

Injection of corticosteroids, hyaluronate, and amikacin into the navicular bursa in horses with signs of navicular area pain unresponsive to other treatments: 25 cases (1999–2002)

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Objective—To determine history, clinical and radiographic abnormalities, and outcome in horses with signs of navicular area pain unresponsive to corrective shoeing and systemic nonsteroidal anti-inflammatory drug administration that were treated with an injection of corticosteroids, sodium hyaluronate, and amikacin into the navicular bursa.

Design—Retrospective study.

Animals—25 horses.

Procedure—Data collected from the medical records included signalment, history, horse use, severity and duration of lameness, shoeing regimen, results of diagnostic anesthesia, radiographic abnormalities, and outcome.

Results—17 horses had bilateral forelimb lameness, 7 had unilateral forelimb lameness, and 1 had unilateral hind limb lameness. Mean duration of lameness was 9.2 months. All horses had been treated with corrective shoeing and nonsteroidal anti-inflammatory drugs for at least 6 months; 18 had previously been treated by injection of corticosteroids and sodium hyaluronate into the distal interphalangeal joint. Fourteen horses had mismatched front feet, and 21 horses had signs of pain in response to application of pressure over the central aspect of the frog. Palmar digital nerve anesthesia resulted in substantial improvement in or resolution of the lameness in all horses. Twenty horses (80%) were sound and returned to intended activities 2 weeks after navicular bursa treatment; mean duration of soundness was 4.6 months. Two horses that received numerous navicular bursa injections had a rupture of the deep digital flexor tendon at the level of the pastern region.

Conclusions and Clinical Relevance—Results suggest that navicular bursa treatment may provide temporary improvement in horses with signs of chronic navicular area pain that fail to respond to other treatments. (*J Am Vet Med Assoc* 2003;223:1469–1474)

It has been estimated that approximately a third of all horses with chronic forelimb lameness have pain localized to the navicular area.¹ Many structures in the caudal aspect of the foot can be a source of pain in horses, including the distal sesamoid (navicular) bone, palmar aspect of the **distal interphalangeal (DIP)** joint, navicular bursa (bursa podotrochlearis manus),

distal aspect of the **deep digital flexor tendon (DDFT)**, digital cushion, collateral sesamoidean (suspensory navicular) ligaments, hoof capsule, and sensitive laminae. A diagnosis of pain localized to the navicular area is usually made on the basis of history, clinical signs, response to **palmar digital nerve (PDN)** or DIP joint anesthesia, and detection of radiographic lesions. However, the diagnosis is not always straightforward. There is a recognized group of horses with clinical signs of navicular area pain that have no radiographic abnormalities.² In addition, interpreting the effects of PDN anesthesia versus DIP joint or navicular bursa anesthesia has become controversial, especially in light of recent evidence indicating that anesthetics injected in the DIP joint may have nonspecific effects.^{3,4} Advanced diagnostic imaging techniques such as nuclear scintigraphy, magnetic resonance imaging, and computed tomography are beneficial, but often unavailable.

Problems associated with localizing the underlying cause in horses with navicular area pain lead to difficulties in developing a therapeutic plan. Many horses with navicular area pain will respond to corrective shoeing, systemic **nonsteroidal anti-inflammatory drug (NSAID)** administration, and injections of corticosteroids and sodium hyaluronate in the DIP joint. However, there are some horses that improve after PDN or DIP joint anesthesia but fail to respond to shoeing, NSAID administration, and DIP joint treatment.

The purpose of the study reported here was to determine history, results of diagnostic testing, clinical findings, radiographic abnormalities, treatment, and outcome in horses with navicular area pain unresponsive to corrective shoeing and systemic NSAID administration that underwent injections of corticosteroids, sodium hyaluronate, and amikacin into the navicular bursa.

Criteria for Selection of Cases

Medical records of all horses examined at Texas A&M University between 1999 and 2002 because of lameness localized to the navicular area were reviewed. Horses that received injections of corticosteroids, sodium hyaluronate, and amikacin in the navicular bursa because of a lack of response to corrective shoeing and systemic NSAID administration were eligible for inclusion in the study.

Procedures

Information obtained from the medical records included signalment, previous treatments, history,

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horse use, shoeing regimen, affected limb or limbs, severity and duration of lameness, diagnostic tests performed, radiographic findings, treatment, and outcome. Diagnostic tests performed to localize the cause of the lameness consisted of perineural anesthesia with a 2% solution of mepivacaine and intra-articular anesthesia of the DIP joint. Diagnostic anesthesia was performed on the more severely affected limb first. In all horses, radiographs of the distal phalanx and navicular bone were obtained with high-detail radiographic film^a and a portable radiographic machine.^b Four radiographic views were obtained: a lateromedial view (65 kVp, 0.14 mAs, and 28-inch film-focal distance [FFD]), a dorsoproximal-palmarodistal oblique (DPr-PaDiO) view of the navicular bone made at 60° proximal to the supporting surface and obtained with a grid^c (70 kVp, 0.24 mAs, and 20-inch FFD), a DPR-PaDiO view of the distal phalanx made at 60° proximal to the supporting surface and obtained without a grid (60 kVp, 0.12 mAs, and 28-inch FFD), and a palmaro-proximal-palmarodistal oblique (PaPr-PaDiO) view of the navicular bone made at 45° proximal to the supporting surface (65 kVp, 0.14 mAs, and 20-inch FFD).

Prior to injection of medications into the navicular bursa, PDN anesthesia was performed at the level of the proximal sesamoid bones, except that if PDN anesthesia had been performed for diagnostic purposes, additional anesthetic was not given. Hair was clipped from the palmar aspect of the pastern region, extending from the coronary band to the abaxial aspects of the proximal sesamoid bones, with a No. 40 blade, and the area was prepared for aseptic injection. Horses were sedated with detomidine (0.006 mg/kg [0.003 mg/lb], IV) if required. Horses were allowed to stand on a clean, dust-free, level surface, and the foot to be injected was placed on a 2 × 6-in board prior to injection to facilitate postinjection radiography. Allowing the horse to stand in stocks was useful but not necessary for the procedure. The hoof was cleaned and wrapped with elastic tape to minimize contamination of the injection site. An 18-gauge, 3-in spinal needle with stylet was used for the injection. The needle was inserted on the midline at the distal extent of the depression created by the junction of the collateral cartilages and the DDF, at the proximal extent of the digital cushion (Fig 1). The needle was angled 10 to 30° downward, relative to the supporting surface. At a depth of 1.5 to 2.0 in, a hard resistance, representing the flexor surface of the navicular bone, was felt.

Correct placement of the needle in the bursa was confirmed by means of fluoroscopy^d or radiography. On a lateromedial radiographic view, the needle was assumed to be correctly placed if the tip was midway between the proximal and distal borders of the flexor surface of the navicular bone (Fig 2). After injection into the navicular bursa, sterile gauze sponges were applied to the injection site and secured in place with elastic tape for 48 hours.

Following navicular bursa injection, horses were treated with phenylbutazone (2.2 mg/kg [1 mg/lb], IV, q 24 h) for 5 days. Horses were confined to a stall or small run for 1 week, followed by 1 week of light riding, before they were allowed to return to normal activ-



Figure 1—Photograph illustrating placement of a needle for injection of drugs into the navicular bursa in a horse. The needle was inserted on the midline at the distal extent of the depression created by the junction of the collateral cartilages and the deep digital flexor tendon and angled downward at a 10 to 30° angle.



Figure 2—Lateromedial radiographic projection of the foot of a horse illustrating correct placement of a needle for injection of drugs into the navicular bursa. The needle was assumed to be correctly placed if the tip was midway between the proximal and distal borders of the flexor surface of the navicular bone.

ities. Therapeutic shoeing was performed as dictated by the horse's conformation, hoof shape, and activity.

Follow-up information was obtained a minimum of 6 months after navicular bursa injection; all horses were examined at the veterinary teaching hospital by 1 of the authors. Outcome was considered successful if the horse returned to its previous level of performance without evidence of lameness.

Results

Signalment—Of 310 horses with a lameness localized to the navicular region that were examined at the veterinary teaching hospital during the study period, 25 (12.4%) fulfilled the criteria for inclusion in the study. There were 21 geldings, 3 mares, and 1 sexually intact male. Mean age was 11.5 years (median, 11.0 years; range, 5.0 to 21.0 years). Twenty-three were Quarter Horses, 1 was a Thoroughbred, and 1 was a Warmblood. Twelve horses were used for roping, 8 were used for barrel racing, 2 were used for ranch work, 1 was used for dressage, 1 was used for combined training, and 1 was used for cutting.

History—Duration of lameness prior to examina-

tion at the teaching hospital ranged from 1 month to 3.5 years (mean, 9.3 months).

Previous treatments—Two horses had been treated with isoxsuprine hydrochloride (1.8 mg/kg [0.8 mg/lb], PO, q 12 h), but owners reported minimal improvement in the lameness. All horses had been treated with phenylbutazone at some time in the past, but all owners reported diminished response to this medication. None of the horses had received phenylbutazone within 10 days prior to examination at the teaching hospital. All horses had had hoof balance and shoeing methods evaluated and corrected, if needed, at least 6 months prior to navicular bursa injection. Sixteen of the 25 (64%) horses had been shod at the teaching hospital by the clinic farrier under the supervision of an attending clinician. After proper trimming, all horses were shod with a rolled or rocker toe^{ef} shoe and a 2° wedge pad.⁵

Eighteen (72%) horses had previously been treated by means of injection of triamcinolone acetate (9 mg/joint) or methylprednisolone (40 to 60 mg/joint) and sodium hyaluronate^b (20 mg/joint) into the DIP joint. Seventeen horses did not have any improvement after this treatment. The remaining horse had some mild improvement but could still not return to normal activities because of lameness.

Clinical findings—Fourteen of 25 (56%) horses had mismatched front feet; in all of these horses, the limb that was more severely lame had a smaller foot and higher heel angle than the opposite limb. Two horses had sheared heels on 1 foot. Eight of 25 (32%) horses had feet with atrophied frogs. Application of pressure over the central frog region with a hoof tester resulted in signs of pain in 21 (84%) horses. Twenty-four of 25 (97%) horses had increased digital pulses on palpation. Seventeen horses had bilateral forelimb lameness, 7 horses had unilateral forelimb lameness (right forelimb in 6 horses and left forelimb in 1), and 1 horse had unilateral left hind limb lameness. Severity of lameness on initial examination ranged from grade 2 to 4 (mean, grade 2.5) on a scale from 1 to 5.

Diagnostic procedures—Diagnostic tests performed to localize the cause of the lameness consisted of PDN anesthesia with a 2% solution of mepivacaine and intra-articular anesthesia of the DIP joint. In horses with bilateral lameness, diagnostic anesthesia was performed on the more severely affected limb first. Palmar digital nerve anesthesia resulted in substantial improvement in or resolution of the lameness in all horses. After PDN anesthesia, 17 horses became lame in the opposite forelimb, but the lameness was typically of lesser severity and also resolved after PDN anesthesia. Sixteen (64%) horses were improved (by approx 1 lameness grade) after anesthesia of the DIP joint, but were still lame. Five (16%) horses did not improve. In the remaining 4 horses, the lameness resolved after anesthesia of the DIP joint.

Radiographic findings—In 17 horses, radiographs of both front feet were obtained, and in the remaining 8 horses, radiographs of only a single foot were

obtained. Thus, radiographs of 42 feet were available for review. Enthesiophyte production was evident at the attachment of the collateral sesamoidean ligament near the proximal recess of the navicular bursa on radiographs of 20 of the 42 feet (48%; Fig 3). Three (7%) feet had mineralization of the DDF; the lateromedial radiographic projection was most useful in identifying these lesions. Decreased density of the sagittal ridge of the flexor surface of the navicular bone or a lucent bone defect involving the flexor surface of the navicular bone was seen in 14 feet (33%; Fig 4). Medullary cavity sclerosis was evident in 22 (52%) feet. Flexor surface lucency and medullary cavity sclerosis were best seen on the PaPr-PaDiO radiographic view. Nine (21%) feet had an osteophyte at the attachment of the distal impar ligament. Six (14%) had a large cystic lesion in the navicular bone, and 4 (10%) had a spur on the lateral extremity of the navicular bone at the attachment of the suspensory ligament of the navicular bone. All feet had larger and more irregular synovial invaginations involving the navicular bone than normal; these changes were best seen on the DPr-PaDiO view obtained with a grid. Radiographic navicular bone

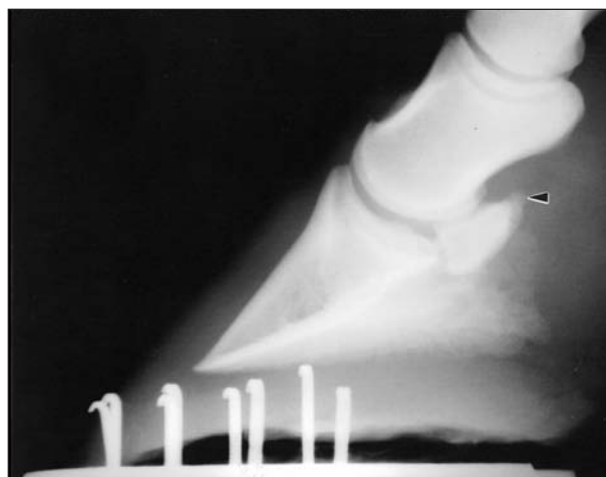


Figure 3—Lateromedial radiographic projection of the foot of a horse with lameness localized to the navicular region. An enthesiophyte is evident at the attachment of the collateral sesamoidean ligament (arrowhead).

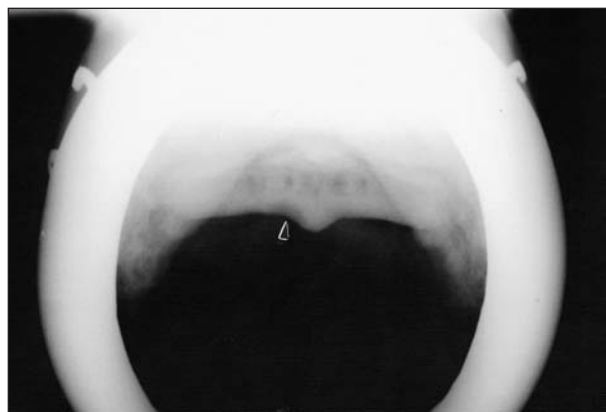


Figure 4—Palmaroproximal-palmarodistal oblique radiographic projection of the foot of a horse with lameness localized to the navicular region. Notice the lucent bone defect involving the flexor surface of the navicular bone (arrowhead).

abnormalities were considered severe in 10 (24%), moderate in 24 (57%), and mild in 8 (19%) of the feet, according to notes from the attending radiologist recorded in the medical record.

Treatment—Corrective shoeing was maintained in all horses. In addition, in all horses, the navicular bursa in the affected limb was injected with a combination of methylprednisolone (40 mg) or triamcinolone (6 mg), sodium hyaluronate (10 mg), and amikacin sulfate (125 mg). Five horses received multiple injections, consisting of a combination of methylprednisolone, sodium hyaluronate, and amikacin, in the navicular bursa at 6-month intervals (mean, 3 treatments).

Short-term outcome—In 20 of the 25 (80%) horses, the lameness resolved after navicular bursa injection, and the horses were able to return to intended activities (9 roping, 8 barrel racing, 1 combined training, and 2 ranch horses) approximately 2 weeks later. These 20 horses remained sound for 3 to 6 months (mean, 4.6 months) after initial treatment. Four (16%) horses remained lame (1 dressage, 1 cutting, and 2 roping horses). Three of these 4 horses had additional lesions causing lameness. One had a DIP joint chip fracture, and 2 had deep digital flexor tendonitis in the pastern region. The remaining horse, which was used for roping, returned to soundness initially after navicular bursa injection, but became acutely grade 4 of 5 lame approximately 6 weeks later. This horse had severe damage to the DDFT at the level of the pastern region. This horse was sold and lost to further follow-up.

Long-term outcome—Fourteen (56%) horses were being used for their intended activities 1 to 3 years after the initial navicular bursa injection (6 barrel racing, 6 roping, 1 ranch work, and 1 combined training), but required periodic navicular bursa injections or systemic NSAID treatment. Three horses (12%; 1 roping, 1 dressage, and 1 ranch horse) underwent posterior digital neurectomy on the affected limb. Two roping horses that had multiple navicular bursa injections had a rupture of the DDFT at the level of the pastern region. One was euthanized, and the other became a broodmare. One barrel horse dropped in performance level to become a child's horse, and a second barrel horse was later retired because of a stifle joint injury. One roping horse was euthanized because of lameness localized to the navicular area. One roping and 1 cutting horse remained lame, but had additional soft tissue injuries in the pastern region.

Discussion

Horses with chronic navicular area pain that fail to improve after therapeutic shoeing, rest, systemic NSAID administration, and injections of corticosteroids and hyaluronate in the DIP joint can be frustrating for owners and clinicians. Many owners are reluctant to have a palmar digital neurectomy performed because of concerns about potential complications associated with the procedure, such as painful neuroma formation, nerve regrowth, DDFT rupture, and severe foot abscess formation, or because of financial constraints. Navicular bursa treatments administered in the present study can be done in the field and

provided temporary relief from clinical signs of lameness in some horses with chronic navicular area pain that had become unresponsive to other treatments.

There has been a perception that injecting drugs into the navicular bursa is difficult.⁵ In our experience, however, the technique described in the present study was easily learned and could be performed in the field. The angle of needle insertion and depth of needle penetration were helpful guides for gaining access to the navicular bursa. In particular, a hard resistance, representing the flexor surface of the navicular bone, was felt after the needle was inserted 1.5 to 2.0 in. Deeper needle penetration usually resulted in placement of the needle ventral to the navicular bone, within the digital cushion. If the angle of needle insertion was too horizontal, the palmar aspect of the DIP joint was entered, and synovial fluid was usually obtained. In our experience, bursa fluid was usually not obtained after needle insertion into the navicular bursa. Regardless, we recommend that correct placement of the needle be confirmed by means of radiography or fluoroscopy.

We did find that injection of medication into the navicular bursa was met with resistance in some horses, presumably because of deep digital flexor adhesions. Retracting the needle 1 to 2 mm reduced resistance in these instances, as did flexing the limb at the carpal joint so that the horse was bearing weight only on its toe. As medication was being injected into the navicular bursa, back pressure in the syringe was often observed after deposition of 1 to 2 mL of medication.

Most horses in this study were middle-aged Quarter Horse geldings, and most of these were Western performance horses. This was not surprising, because many Quarter Horses have navicular area lameness problems, and geldings are often preferred in Western performance events. In a previous study⁶ involving palmar digital neurectomy in horses with navicular disease, 77% of 57 horses were between 7 and 15 years old, and Quarter Horses were overrepresented, compared with the hospital population.

Clinical findings in the present study were typical of those expected for horses with navicular area pain and included increased digital pulses, signs of pain in response to the application of pressure over the navicular region, and atrophy of the frog. Twenty-one of 25 (84%) horses in this study showed signs of pain in response to application of pressure over the central aspect of the frog, which was higher than the 45% of horses in a previous study⁷ of horses with navicular disease that had this response. Fourteen of 25 (56%) horses in this study had mismatched front feet, with the limb having the smaller foot and higher heel angle being more severely affected. This was higher than the percentage of horses with navicular disease that had hoof pair asymmetry (29%) in a previous study.⁸ It is possible that the smaller size of the hoof and abnormal hoof shape are a result of abnormal loading or weight bearing secondary to chronic pain.

In the present study, the lameness substantially improved or resolved in all horses after PDN anesthesia and in 20 horses after DIP joint anesthesia, but 17 of the 18 horses did not improve after injection of cor-

ticosteroids and hyaluronate into the DIP joint. Such cases represent a diagnostic and therapeutic challenge, especially with recent revelations concerning which structures are desensitized with PDN or DIP joint anesthesia. The previous concept that PDN anesthesia desensitizes structures in the palmar third of the foot and pastern region seems to underestimate the potential area of distribution. Lameness caused by certain conditions of the DIP joint,⁹ osteoarthritis of the proximal interphalangeal joint,⁹ lamellar pain,¹⁰ pedal osteitis,^{9,10} fractures involving the proximal aspect of the first phalanx,¹¹ and solar pain¹² can be eliminated or improved by PDN anesthesia. Distal interphalangeal joint anesthesia can attenuate pain arising from the DIP joint, the navicular bursa,¹³ and the solar region,¹² depending on volume of anesthetic injected and time after injection.³ Additionally, horses that improve after DIP joint anesthesia could have lesions involving the articular surface of the navicular bone, the dorsal or abaxial surfaces of the collateral suspensory ligament of the navicular bone, or the navicular impar ligament, as there may be diffusion of the anesthetic into the navicular bursa.^{4,14,15}

The navicular bursa is a thin synovial structure located between the navicular suspensory apparatus and the distal aspect of the DDFT. It has been suggested that lame horses that improve after navicular bursa anesthesia have lesions involving the navicular bursa or palmar aspect of the collateral suspensory ligament of the navicular bone, inflamed tissue or adhesions between the distal portion of the DDFT and navicular bone, or exposed subchondral nerves on the flexor surface of the navicular bone.^{14,15} Because horses in this study improved after PDN anesthesia, but not after injection of corticosteroids and hyaluronate into the DIP joint, we suspect that 1 of these other structures was damaged, causing clinical signs of pain and lameness in these horses.

We did not use navicular bursa anesthesia to localize the cause of the lameness in the horses in the present study, although this procedure has been advocated by other authors.¹⁶ In our experience, inserting a needle into the navicular bursa in horses without first performing PDN anesthesia requires heavy sedation of the horse, which could alter the interpretation of the response to navicular bursa anesthesia. Additionally, there are risks associated with navicular bursa injection, including DDFT trauma and sepsis, and we thought it would be better to limit the number of times a needle was inserted into the navicular bursa. In addition, although it would have been beneficial to know the outcome of navicular bursa anesthesia, we thought that there were limited therapeutic options for these horses. Once a diagnosis of navicular pain unresponsive to systemic NSAID administration and corrective shoeing was made, we elected to treat the navicular bursa directly and use the response to treatment as a diagnostic tool. Other diagnostic methods, such as navicular bursography,¹⁷ nuclear scintigraphy,² and magnetic resonance imaging, may have assisted in localizing the cause of the pain in these horses, and a few horses in this study did undergo nuclear scintigraphy. However, the procedure lacked specificity for indi-

vidual structures in the navicular region. Navicular bursography has similar problems as does navicular bursa anesthesia, and magnetic resonance imaging was not available.

Thirty-four of 42 feet for which radiographs were obtained in the present study had moderate (24; 57%) or severe (10; 24%) radiographic abnormalities of the navicular bone. This was not unexpected, given the long duration of lameness prior to examination at the teaching hospital (mean, 9.3 months). Twenty of the 42 (48%) feet had enthesiophytes at the attachment of the collateral sesamoidean ligament near the proximal recess of the navicular bursa. These horses may have had previous damage to or avulsion of the ligament, resulting in bony remodeling of its attachment to the proximal aspect of the navicular bone. This is consistent with previous reports^{14,15} that suggest that horses with lesions involving the palmar aspect of the collateral sesamoidean ligament would not improve after DIP joint anesthesia. Although some of the horses in this report improved after DIP joint anesthesia, they did not respond to injection of corticosteroids and hyaluronate into the DIP joint. A recent study¹⁸ found that mepivacaine diffuses from the DIP joint to the navicular bursa or vice versa in horses. We suspect that local anesthetics may have different diffusion capabilities than corticosteroids and hyaluronate, but studies of the diffusion of various medications and anesthetics after intra-articular administration are needed. Nevertheless, our findings do suggest that horses with radiographic lesions suggesting previous collateral sesamoidean ligament damage may respond to treatment of the navicular bursa if other treatments have failed.

Drugs injected into the navicular bursa in horses in the present study were selected on the basis of the attending clinician's preference. The volume of the navicular bursa is approximately 3 mL,¹⁷ and during navicular bursa injections, strong resistance was felt after approximately 2 mL of fluid had been injected. We suspected that the capacity of the bursa may have been decreased by adhesions between the DDFT and navicular bursa in some horses. The small volume of the bursa limits the quantity of medication that can be injected. Generally, 1.0 mL of sodium hyalurate combined with 1.0 mL of a corticosteroid and 0.5 mL of amikacin was used in these horses.

Results of the present study do not provide any clear-cut indications for which horses will benefit from navicular bursa treatment, and response to treatment is likely to be dependent on many factors, including severity of disease, horse use, conformation, and owner compliance. On the basis of our experience, we believe that horses with lameness localized to the navicular area that have radiographic evidence of enthesiophytes at the attachment of the collateral sesamoidean ligament near the proximal recess of the navicular bone or that have radiographic evidence of mild erosive lesions along the flexor surface of the navicular bone suggestive of mild DDFT lesions or adhesions may respond to navicular bursa treatment. In addition, we believe such treatment may be indicated in horses with navicular area pain, with or without radiographic abnormalities,

that do not respond to corrective shoeing, systemic NSAID administration, or injections of corticosteroids and sodium hyaluronate into the DIP joint and in competition horses with navicular area pain unresponsive to other treatments that need temporary pain relief until palmar digital neurectomy can be performed.

Three of the 25 (12%) horses in the present study had severe damage to or rupture of the DDFT after navicular bursa treatment. One horse initially became sound after the navicular bursa was treated, but became severely lame 6 weeks later. Although we recommended that the horse be maintained in corrective shoes after the navicular bursa treatment, the owner elected to remove the shoes and turn the horse out into a large pasture. We suspect that this horse had DDFT lesions or adhesions prior to navicular bursa treatment and that additional damage to the DDFT was sustained during the turnout period because of a decrease in pain associated with the navicular bursa treatment. The other 2 horses had received numerous navicular bursa injections over a 2-year period. During necropsy of the horse that was euthanatized, severe DDFT adhesions with tearing of the DDFT at the flexor surface of the navicular bone was seen. Because horses responding to navicular bursa treatment may have DDFT lesions or adhesions, we recommend adding a 2 or 3° heel wedge pad or shoe to the horse's shoeing regime to decrease pressure between the DDFT and flexor surface of the navicular bone.¹⁹ It seems that repeated injections into the navicular bursa increase the likelihood of complications and should be performed with caution. One horse in the present study had an immediate increase in the severity of lameness following navicular bursa injection, possibly because of an acute reaction to the medication.

Horses with acute ligamentous lesions in the navicular area are not good candidates for navicular bursa treatment, as lessening the pain in these horses may allow a return to normal activity and progression of the ligament damage. We usually treat such horses with heel elevation and extended stall rest. Horses with radiographic evidence of severe erosive lesions along the flexor surface of the navicular bone are also not candidates for navicular bursa treatment, because of the risk of DDFT tearing or rupture.

Results of the present study suggest that horses with chronic lameness localized to the navicular region that are unresponsive to corrective shoeing and systemic NSAID administration may respond to injections of corticosteroids and sodium hyaluronate into the navicular bursa. Twenty of 25 (80%) horses returned to their intended activity within 2 weeks after this treatment and remained free from lameness for a mean of 4.6 months. Fourteen (56%) horses were being used for their intended purpose 1 to 3 years after navicular bursa treatment. Because none of these horses were able to perform their intended activities prior to the navicular bursa treatment, the 56% success rate, although low, seems reasonable. Navicular bursa treatment may provide temporary improvement in horses with chronic navicular area pain that fail to respond to other treatments.

^aKodak Etascan Ed-RA-1, Ultradetail Plus, 3M Co, St Paul, Minn.

^bHF80 Plus, MiniRay Inc, Northbrook, Ill.

^cX-ray grid, 6:1 ratio, 40 lines/cm, Mallinckrodt Inc, Hazelwood, Mo.

^dFluro machine, Xiscan, Xitec Inc, East Windsor, Conn.

^eNatural Balance Shoe, Equine Digital Support System, Penrose, Colo.

^fTennessee navicular shoe, C&M Horseshoes, Conroe, Tex.

^gH 2° pad, C&M Horseshoes, Conroe, Tex.

^hHylartin-V, Pharmacia & Upjohn Co, Kalamazoo, Mich.

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