Tutorial Article

Current concepts of navicular disease

S. Dyson*, R. Murray, T. Blunden† and M. Schramme‡

Centre for Equine Studies and †Centre for Preventive Medicine, Animal Health Trust, Lanwades Park, Kentford, Newmarket, Suffolk CB8 7UU, UK.

Keywords: horse; navicular disease; lameness.

What is navicular disease?

Navicular disease is a chronic forelimb lameness associated with pain arising from the distal sesamoid or navicular bone. It is well recognised that in association with advanced navicular disease, fibrillation of the dorsal aspect of the deep digital flexor tendon (DDFT), with or without adhesion formation between the tendon and the navicular bone, are common features (Figs 1 and 2). Recent clinical studies using magnetic resonance imaging (MRI) (Dyson et al. 2005) and post mortem studies (Blunden et al. 2006a,b) have demonstrated that there may also be abnormalities of closely related structures, including the collateral sesamoidean ligaments (CSLs), distal sesamoidean impar ligament (DSIL) and navicular bursa. For the purposes of this article this complex of degenerative changes will be referred to as navicular disease. Primary injury of the DDFT is considered to be a separate condition (Dyson et al. 2003), which may have a different aetiopathogenesis.

Although historically considered to be a single disease, given the variety of clinical presentations it is likely that there are a number of different clinical conditions, of different aetiologies, that give rise to pain in the navicular apparatus. It is difficult to conceive of a single disease which can result in an insidious onset, slowly progressive bilateral forelimb lameness, or an acute onset, relatively severe unilateral forelimb lameness, each with a variety of different radiological manifestations and with some horses never developing radiological changes. It is curious that sometimes clinical signs become apparent in young horses just commencing work, whereas more typically lameness is seen in mature riding horses. It is also seen in horses with vastly different distal limb conformation. It is a common condition in Quarter Horses, which tend to have narrow, upright, boxy feet, small relative to their body size, as well as in European Warmblood horses, many of which have relatively tall narrow feet. It is also common in Thoroughbred horses, which frequently have

*Author to whom correspondence should be addressed. ‡Present address: Department of Clinical Sciences, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606, USA.
rather flat feet with low collapsed heels, often associated with dorsopalmar foot imbalance (Fig 3). Evidence has recently been presented suggesting that there is a heritable tendency towards the development of navicular disease in Dutch and Hanoverian Warmblood horses (Dik and van den Broek 1995; Dik et al. 2001a; Stock et al. 2004).

The factors causing pain in the navicular apparatus, and therefore lameness, are poorly understood. Experience with both radiography and MRI suggests that lesions in both the navicular bone and closely related structures are likely to predate the onset of lameness in some horses (S. Dyson et al., unpublished data). In some horses, a trigger factor apparently promoting pain and lameness has been a period of enforced rest for an unrelated cause.

There have been no good epidemiological studies investigating risk factors for the development of navicular disease. Information is therefore largely anecdotal. The frequency of occurrence of navicular disease appears to vary between breeds. Quarter Horses (Ackerman et al. 1997), Warmblood horses (Mey et al. 1967) and Thoroughbred-cross horses (Colles 1987) have a relatively high incidence, whereas the occurrence and/or recognition in some breeds such as the Finnhorse, Arab (Turner 1993) and Friesian is relatively low.

Biomechanical considerations

The navicular apparatus comprises the navicular bone, CSLs, DSIL, navicular bursa, DDFT and distal digital annular ligament. The navicular bone, which articulates with the middle and distal phalanges, provides a constant angle of insertion and maintains the mechanical advantage of the DDFT, which exerts major compressive forces on the distal one third of the bone. Contact studies between the phalanges in isolated limbs have demonstrated that the greatest forces are applied in the propulsion phase of the stride. This occurs during extension of the distal interphalangeal (DIP) joint, with increased pressure of the DDFT on the palmar aspect of the navicular bone, increased contact between the navicular bone and the middle phalanx and increased tension in the CSLs (Denoix 1999a; Bowker et al. 2001; Wilson et al. 2001). Tension in the DDFT and distal digital annular ligament promotes stability of the DIP joint. Forces may be altered by foot conformation; in a horse with ‘weak heels’ there is greater extension of the DIP joint than in a horse with ‘strong’ heels, which results in increased pressure concentrated on the distal aspect of the navicular bone (Denoix 1999b).

Compressive forces and stress on the navicular bone have previously been compared in clinically sound horses and horses with navicular disease (Wilson et al. 2001). Although the mean peak force and stress were similar, the force and stress in horses with navicular disease were approximately double early in the stance phase of the stride. This early peak stress resulted in a much higher loading rate of the navicular bone in the navicular disease group. The difference in loading patterns was associated with an increased force in the DDFT in the early and mid-stance phases, probably due to increased contraction of the DDF muscle. This contraction of the DDF muscle may result in toe-first ground contact, seen in some horses with navicular disease. It is suggested that pain associated with the navicular bone may result in positive feedback, by increasing the force in the DDFT to avoid heel-
first landing, and hence paradoxically increasing the compressive force on the navicular bone. This hypothesis is supported by reduction in peak forces on the navicular bone throughout the stance phase in horses with navicular disease after perineural analgesia of the palmar digital nerves (McGuigan and Wilson 2001).

Although low, collapsed heel conformation has anecdotally been associated with navicular disease, a recent study (Eliashar et al. 2004) in Irish draught-cross-type horses showed no correlation between the peak force exerted on the navicular bone by the DDFT and the conformity of the hoof capsule, contrary to the earlier results of Denoix (1999b). However, a 1° decrease in angle of the solar border of the distal phalanx resulted in a 4-fold increase in peak force on the navicular bone (Eliashar et al. 2004). There was no correlation between the angle of the solar border of the distal phalanx and degree of heel collapse.

The shape of the navicular bone may be determined at birth and this may influence the biomechanical forces subsequently applied to the bone, and hence influence the risk of development of navicular disease (Dik et al. 2001a,b). Finnhorses and Friesian horses tend to have a straight or convex contour of the proximal articular border of the navicular bone and rarely develop navicular disease. There is a much higher incidence of navicular disease in the Dutch Warmblood breed, and horses in which the proximal articular margin is concave or undulating appear to be at highest risk of development of the disease (Dik and van den Broek 1995; Dik et al. 2001a).

**Histopathological studies**

Navicular disease has not been reproduced experimentally; therefore, all proposed aetiologies remain speculative. Earlier theories suggesting a vascular aetiology with arteriosclerosis (Rijkenhuizen et al. 1989), or thrombosis, resulting in ischaemia within the navicular bone (Colles and Hickman 1977), have largely been rejected due to failure to identify ischaemic bone or thrombosis, failure to reproduce clinical signs or pathological changes by occluding blood supply to the bone and expanding evidence demonstrating increased bone modelling (Ostblom et al. 1982; MacGregor 1984; Pool et al. 1989; Wright et al. 1998). Post mortem studies to date have focused principally on long-term, chronic cases, generally with advanced radiographic abnormalities, reflecting the end stage of a disease complex. These studies identified striking similarities between the pathological features of navicular disease and osteoarthritis in both people and horses (Pool et al. 1989; Wright et al. 1998).

Studies of ageing changes in the navicular bone of normal immature and mature horses suggested that there is a degenerative ageing process similar to that seen in joints (Wright et al. 1998). However, a more recent study investigating not only the navicular bone, but also the DDFT, CSLs, DSIL and navicular bursa demonstrated no age-related differences between mature horses aged 4–15 years with no history of foot-related lameness (Blunden et al. 2006a,b). This suggests that there may be an individual susceptibility to degenerative change. Nonphysiological biomechanical factors may promote this susceptibility to degenerative change (Pool et al. 1989; Wright and Douglas 1993; Wilson et al. 2001).

The explanation for pain and lameness in horses with no detectable radiological change has been poorly investigated by post mortem studies. However, recent clinical experience with MRI has indicated that many horses with evidence of increased modelling of the navicular bone, based on increased radiopharmaceutical uptake (IRU) detected using nuclear scintigraphy, do have pathological abnormalities of the navicular bone detectable using MRI, with or without concurrent changes in the DDFT, CSLs, DSIL and navicular bursa (Dyson et al. 2005; S. Dyson and R. Murray, unpublished data).

Degenerative changes in the fibrocartilage on the palmar aspect of the navicular bone occur principally in the distal half of the bone, especially centred around the sagittal ridge in both sound and lame horses (Blunden et al. 2006a). In horses with navicular disease there is a greater degree of fibrocartilage damage, which may extend into the subchondral bone (Figs 1 and 4). Partial thickness loss of fibrocartilage in this location was one of the most common lesions significantly associated with navicular disease in one study (Wright et al. 1998). It is likely to represent some of the earliest pathology of one form of this disease, but remains difficult to identify in vivo, even with the use of MRI (S. Dyson et al., unpublished data). Degenerative change of the spongiosa is generally seen only dorsal to extensive fibrocartilage damage. Physiological forces result in adaptive remodelling of the subchondral bone in immature horses, with cortical thickening (Wright et al. 1998). Nonphysiological forces may result in focal fibrocartilage and/or flexor cortex damage, with adjacent subchondral sclerosis dorsal to it, associated with thickening of trabeculae and focal areas of lysis. There may also be oedema, congestion and fibrosis of the marrow stroma within the medullary bone, which may result in a cyst-like lesion.

Concurrently, there may be fibrillation of the dorsal surface of the DDF, which may predispose to adhesion formation between the DDF and regions of partially or fully eroded fibrocartilage on the palmar aspect of the navicular bone. Whether lesions in the DDF are primary or secondary to pre-existing damage of the fibrocartilage currently remains open to debate. However, recent post mortem evidence suggests that there may be nonage-related degenerative vascular and matrix changes in the dorsal aspect of the DDF in both lame and clinically normal horses (i.e. similar frequency of occurrence in young and old horses) (Blunden et al. 2006b) (Fig 5). Although other authors have suggested that vascular occlusion and matrix changes in the DDF may be age-related (Wright et al. 1998), the results of our study showed that the severity of these changes was greater in horses with palmar foot pain than in control horses. Minor fibrillation of the dorsal aspect of the DDF was seen in both lame and control horses, whereas deep sagittal splits were seen only in lame horses. Complete occlusion of blood vessels, replacement of normal tendon architecture by focal fibroplasia and areas of fibrocartilaginous metaplasia were common in the lame horses. As these changes are predominantly seen in the intratendinous septa, there is a
strong possibility that they predispose to the development of sagittal splits in the dorsal surface of the tendon along these septal planes. Sharp edges of splits in the DDFT (Fig 6) extending from the dorsal surface may cause ulceration of the fibrocartilage of the navicular bone and thus predispose to lesions extending into the medulla.

Fig 5a: Section of a deep digital flexor tendon with partial occlusion of vessels (black arrows) in a pale septum (arrowheads). There is fibrocartilaginous metaplasia within the septum. Note the chondrones (white arrows). White areas between the tendon fascicles are processing artefacts. H&E; magnification x400.

Fig 5b: Section of a deep digital flexor tendon with early fibrocartilaginous metaplasia (black arrows). Several blood vessels are occluded (arrowhead). White areas between the adjacent tendon fascicles are processing artefacts. H&E; magnification x200.

There is an association between changes of the flexor aspect and distal and proximal borders of the navicular bone (Blunden et al. 2006a). Similar types of change occur at the proximal and distal aspects, but tend to be more extensive distally. Enlarged synovial invaginations are the result of recruitment and activation of osteoclasts following the course of the nutrient vessels into the spongiosa (Pool et al. 1989). This may be associated with local medullary osteonecrosis, and the presence of foci of fibrocartilaginous metaplasia and/or entheseous new bone close to the interface between the DSIL and the navicular bone.

Ageing changes were described in the articular cartilage of the navicular bone and the opposing face of the distal phalanx (Bowker 2003). There was loss of proteoglycan and tidemark advancement, thought to reflect excessive shear stress in the zone between the calcified and noncalcified articular cartilage. A greater number of tidemarks were seen in horses with clinical signs of navicular disease than normal horses of similar age. However, a more recent study failed to identify significant age-related changes, and low-grade degenerative changes in the articular cartilage were common in both control horses and those with navicular disease (Blunden et al. 2006a).

Observations from nuclear scintigraphy and MRI

Previous studies using tetracycline labelling of bone (MacGregor 1984), histomorphometry (Ostblom et al. 1982) and scintigraphy (Keegan et al. 1996) have indicated that there is evidence of increased bone turnover in association with navicular disease, even in the absence of radiological abnormalities of the bone. IRU predominantly reflects
increased osteoblastic activity (Dyson and Weekes 2003),
but is not synonymous with either pain or lameness (Dyson
2002). IRU may reflect a functional adaptation to foot
conformation and the biomechanical forces on the navicular
bone. Comparison between scintigraphy and MRI has
demonstrated that many horses with focal moderate or
intense IRU have abnormalities of the navicular bone
detectable using MRI (S. Dyson, unpublished data).
However, scintigraphy can also produce false-negative
results, indicating that pathological abnormalities of the
navicular bone are not always associated with increased
osteoblastic activity.

A comparison of MRI findings in control horses with no
history of foot-related pain and horses with chronic palmar
foot pain showed significant alterations of the navicular
apparatus in the lame horses (Murray et al. 2006a). A
comparative MRI and post mortem study showed good
correlation between the lesions identified using MRI and
histopathological findings (Murray et al. 2006b). Clinical
experience with MRI in horses with foot pain provides support
for the progression of lesions as outlined above and has
demonstrated some earlier lesions than those investigated
post mortem (S. Dyson and R. Murray, unpublished data)
(Figs 7–10, illustrating lesions of decreasing severity).

However, a group of horses has also been identified with
no detectable abnormalities of the flexor fibrocartilage or
cortex, but with diffuse abnormalities of the medulla
characterised by increased signal intensity in fat-suppressed
images (Fig 11). Post mortem examination of one such horse
revealed evidence of early fat necrosis with a moth-eaten

**Fig 7a: Parasagittal SPGR image of a navicular bone.** There is
a full-thickness defect in the palmar cortex of the bone
(arrow). There is a diffuse area of reduced signal intensity
within the spongiosa of the bone. On a palmaro45°proximo-
palmarodistal oblique radiographic view there was a focal area
of subtle reduced opacity of the palmar cortex coincident with
the defect in the MR image. No other radiological
abnormality was detected. There was focal intense increased
radiopharmaceutical uptake in the navicular bone.

**Fig 7b: Transverse SPGR image of the foot in Figure 7a.** There is
a full-thickness defect of the palmar cortex of the navicular
bone medial to the sagittal ridge (white arrow); there is
decreased signal intensity in the medulla in the medial half
of the bone. There is a sagittal plane split in the deep digital flexor
tendon (black arrow).

**Fig 7c: Parasagittal T2* GRE image of the navicular bone in
Figure 7a.** There is disruption of the flexor cortex (arrow) and
focal increased signal intensity extending into the subchondral
bone. Dorsally there is a diffuse area of intermediate signal in
the medulla. There is endosteal irregularity of the flexor cortex
proximally (arrowhead).

**Fig 7d: Sagittal STIR image of the navicular bone in Figure 7a.**
There is focal disruption of the palmar cortex of the navicular
bone with a focal area of hyperintense signal extending into
the subchondral bone (arrow). There is a mild, diffuse increase
in signal in the medulla.
appearance of the trabeculae (i.e. disruption of the normally smooth borders of the trabeculae), with necrosis of bone edges. This may have a different aetiopathogenesis.

Hyperintense signal in the medulla of the navicular bone has been ascribed to the presence of oedema in the marrow spaces (Schneider et al. 2003), but this was not validated post mortem. Further research is required to determine the true causes of this phenomenon. In our study, mild or moderate focal or generalised increased signal intensity in fat-suppressed images was associated with trabecular thinning and widened inter trabecular spaces (Murray et al. 2006b). High signal intensity in fat-suppressed images associated with irregular decreased signal intensity in T1- and T2-weighted images was associated with generalised osteonecrosis and fibrosis, with irregular trabeculae, adjacent adipose tissue oedema and prominent capillary infiltration. A recent post mortem study of feet with advanced radiological abnormalities of the navicular bone demonstrated that increased signal intensity in fat-suppressed images correlated with areas of degenerate adipose tissues, with haemorrhage or replacement by fibrocollagenous material, or fluid-filled cystic spaces (Busoni et al. 2005).

In some horses, fluid-filled osseous cyst-like lesions have been seen in the distal aspect of the bone, apparently separate from synovial invaginations, and not associated with any detectable abnormality of the flexor aspect of the bone (Fig 12). Such lesions have not yet been characterised histologically and their aetiology remains speculative, although may be associated with lesions of the DSIL.
Occasionally, horses have been identified with new bone on the palmar aspect of the navicular bone, centred on the sagittal ridge. The cause of this is currently unknown.

**Enthesous changes**

The presence of entheseous new bone on the proximal border of the navicular bone, reflecting previous insertional desmopathy of the CSL, is well documented radiographically (Verschooten et al. 1989; Wright 1993) and at post mortem examination (Pool et al. 1989; Wright et al. 1998) in both clinically normal horses and those with navicular disease. Its clinical significance remains uncertain, although more extensive new bone in this location tends to be associated with other signs of navicular disease (Verschooten et al. 1989; Wright et al. 1998). Recent experience with MRI has confirmed this (Dyson et al. 2005) (Fig 13). Rarely, an avulsion fracture is identified at the insertion of the CSL into the navicular bone (S. Dyson et al., unpublished data). Mineralised and osseous fragments in the DSIL have also been recognised in both normal horses and those with navicular disease, and their clinical significance remains difficult to determine. Fragments were unusual in sound horses undergoing prepurchase radiographic examination (Kaser Hotz and Ueltschi 1992), although their true incidence may be underestimated by radiographic examination compared with MRI or computed tomography. In 2 post mortem studies, fragments associated with a defect in the distal margin of the navicular bone were more common in horses with navicular disease than in age-matched controls (Wright et al. 1998; Blunden et al. 2006a). This has also been our clinical experience (Fig 14).

Fibrocartilaginous metaplasia in the body of the DSIL was more extensive in horses with navicular disease compared with age-matched control horses (Blunden et al. 2006a). However, no significant differences between groups were seen in the CSLs.

Ageing changes have been seen in the region of insertion of the DSIL and the DDFT, with a change in fibroblast shape and an increase in proteoglycans (Bowker et al. 2001). The functional significance of this is not yet known. Evidence of inflammation has recently been recognised histologically at the intersection of the DSIL and DDFT in horses with clinical signs of navicular syndrome (van Wulfen and Bowker 1997). Bowker (2003) demonstrated changes reflecting ‘abnormal stress’ at the insertion of the DSIL and DDFT in horses with poor foot conformation. This region is rich in sensory nerve endings, with many arteriovenous complexes which are damaged in horses with navicular disease (van Wulfen 1999).
result of abnormal stresses at the attachments of the CSL and DSIL on the navicular bone, and this may reflect a different mechanism of navicular disease development.

Although endosteal irregularity at the insertion of the DSIL on the distal phalanx may be seen in both horses with and without foot pain (Murray et al. 2006a,b), in some lame horses there is evidence of insertional desmopathy, characterised by axial cortical disruption and/or increased signal intensity in the bone at this site in fat-suppressed images, reflecting bone oedema or necrosis (S. Dyson and R. Murray, unpublished data).

The navicular bursa

The incidence and aetiology of primary bursitis of the navicular bursa is not known, nor is its relationship to the development of navicular disease. Villous hypertrophy, hyperplasia of synovial lining cells and venous congestion have been described in association with navicular disease, whereas the synovial membrane appeared uniform in 6 normal horses of undetermined age (Svalastoga and Nielsen 1983). However, in another study comparing immature horses, those with navicular disease and age-matched controls, 3 of 25 age-matched controls had evidence of asymptomatic chronic synovitis. In both the navicular disease group and the age-matched controls, mild hyperplasia and hypertrophy was seen compared with immature horses up to 3 years of age (Wright et al. 1998). In a more recent study, there was no evidence of acute inflammation within the navicular bursa in horses with palmar foot pain or age-matched control horses (Blunden et al. 2006a); however, lame horses had marked chronic synovial proliferation compared with control horses (Blunden et al. 2006a; Murray et al. 2006a). There was a positive association between abnormalities of the bursa and lesions of either the dorsal aspect of the DDFT or the flexor aspect of the navicular bone. Clinical experience with MRI has indicated that abnormal distension of the bursa is a frequent finding in lame horses, but is rarely seen in isolation (Dyson et al. 2005) (Fig 16).
Associations between injuries

It is clear from our recent post mortem study, and from clinical experience using MRI, that frequently several structures are affected concurrently. It is common to see various combinations of abnormalities of the navicular bone, DDFT, DSIL, CSL and collateral ligaments (CL) of the DIP joint. Clinical MR examination of 263 horses with forelimb foot pain revealed 6 with abnormalities of the navicular bone alone; 29 with concurrent DDFT and navicular bone abnormalities; 60 with various combinations of abnormalities of the navicular bone, DSIL, DDFT or CSL; 46 with CL injury of the DIP joint in combination with lesions of the DDFT, CSL, DSIL or navicular bone; and 25 horses with abnormalities of 5 or more structures (S. Dyson and R. Murray, unpublished data). The sequence of injury occurrence remains speculative. It is possible that degenerative changes in several structures may predispose to concurrent injury. The navicular bone, CSL and DSIL act as a unit, so presumably undergo similar biomechanical stresses. Alternatively, injury to one structure may cause low-grade instability, predisposing to injury of closely related structures.

What causes pain?

Pain associated with navicular disease may be due to venous congestion of the navicular bone. Dilated venules and sinusoids entrapped in fibrous marrow have only been identified in horses with navicular disease (Pool et al. 1989). Raised intraosseous pressure has been measured in horses with navicular disease (Svalastoga and Smith 1983; Pleasant et al. 1993). Distension of the navicular bursa may cause pain. The contribution of other causes or sources of pain remains open to speculation, although many sensory nerve endings have also been identified in the CSLs and DSIL (Bowker et al. 1993, 1997) and, given the high frequency of occurrence of concurrent abnormalities in these structures, it is likely that these nerve endings may be important in pain mediation.
Fig 12a: Parasagittal SPGR image of a navicular bone. There is a mineralised band traversing from the dorsal to palmar cortices (arrows), distal to which is a diffuse area of intermediate signal intensity. There was a single axial discrete radiolucent zone proximal to the distal border of the bone and mild focal increased radiopharmaceutical uptake in the bone.

Fig 12b: Parasagittal T2* GRE image of the navicular bone in Figure 12a. There is a similar mineralised band traversing from the dorsal to palmar cortices of the bone (arrows), distal to which is an area of hypointense signal, consistent with proteinaceous fluid.

Fig 12c: Parasagittal STIR image of the navicular bone in Figure 12a. There is a band of mineralisation from the dorsal to palmar cortices (arrows), distal to which is a diffuse area of hyperintense signal.

Fig 13a: Sagittal T2* GRE image of a navicular bone. There is an enthesophyte (arrow) in the collateral sesamoidean ligament. There is slight endosteal irregularity of the dorsal and distal cortices. There was slight irregularity of the proximal and distal borders of the bone seen radiographically. Uptake of radiopharmaceutical was normal.

Fig 13b: Dorsal SPGR image of the navicular bone in Figure 13a. There is marked irregularity of the proximal and distal cortices axially. Note also the focal area of intermediate signal proximal to the distal border of the bone axially (arrow).

Fig 13c: Sagittal STIR image of the navicular bone in Figure 13a. There is focal hyperintense signal immediately distal and dorsal to the insertion of the collateral sesamoidean ligament (black arrow). There is focal hyperintense signal proximal to and extending into the distal sesamoidean impar ligament (white arrow). There is a focal area of fluid accumulation palmar to the bone midway between the proximal and distal borders of the bone (open arrow), reflecting loss of fibrocartilage.
The future

It is clear that degrees of adaptive and reactive change occur in the navicular apparatus of all horses. We need to understand better both the factors that stimulate their progression and what causes pain. Identification of genetic and biomechanical risk factors would be useful. Study of early cases of navicular disease should help to establish better the interrelationship between abnormalities of the DDFT, navicular bone, CSL and DSIL. We need to determine what factors lead to vascular and matrix changes in the DDFT. Further research into the sensory nerve supply to the navicular apparatus may help in understanding what causes pain and therefore lameness, and how it may be treated.

Acknowledgements

We thank The Home of Rest for Horses for financial support.

References


of nerves potentially desensitised by injections into the distal interphalangeal joint or the navicular bursa of horses. J. Am. vet. med. Ass. 203, 1708-1714.


