Lamenesses that are dramatically improved or eliminated with a palmar digital nerve block (PDNB) are numerous and can be difficult to specifically characterize and/or localize. In one report, it was estimated that more than one third of all chronic equine lamenesses were temporarily improved or eliminated with a PDNB. Numerous anatomic structures can be involved in “heel-origin” lameness, and with the advent of alternate imaging modalities (such as scintigraphy and computed tomography), more specific localization can sometimes be made. However, the nonspecificity and lack of correlation between radiographic and clinical findings can often result in confusion regarding a specific diagnosis.

Generally, lamenesses that originate in the palmar one third to one half of the foot are considered when improvement in lameness is seen after PDNB. However, the palmar digital nerve courses forward to innervate most of the sole, so this characterization is not completely accurate. Further, anecdotal support exists for improvement in certain lamenesses originating from regions outside this classically considered area. Structures that could be “blocked out” from a PDNB include some or all of the distal phalanx, the distal aspect of the deep digital flexor (DDF) tendon, the digital cushion, ligaments associated with the navicular bone, the navicular bone, the navicular bursa, and the hoof capsule and sensitive laminae. These structures therefore encompass a wide range of possibilities for “heel-origin” lameness.

One problem encountered in attempting to sort out lameness of heel origin is the nonspecificity of clinical signs associated with many of these conditions. Generally navicular syndrome horses have been considered to be those with a chronic, insidious onset (often bilateral) forelimb lameness, with pain to hoof tester pressure over the middle third of the frog, that show dramatic improvement to PDNB, and that have variable radiographic changes. However, similar signs can be seen in horses with certain foot imbalances or horses with underrun heels or insufficient heel support. Also, disagreement exists over the value of the hoof tester response in predicting navicular region pain vs. pain from other sources (such as the distal interphalangeal [DIP] joint), or the insertion of the DDF tendon (DDF insertional tenopathy). In one report on 118 affected horses, only 11% were positive to hoof tester pressure, which is a much lower percentage than many veterinarians feel they get clinically.

In another study, the predictive values of diagnostic tests for navicular pain were evaluated. The hoof tester response was determined to be the least sensitive manipulative test for navicular pain (compared with distal limb flexion and the frog wedge test), and was more likely to elicit a positive response in other types of palmar heel pain rather than navicular syndrome. Despite this finding, most veterinarians in the field still seem to rely more on hoof tester response than they do on flexion tests and
wedge tests. Other unexpected clinical findings were documented in a study where kinematic variables were evaluated in horses jogging on a treadmill before and after PDNB. Navicular syndrome horses had a shorter cranial stance phase after PDNB than they did before PDNB. Their total stance phase was also significantly shorter after PDNB. There are plausible explanations for these findings, but they underscore the fact that some of our conventionally held beliefs regarding this clinical entity are not necessarily supported by research findings.

A further impediment to confirmation of a specific diagnosis is the confusion that currently surrounds responses to regional and intrasynovial anesthesia of the distal limb. Traditionally decreased lameness after PDNB suggested that the navicular region was the source of pain. Decreased lameness after DIP joint anesthesia was thought to target the joint as the source of pain, and navicular bursal injections targeted the bursa or DDF tendon. Considerable basic and clinical research has been completed examining the sensory innervation to the foot and the relevance of the different clinical approaches to distal limb anesthesia.1–3,5–9

For years questions existed regarding potential communication between the DIP joint and the navicular bursa. Despite anatomic studies showing only rare communication when contrast media was injected into either synovial space, Keegan et al.8 demonstrated effective tissue levels of mepivicaine in the navicular bone and navicular bursa after DIP joint injection. Bowker et al.9 demonstrated that potential sites for a “filtering membrane” type of diffusion for local anesthetics were through the impar ligament at its intersection with the deep digital flexor tendon, and the loose connective tissue proximal to the collateral sesamoidean ligaments. They also showed that injections into the navicular bursa resulted in dye diffusion that surrounded the neurovascular bundle (palmar digital nerves) within the digit. DIP injections also resulted in dye staining within the medullary cavity of the navicular bone. Other work by this group documented the presence of sensory nerves within the dorsal and palmar parts of the collateral sesamoidean ligaments, the impar ligament, and the periaricular connective tissues, recognizing that anesthetic diffusion could affect multiple sensory nerves supplying multiple anatomic structures of the distal limb.7

Clinical comparisons have also been made regarding improvement in lameness after PDNB, DIP joint anesthesia, and navicular bursal anesthesia.3,5,6 Early conclusions from some of these studies were that DIP joint anesthesia could also eliminate pain emanating from non-DIP joint structures but that bursal anesthesia was specific for bursal anatomic structures. DIP joint anesthesia is thought to desensitize the joint itself, as well as the navicular suspensory apparatus, portions of the third phalanx and of the DDF tendon, and periaricular neuroreceptors present in the loose connective tissues associated with the neurovascular bundle.7 However, it remains unclear as to the extent of desensitization of navicular bursal structures that results from DIP joint anesthesia. Turner reported on 80 horses with palmar heel pain, where all horses received PDNB, DIP joint anesthesia, and bursal anesthesia over a 4- to 5-day period.3 The goal of the study was to characterize the utility of diagnostic tests in horses with caudal foot lameness. Cases were categorized as those having navicular region pain (NRP) vs. those having other palmar heel pain (PHP). Specificities of these blocks (ability of the test to detect only the condition in question) for NRP were 0% for PDNB, 87% for DIP joint anesthesia, and 59% for bursal anesthesia. The positive predictive value (ability of a positive test to predict disease) was 66% for PDNB, 89% for DIP anesthesia, and 72% for PB anesthesia. From this study, Turner concluded that DIP anesthesia was the best “block” to use to confirm NRP. Turner and others have also reported that a positive response to DIP anesthesia within 5 to 10 minutes suggests a DIP joint problem, whereas improvement that takes 20 to 30 minutes is probably associated with lameness originating in an area associated with further diffusion of the anesthetic.5,7 Some of Bowker’s anatomic observations would support this clinical assumption based on relative proximity of the different sensory nerves to the joint surface. However, Pleasant et al.6 induced temporary lameness in horses by injecting the navicular bursa with amphotericin B. Anesthetic was then injected into the DIP joint, and blinded evaluation of videotape by four observers confirmed improvement in navicular bursal lameness by 5 minutes after DIP joint anesthesia, and improvement was still present 30 minutes later. This finding underscores the relative uncertainty surrounding what significance to attach to improvement in lameness after some of these distal limb “blocks.” There is also some evidence that navicular bursal anesthesia can yield results similar to a low PDNB, although this finding is not consistent.3 The clinical relevance of this composite of information is that anesthetic placed into the DIP joint will likely desensitize the navicular bursa as well as the DIP joint but could also desensitize all structures innervated by the palmar digital nerves. Advances in imaging of the foot may also help sort out some of the confusion that exists regarding palmar heel pain. Using scintigraphy, technetium uptake in the navicular bone is observed in some horses with palmar heel pain that have negative radiographic findings.3,10,11 Soft tissue and bone phase scintigrams have been shown to provide similar information, although in selected cases only one or the other phases show positive findings.11 Lateral view bone phase images were also found to be less sensitive than palmar view bone phase images, and images taken 1 hour after technetium administra-
tion were as diagnostic as those taken 2 to 4 hours after administration.\textsuperscript{10} Navicular contrast bursography has also shown promise in the diagnosis of flexor defects of the navicular bone, as well as in detecting adhesions between the DDF tendon and the bone.\textsuperscript{3}

More recently, Bowker \textit{et al.}\textsuperscript{12} examined anatomic structures of the foot and published findings that necessitate reexamination of functional foot anatomy. They identified a much more substantial ungual cartilage complex in clinically “healthy” feet. They also reported previously undescribed ungual cartilage attachments to the distal phalanx, the deep digital flexor tendon, and the navicular bone. Additionally they identified a more extensive vascular system within thicker ungual cartilages that contained prominent venovenous anastomoses. They theorized that these findings may support a more integrated system of force dissipation within the foot, with this ungual cartilage vascular system having a hemodynamic function. This is at least one possible explanation why feet with long toes and underrun heels (where the ungual cartilages and vasculature are less well developed) may be less effective at distributing forces and eventually develop heel and foot pain. Furthermore, their finding of tissue morphology differences in digital cushions between healthy feet vs. feet with “weak heels” suggests a more functional role for the digital cushion in force dissipation. Their other discovery of peptidergic sensory nerve endings associated with the vascular channels of the ungual cartilages and the recognition that they are closely allied with previously described Pacinian-like corpuscles in this region suggest that this part of the foot may respond to sensitive and proprioceptive stimuli. These findings further demand that we reexamine some previously held theories about evaluation of foot function.

References
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