaesthetic) the median nerve, which contains the motor fibres to most of the DDF muscle, in horses with navicular disease to see if this returned the navicular bone force time plot to that reported for normal horses.

The high early navicular bone force peak, was both consistent and repeatable and was the result of these horses landing toe first (PZM = 0–10% of foot length behind the toe, Fig 4). This means that the lever arm on the DIP joint was very high in early stance, hence the navicular bone force was also high. This peak is much less marked in normal horses (Wilson *et al.* 1998; Willemen *et al.* 1999) presumably due to a flatter foot orientation at landing and/or a more compliant limb due to less DDF muscle contraction. The force peak was repeatable between runs and horses and of too low a frequency to have been due to forceplate resonance or the vibrational component of the navicular bone loading alluded to by Rooney (1969). This high force just after foot placement may however explain why horses with navicular region pain appear uncomfortable at foot placement and that this effect is most evident on hard ground.

Despite the improvement in soundness and symmetry after nerve block, there were no changes in the kinematic variables measured. This may mean that inappropriate parameters were measured since much of the clinical impression is based on head movement (Keegan *et al.* 1997) or because the clinical assessments were undertaken on a hard asphalt surface and the motion and forceplate analysis was undertaken on 6 mm thick rubber matting. Finally, measurements were undertaken under bilateral conditions of lameness and analgesia and, therefore, there was much less scope for compensatory weight transfer between contralateral limbs. This finding is however similar to that reported in studies in man where substantial changes in limb weight distribution can be made with minimal change in limb kinematics (Nigg *et al.* 1998).

There was a significant and consistent drop in the force experienced by the navicular bone throughout stance after PDNB. This means that horses with pain in the navicular region

actually increase the compressive load experienced by the navicular bone. This effect may be specific to horses with navicular pain or may represent the response to any heel pain. The increase in force was mainly the result of an, in itself, nonsignificant caudal displacement of the PZM after PDNB. This could have been due to a loss of proprioception with a generalised effect on neuromuscular control of movement. A previous study on the effect of local analgesia of the distal limb on kinetics of sound horses found the only parameter to be altered was the timing of the change from a decelerative to accelerative force in the cranio-caudal GRF (Keg *et al.* 1996). A change in this parameter could not account for the differences in navicular bone loading pattern reported here.

Studies on the change in kinematics in response to local analgesia have been performed on normal horses (Keegan *et al.* 1997; Drevemo *et al.* 1999) and those with navicular disease (Keegan *et al.* 1997). In these papers, different areas of the distal limb were desensitised so it is not possible to compare the results directly, however, both report changes in MCP joint angle range of motion in normal horses. This is closely related to limb vertical force (Wilson *et al.* 2000) which does not change after PDNB in this study. Keegan *et al.* (1997) assessed horses with navicular disease trotting on a treadmill after unilateral (left) and bilateral PDNB. They report a decrease in stance time and cranial stance time for the right limb after the bilateral PDNB was administered compared to the unilateral PDNB. We did not observe such a decrease but our study was performed overground and horses were not observed in a unilaterally blocked state. Therefore, although changes in kinematics due to a loss of proprioception may occur, it is unlikely that they could account for the changes seen in the navicular bone loading that we report.

An alternative mechanism that would account for the data reported here is as follows: a horse with pain in the heel region will attempt to unload the heels by moving its weight (PZM) towards the toe. It does this by increasing the force in the DDFT (via contraction of the DDF muscle) and as a result the compressive force on the navicular bone (Wilson *et al.* 2001). In horses with chronic heel pain this increase in loading rate on the navicular bone may result in bone remodelling and radiographic/scintigraphic changes. After PDNB the heels will no longer be painful so the DDF muscle is relaxed and the PZM moves towards the heels. The navicular bone receives its sensory innervation along with the heel region via the palmar digital nerves (Bowker *et al.* 1995). If the horse reacted to general heel pain rather than localising it to the navicular bone (certainly this level of discrimination of sensation is not possible in the human digit), or if structures other than the navicular bone itself were also painful, this would provide an explanation for the data reported here and perhaps even the development and progression of navicular disease in horses with chronic heel pain. In conclusion, when horses with heel pain due to navicular disease have their heels desensitised by a bilateral PDNB the force on the navicular bone is reduced. This suggests that their response to pain in the navicular region is to unload the heels which results in an increase in the force on the navicular bone.

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Manufacturers' addresses

¹Arnolds Veterinary Supplies, Shrewsbury, UK.

²Bostik Ltd., Leicester, England.

³Kistler Instrumente AG Winterthur, CH-8408 Winterthur, Switzerland.

⁴National Instruments, Newbury, Berkshire, UK.

⁵Qualisys AB, Sävedalen, Sweden.

⁶Microsoft Corporation, Redmond, Washington, USA.

References

- Bartel, D.L., Schryver, H.F., Lowe, J.E. and Parker, R.A. (1978) Locomotion in the horse: a procedure for computing the internal forces in the digit. *Am. J. vet. Res.* **39**, 1721-1727.
- Bobbert, M.F. and Schamhardt, H.C. (1990) Accuracy of determining the point of force application with piezoelectric force plates. *J. Biomech.* **23**, 705-710.
- Bowker, R.M., Linder, K., Sonea, I.M. and Holland, R.E. (1995) Sensory innervation of the navicular bone and bursa in the foal. *Equine vet. J.* **27**, 60-65.
- Colles, C.M. (1979) Ischaemic necrosis of the navicular bone and its treatment. *Vet. Rec.* **104**, 130-137.
- Dow, S.M., Leendertz, J.A., Silver, I.A. and Goodship, A.E. (1991) Identification of subclinical tendon injury from ground reaction force analysis. *Equine vet. J.* **23**, 266-272.
- Drevemo, S., Johnston, C., Roepstorff, L. and Gustås, P. (1999) Nerve block and intra-articular anaesthesia of the forelimb in the sound horse. *Equine vet. J., Suppl.* **30**, 266-269.
- Dyson, S.J. and Kidd, L. (1993) A comparison of responses to analgesia of the navicular bursa and intra-articular analgesia of the distal interphalangeal joint in 59 horses. *Equine vet. J.* **25**, 93-98.
- Hermanson, J.W. and Cobb, M.A. (1992) Four forearm flexor muscles of the horse, *Equus caballus*: anatomy and histochemistry. *J. Morphol.* **212**, 269-280.
- Jansen, M.O., van den Bogert, A.J., Riemersma, D.J. and Schamhardt, H.C. (1993) In vivo tendon forces in the forelimb of ponies at walk, validated by ground reaction force measurements. *Acta Anat (Basel)* **146**, 162-167.
- Keegan, K.G., Wilson, D.A., Lattimer, J.C., Twardock, A.R. and Ellersieck, M.R. (1996) Scintigraphic evaluation of ^{99m}Tc-methylene diphosphonate in the navicular area of horses with lameness isolated to the foot by anesthesia of the palmar digital nerves. *Am. J. vet. Res.* **57**, 415-421.
- Keegan, K.G., Wilson, D.J., Wilson, D.A., Frankeny, R.L., Loch, W.E. and Smith, B. (1997) Effects of anaesthesia of the palmar digital nerves on kinematic gait analysis in horses with and without navicular disease. *Am. J. vet. Res.* **58**, 218-223.
- Keg, P.R., Schamhardt, H.C., van Weeren, P.R. and Barneveld, A. (1996) The effect of diagnostic regional nerve blocks in the forelimb on the locomotion of clinically sound horses. *Vet. Quart.* **18** (Suppl. 2), S106-109.
- Nigg, B.M., Khan, A., Fisher, V. and Stefanyshyn, D. (1998) Effect of shoe insert construction on foot and leg movement. *Med. Sci. Sports Exerc.* **30**, 550-555.
- Nigg, B.M. and Herzog, W. (1999) *Biomechanics of the Musculoskeletal System*. 2nd edn., John Wiley and Sons Ltd. Chichester, England. pp 271-275.
- Ostblom, L., Lund, C. and Melsen, F. (1982) Histological study of navicular bone disease. *Equine vet. J.* **14**, 199-202.
- Pool, R.R., Meagher, D.M. and Stover, S.M. (1989) Pathophysiology of navicular syndrome. *Vet. Clin. N. Am.: Equine Pract.* **5**, 109-129.
- Rooney, J.R. (1969) *The Biomechanics of Lameness in Horses*. Williams and Wilkins, Baltimore. pp 181-186.
- Rubin, C.T. and Lanyon, L.E. (1987) Kappa Delta Award paper: Osteoregulatory nature of mechanical stimuli: function as a determinant of adaptive bone remodelling. *J. Orthop. Res.* **5**, 300-310.
- Schryver, H.F., Bartel, D.L., Langrana, N. and Lowe, J.E. (1978) Locomotion in the horse: kinematics and external and internal forces in the normal equine in the walk and trot. *Am. J. vet. Res.* **39**, 1728-1733.
- Stashak, T. (1987) *Adam's Lameness in Horses*, 4th edn., Lea and Febiger, Philadelphia. pp 499-514.
- Trout, D., Hornhof, W. and O'Brien, T. (1991) Soft tissue and bone phase scintigraphy for diagnosis of navicular disease in horses. *J. Am. vet. med. Ass.* **198**, 73-77.
- Turner, T.A. (1989) Diagnosis and treatment of navicular syndrome in horses. *Vet. Clin. N. Am.: Equine Pract.* **5**, 131-144.
- Willemsen, M.A. (1997) *Horseshoeing, a Biomechanical Analysis*. PhD Thesis, University of Utrecht.
- Willemsen, M.A., Savelberg, H.H. and Barneveld, A. (1999) The effect of orthopaedic shoeing on the force exerted by the deep digital flexor tendon on the navicular bone in horses. *Equine vet. J.* **31**, 25-30.
- Williams, G.E., Silverman, B.W., Wilson, A.M. and Goodship, A.E. (1999) Disease-specific changes in equine ground reaction force data documented by use of principal component analysis. *Am. J. vet. Res.* **60**, 549-555.
- Wilson, A.M., van den Bogert, A.J. and McGuigan, M.P. (2000) Optimization of the muscle-tendon unit for economical locomotion in cursorial animals. In: *Skeletal Muscle Mechanics: From Mechanisms to Function*. Ed: W. Herzog Wiley and Sons, Chichester, England.
- Wilson, A.M., Seelig, T.J., Shield, R.A. and Silverman, B.W. (1998) The effect of hoof imbalance on point of force application in the horse. *Equine vet. J.* **30**, 540-545.
- Wilson, A.M., McGuigan, M.P., Fouracre, L. and McMahon, L. (2001) The force and contact stress on the navicular bone during trot locomotion in sound horses and horses with navicular disease. *Equine vet. J.* **33**, 159-165.
- Wright, I.M. (1993a) A study of 118 cases of navicular disease: clinical features. *Equine vet. J.* **25**, 488-492.
- Wright, I.M. (1993b) A study of 118 cases of navicular disease: radiological features. *Equine vet. J.* **25**, 493-500.
- Wright, I.M. and Douglas, J. (1993) Biomechanical considerations in the treatment of navicular disease. *Vet. Rec.* **133**, 109-114.
- Wright, I.M., Kidd, L. and Thorp, B.H. (1998) Gross, histological and histomorphometric features of the navicular bone and related structures in the horse. *Equine vet. J.* **30**, 220-234.
- Wyn-Jones, G. (1988) *Equine Lameness*. Blackwell Scientific Publications, Oxford, England. pp 53-64.