Navicular Disease

by
James Rooney, D.V.M.

Despite thousands, or perhaps millions, of words, navicular disease remains a subject of confusion and error. I have written as clearly as I can on the subject, defining what the condition actually is and how it may develop. My opinions are based on years of observation, primarily in the postmortem room, together with some in vitro studies. While it might be convenient to have all that repeated here, I can only hope that you will check the references which are readily available. Here I want to add information from a more recent in vitro study, make some additional observations, and try to clarify some points.

Based on earlier in vitro work increased friction because of increased force between the face of the deep flexor tendon and the palmar surface of the navicular fibrocartilage was suggested as the immediate cause of damage to both tendon and fibrocartilage. Toe-first impact was simulated in a testing machine and damage was found histologically in the areas indicated in Fig. 1. That will be considered further below. The normal force (the force component perpendicular to the surfaces) was caused by either toe-first impact of the foot with the ground and/or rocking back and forth of the hoof on the ground (giving rapidly repeated toe-first configurations). The general case for both of these situations is vibration. In the appendix to this paper I try to show how this works in somewhat more detail for those willing to work through the material.

Routine examination of the navicular at postmortem in thousands of horses made it clear – at least to me – that the earliest naked eye evidence of navicular cartilage damage is a yellowish brownish, often translucent, discoloration with superficial fraying of the cartilage in the two areas indicated schematically in Fig. 1. These are proximal and distal ridges on the surface. The distal ridge is more pronounced, that is, has greater curvature and, so, there is greater normal force exerted upon it. The lesions appear on the distal ridge first and more extensively than on the less curved proximal ridge. This is shown in the schematic Side View and in a photograph (Fig. 3) in Ref. 3.
One recognizes, of course, that the damage occurs simultaneously or nearly so on both the navicular fibrocartilage and the fibrocartilage in that part of the deep flexor tendon articulating with the navicular cartilage fibrocartilage.

The yellow discoloration is, in fact, the earliest naked eye evidence of damage to equine articular cartilage wherever it be and whether it be hyaline or fibrocartilage. The yellow color is not caused by a pigment but rather it is the result of structural change in the cartilage. The precise nature of that change is not known and would be a fine subject for study. What is immediately evident histologically is loss of chondrocytes in the yellow area together with fraying of the superficial layer of fibrocartilage and increased eosinophilia of the cartilage matrix.

Given the hypothesis that vibration is the proximate cause of navicular cartilage and deep flexor tendon damage, the next hypothesis is that such vibration causes the fraying and yellow discoloration. Since heat is the major product of vibration, a small experiment was done. Fresh articular cartilage was exposed to the heat of a photoflood lamp until the synovial fluid bubbled and the cartilage turned yellow\(^2\). This only took a few moments exposure. Histologically, the chondrocytes had disappeared in the more superficial part of the cartilage, and the sections looked like those of the naturally occurring early stages of fibrocartilage damage.

In the earlier experiment, reference 1, there was fraying of the superficial cartilage layers as well as increased eosinophilia of the cartilage matrix and some eosinophilic debris on
the surface. Chondrocytes had not disappeared and there was no yellow color apparent to the naked eye. This obviously poses a problem for the pathogenesis involving heat. Until and unless additional experimental work is done, I can suggest that the normal forces generated in the experiment were not of the magnitude of those occurring in vivo (and certainly did not produce the amount of heat in the photoflood experiment). Sufficient oscillation and normal forces were generated to cause fraying and eosinophilia but insufficient to kill chondrocytes.

Obviously, these two experiments cannot be considered decisive, but they do show at least that heat can produce damage similar to that seen in the naturally occurring lesion, and that vibration can cause damage to the articulating surfaces. I cannot conceive of an experiment in which one could measure in vivo the generation of heat sufficient to damage the navicular articular cartilage (and the deep face of the deep flexor tendon). Perhaps such an experiment could be done with some larger joint and sophisticated, miniaturized temperature sensors. Such an experiment is, however, beyond my means and ability.

This scenario while in some measure hypothetical does agree with the available facts. Vibration as a basic cause of navicular disease is certainly supported by the type of animal (large body/small foot) and type of work (jumping, work on hard roads). Central to this hypothesis is that the initial damage is to the surface of navicular fibrocartilage surface of the fibrocartilage of the deep flexor tendon (kissing lesions).

Other Hypotheses

The major competing hypothesis is that navicular disease is a vascular disease. While Colles was an early proponent of this idea, it can be found in even older literature. Colles believed he saw thrombi/emboli in small vessels in the navicular bone and that these were the primary cause of the damage. The failure here was simply that what he considered to be thrombi and/or emboli were, in fact, only postmortem sludging of blood in smaller vessels. Unfortunately, heparin and, later, other anticoagulants were bruited as the “cure.”

It is difficult to have coherent arguments with clinical colleagues who insist that anticoagulants are effective. There are never controls or even convincing uncontrolled data. One of the standard statements is: “in properly selected cases this therapy works.” No doubt that is sometimes correct, but the faulty logic is glaring: if the therapy didn’t work, the case was not properly selected and, so, the therapy is never incorrect! That will lead me, below, to say something about the diagnosis of navicular disease.

First, however, another vascular “lesion” proposed by clinicians in Utrecht needs consideration. The hypothesis was that small vessels in the digit had greatly thickened muscular coats which impeded circulation to the navicular bone. Unhappily the Dutch workers were as inexperienced with histological examination of the tissues in this area as was the case in England. What they described was simply the normal appearance of many small arteries in the digit of horses. It is sad to say, but true, that if either group had
consulted experienced pathologists and/or histologists these errors – now of many years duration – need not have occurred.

In pursuit of the vascular hypothesis Dutch workers later carried out some beautiful experiments in which the blood vessels entering the distal border of the navicular bone were blocked (I forget just how). After a period of time angiograms showed that vessels entering the proximal border of the bone appeared to have dilated/proliferated as seen in cases of bona fide navicular disease. Although they apparently did not recognize the fact, such “activation” of collateral circulation can follow any sort of damage to a tissue and does not necessarily imply blockage of vessels as the primary event. It was also the case that the interruption of the distal vessels did not, in fact, produce damage to the articular cartilage – did not produce navicular disease.

Another hypothesis appearing from time to time suggests that sclerosis of the cancellous inner structure of the navicular bone causes the later onset of surface damage. Experiments have shown that injecting stiff material such as methylmethacrylate into the subchondral areas of rabbit joints will lead to arthrosis, but sclerosis sufficient to mimic that artificial effect is not seen until the late stages of arthrosis of any joint in any animal or human. That is, the sclerosis follows the surface damage and does not precede it.

There is a gradual increase of bone mass and density in the navicular, proximal sesamoids, carpal and tarsal bones, among others, with age and work. The distinction to be made is between normal weight- and work-induced increase of bone and true sclerosis with the latter being a pathological process. The distinction is not an easy one and certainly not to be made with radiographs alone.

Recently the fad for novel hypotheses for navicular disease has spread to the ligamentum impar, that ligament running from the distal border of the navicular bone to the coffin bone. I frankly have not seen a convincing argument or even a modest argument for involvement of the impar and discuss it no farther.

**Diagnosis**

As already suggested misdiagnoses can lead to remarkable “cures” of navicular disease. Pathological observations clearly show that real damage to the surfaces of the navicular and the deep flexor tendon is irreversible; progression to catastrophe the only outcome. A classic example of misdiagnosis lives with us still and will repay discussion. In the ‘60s a radiologist in Australia was diagnosing navicular disease based on the size and shape of the synovial loculi on the distal border of the bone. These loculi were called vascular channels which they are not. This radiologist stated that some large percentage (I forget the actual number) of the Standardbred horses admitted to the Sydney clinic for lameness examination had navicular disease. This came as a huge surprise. Having examined the navicular area of hundreds of racing Standardbreds at postmortem, I could categorically state that these horses never had the disease. Only if put to buggies by the Amish, trotting on macadam roads, or turned to jumping would these horses develop navicular disease.
Despite my protests then, in Sydney and later almost everywhere else, this myth of diagnosis by “vascular channel radiography” persists.

At one time a well-known radiologist and I agreed to follow up any case with “vascular channel lesions.” He would let me know when such a horse which he had so diagnosed on radiographs reappeared for postmortem for whatever reason. There were not many, of course, but some. He would come to the postmortem room while I dissected the legs, and it was clear that the frequency of damage to the navicular was completely unrelated to the previous radiographic observations. That was some years ago. I left that venue and the last I heard that radiologist was back to the vascular channels again! C’est la vie.

I might note that navicular disease does not occur in Thoroughbred racing horses either. It can occur, again, when the animal is used for purposes other than racing. Thoroughbred race horses, among others, certainly can have painful feet (low heels, long toes, etc.) which can be “cured” by heel nerving and even with blood thinning agents, chiropractic, acupuncture, and even aroma therapy. All these cures work (sic) because the animal did not have navicular disease to start with. So-called navicular shoeing (rolled toe, raised heels) will frequently alleviate the pain in these animals whether or not the navicular area is involved or some other undiagnosed process is at work.

The literature and many talks on navicular disease reflect the confusion in the minds of horsemen and veterinarians. One hears often of “navicular syndrome” or, in the European literature, “podotrochlosis.” These terms are most unfortunate. What is really being said is that the horse is sore in a foot, the navicular and deep flexor surfaces are not obviously affected (true navicular disease is not present), and the writer/speaker is giving a name to an undiagnosed condition.

**Appendix**

\[ \text{DFb-(Fa+CEc)=0} \]  \[ \text{[1]} \]
Fig.2: I present this picture once again with little hope that it will be attended to.

However, I try:

$F =$ ground reaction force experienced by the foot
$a =$ moment arm of $F$ around the center of rotation in the distal end of the middle phalanx (P2). This force acting around this distance (arm) causes a turning force or torque which is known as a **moment**.

$CE =$ tensile force in common extensor tendon and extensor branches of the suspensory.
$c =$ moment arm of $CE$

$DF =$ tensile force in the deep flexor tendon
$b =$ moment arm of $DF$

This position of the foot is obviously that of the standing horse but also that of the horse moving at slower speeds with the foot impacting flat-footed unto the surface. The coffin joint is rotating clockwise (in this view) which decreases tension in the deep flexor tendon ($DF$) while the fetlock is rotating counterclockwise which increases the tension in the deep flexor tendon. This is the normal situation with either flat-foot or heel-first impact of the foot with the surface. NOTE: the clockwise rotation at the coffin joint is defined as the movement of P2 on P3.
Fig. 3: (above): Toe first impact of the foot with the surface as in a horse landing from a jump. \( a \), the moment arm of \( F \) is much larger. As the foot rotates very rapidly from this to the flat foot position, the coffin joint is rotating counterclockwise while the fetlock joint is rotating counterclockwise (closing its front, dorsal angle). As a result there is a momentary increase beyond normal in the tensile force, \( DF \), and so the normal force at right angles to \( DF \). Remember that the direction of rotation of the coffin joint is defined as the movement of \( P2 \) relative to \( P3 \).
Fig.4: Heel first impact as seen at the faster gaits. The coffin joint is rotating clockwise which decreases the tension $DF$ as the fetlock is closing and increasing the tension $DF$. This is the same as the situation with flat-foot impact.

Figs.1 and 3 represent the normal situation at slow and faster speeds, respectively. Fig.2 is not normal and is the type of impact seen routinely with jumping horses and tired horses and as a phase of the vibratory or rocking movement of the foot in horses working on hard surfaces. On these hard surfaces, such as police and carriage horses on city streets, the foot may impact in any of these positions and then rock back and forth with consequent repeated increases of the normal force of $DF$, in Fig.5. Such “rocking” can barely if at all be appreciated by the naked eye. If one listens carefully to horses walking or, better, trotting on pavement, one can often hear two or more impact sounds. Accelerometer studies of hoof impact on soft and hard surfaces would be interesting.

Fig.5: Schematic of the rocking back and forth ,1-2-3 : 3-2-1, of the foot on hard surfaces. Exaggerated.

The question occasionally arises as to why the fore feet are subject to navicular damage while the hind feet are much less so. There are two answers: the first is that the hind feet
do not impact toe-first when the horse lands from a jump. A more important answer, perhaps, is that the interaction of the coffin and fetlock joints is somewhat different in the hind feet. That is, upon impact of the hind foot, the coffin joint rotates almost completely to its midsupport position before the fetlock joint begins to rotate. Thus, no matter the toe-first impact only the coffin joint is moving and increasing tension in the deep flexor tendon. The increasing normal force exerted on the navicular bone, then, from impact to midsupport is occasioned by the movement of the coffin joint only.


[2] Dr. Neil Williams of the Livestock Disease Diagnostic Laboratory in Lexington, Kentucky did this experiment for me.