Excision of the Deep Branch of the Lateral Palmar Nerve of Horses to Resolve Lameness Caused by Proximal Suspensory Desmitis

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Objective: To assess outcome after neurectomy of the deep branch of the lateral palmar nerve (DBLPaN) as a treatment for horses with persistent lameness associated with chronic proximal suspensory desmitis (PSD) of the thoracic limb.
Study Design: Case series.
Animals: Adult, mixed-breed horses (n = 4), weighing 510–585 kg, used for amateur show-jumping.
Methods: Records of 4 horses chronically lame because of PSD of one or both thoracic limbs that were treated by neurectomy of the DBLPaN were reviewed. The site of pain causing lameness was localized using regional anesthesia. The proximal aspect of the suspensory ligament of the affected limb(s) of all horses were enlarged on ultrasonographic examination, but fiber disruption was not observed. All horses remained lame after conservative therapy. Neurectomy was performed with the horses anesthetized and positioned in dorsal recumbency.
Results: All 4 horses were sound at 6 weeks and remained sound for at least 12 months after neurectomy.
Conclusion: Lameness in horses caused by chronic PSD can be resolved by neurectomy of the DBLPaN in horses that are refractory to conservative management.

Desmitis is a frequently diagnosed condition of the proximal aspect of the suspensory ligament (PSL) of the pelvic or thoracic limb of performance horses.[1,2] Proximal suspensory desmitis (PSD) can occur unilaterally or bilaterally.[3,4] Treatments include confinement[4]; injection of a corticosteroid into the tissue surrounding the proximal aspect of the suspensory ligament[4]; intralesional injection of autogenous bone marrow[5] or autogenous platelet-rich plasma[6]; radial pressure wave therapy[7]; and application of an egg-bar shoe.[8] About 90% of horses lame because of acute PSD of a thoracic limb become sound after confinement followed by graduated return to exercise.[9] Premature resumption of training may result in recurrence of injury.[4] When desmitis is chronic, lameness can be persistent, and treatments ineffective.[4]

Unlike horses with PSD of a thoracic limb, horses with PSD of a pelvic limb usually remain lame after being treated by confinement followed by a program of controlled exercise.[10,11] In one study, this treatment enabled only 14% of 42 horses with PSD of one or both pelvic limbs to return to their previous level of activity for >1 year without recurrence of lameness.[10] Only 18 of 43 (41%) horses lame for at least 3 months because of PSD of one or both pelvic limbs were sound and in work 6 months after being treated by a period of confinement coupled with radial pressure wave therapy.[7]

The suspensory ligament of the pelvic limb is innervated by the lateral and medial plantar metatarsal nerves, which originate from the deep branch of lateral plantar nerve (DBLPlN), a branch of the lateral plantar nerve, which branches from the tibial nerve,[9] and consequently, horses chronically lame because of PSD of one or both pelvic limbs have been treated with good results by tibial neurectomy or by resection of part of the DBLPlN, with or without transection of the fascia plantar to the suspensory ligament (ie plantar fasciotomy).[9,12–15] In a study of 271 horses with PSD of one or both pelvic limbs, 214 (79%) were able to return to their previous level of exercise after undergoing plantar fasciotomy and excision of a portion of the DBLPlN of one or both pelvic limbs.[16] In a recent study, 78/84 horses (91%) that had bilateral neurectomy of the DBLPlN for the treatment of PSD returned to their intended use.[17]

The proximal aspect of the suspensory ligament of the thoracic limb is innervated by the deep branch of the lateral palmar nerve (DBLPaN) and the lateral and medial palmar metacarpal nerves, which originate at the level of the proximal end of the 4th metacarpal bone from the DBLPaN,

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which branches from the lateral palmar nerve at the level of the midcarpal region, and contains fibers from the ulnar and the median nerve.[18,19] Based on the good results achieved by excising a portion of the DBLPlN in returning horses lame because of chronic PSD of one or both pelvic limbs to soundness, we theorized that horses lame because of chronic PSD of one or both thoracic limbs could be returned to their previous level of exercise by excising part of the DBLPaN.

MATERIALS AND METHODS

Medical records (2010–2011) of horses with chronic PSD in one or both thoracic limbs and treated by resection of a portion of the DBLPaN were reviewed.

Inclusion Criteria

For horses to be included, lameness had to have been substantially improved after the lateral palmar nerve of the lame limb was anesthetized proximal to its deep branch or after local anesthetic solution was infiltrated around the origin of the suspensory ligament, after the horses showed no improvement in lameness following a low, palmar nerve block (ie a low, four-point nerve block). Lameness was graded according to the scoring system devised by the American Association of Equine Practitioners.[20] Evaluation of the affected limb(s) included ultrasonographic examination of the palmaroproximal tissues of the metacarpus (Esaote MyLab 30, 12-mHz linear transducer with stand-off, Esaote, Genova, Italy), performed by a clinician experienced in ultrasonography, and radiographic examination of the proximal aspect of the metacarpus and carpus.

All horses had the same medical treatment before neurectomy. This treatment included infiltration of a corticosteroid (10 mg triamcinolone acetonide) around the origin of the affected suspensory ligament(s), performed at the time of diagnosis; radial pressure wave therapy (Masterplus MP100, Storz Medical AG, Switzerland) performed 3 times at 2-week intervals; and stall confinement for 2 weeks, beginning at the time of diagnosis, followed by a program of incrementally increasing exercise. The program of exercise entailed 6 weeks of daily walking, either in hand or on a walker, followed by 4 weeks of daily walking and trotting, under saddle, with increasing intensity. Horses were re-evaluated when presented for each treatment of radial pressure wave therapy and sporadically during the period of controlled exercise. At 12 weeks after the initiation of medical treatment (ie at the end of the exercise program), if horses were still lame, a lameness investigation, which included nerve blocks and ultrasonographic and radiographic examinations, was repeated.

Surgical Technique

Neurectomy was performed with the horse anesthetized and positioned in dorsal recumbency with the affected limb(s) extended. Hair at the surgical site was clipped, and the surgical site was prepared for aseptic surgery. A 5-cm, longitudinal, skin incision was made adjacent the lateral border of the superficial digital flexor tendon, extending from 1 cm distal to the palmarodistal aspect of the accessory carpal bone to the level of the head of the 4th metacarpal bone or slightly distal to it (Figs 1 and 2). A stab incision was made in the exposed peritendinous fascia and extended to the length of the skin incision using scissors, taking care to avoid the underlying neural and vascular structures (Fig 2). The peritendinous fascia was retracted with Gelpi retractors, and the lateral palmar nerve was located adjacent to the distal border of the accessoriometacarpal ligament by using blunt dissection with a Halstead mosquito hemostat (Fig 3).

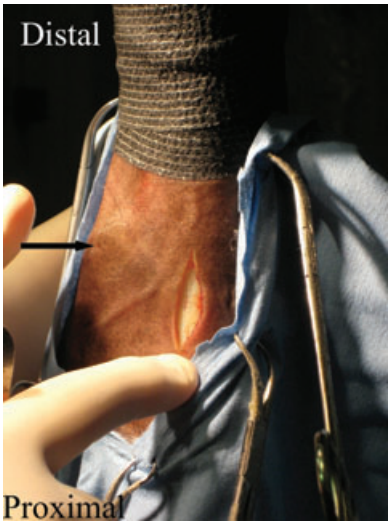

Figure 1 Landmarks of surgical approach to DBLPaN. Palmarolateral view of the right carpal region with the horse in dorsal recumbency. The incision is made adjacent the lateral border of the superficial digital flexor tendon, extending from 1 cm distal to the palmarodistal aspect of the accessory carpal bone (fingertip) to the level of the head of the 4th metacarpal bone (black arrow), exposing peritendinous fascia.

The DBLPaN was located either closely adjoined to the lateral palmar nerve or coursing toward it in a dorsodistal to palmaroproximal direction. The DBLPaN was distinguished from the lateral palmar nerve by its smaller size and by its position deep to the lateral palmar nerve and palmar vein. The DBLPaN was elevated with a curved Halstead mosquito hemostat, and ~1 cm of the nerve was excised with a scalpel (Fig 4). The nerve was transected distally and then proximally at the site where it merged with the lateral palmar nerve. As long of a portion of DBLPaN as possible was removed to reduce the likelihood of re-innervation of the proximal aspect of the suspensory ligament.

The peritendinous fascia and subcutaneous tissue were closed separately with 2–0 polydioxanone suture using a simple-continuous pattern, and the skin incision was closed with the same suture using a simple-interrupted pattern. The surgical site was covered with a sterile dressing, and a padded bandage that extended from the foot to the

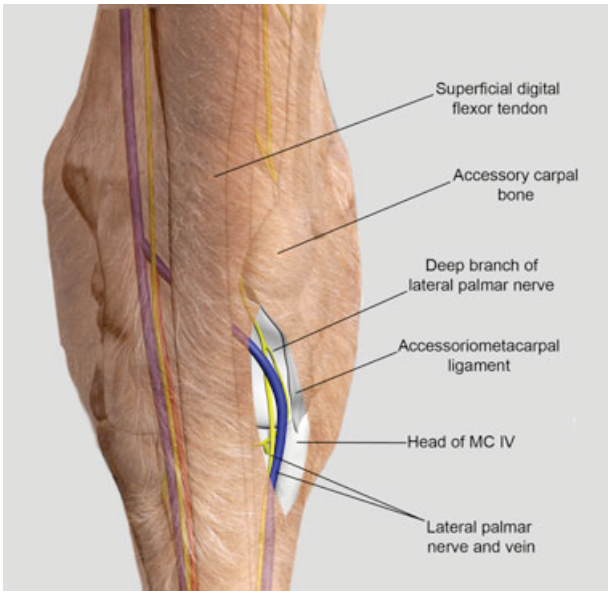

Figure 2 Palmar view of a right carpus, showing anatomy and landmarks for incision. The incision begins about 1 cm distal to the palmarodistal aspect of the accessory carpal bone and extends ~2 cm distal to the level of the head of the 4th metacarpal bone. (Picture courtesy of John Schumacher and Ray Wilhite.).

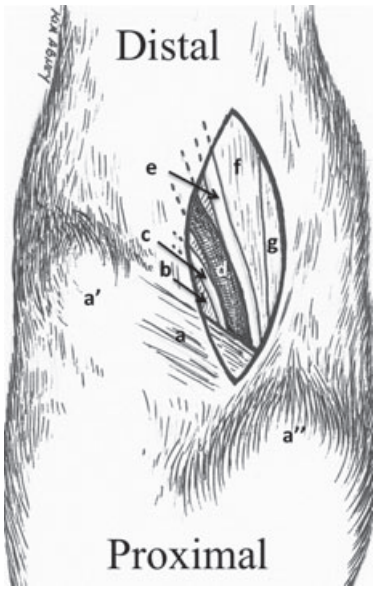

Figure 3 A slightly oblique view of the right carpus, with horse in dorsal recumbency, showing: a, carpal fascia (accessoriometacarpal ligament); a', head of fourth metacarpal bone; a'', accessory carpal bone; b, accessory ligament of the deep digital flexor tendon (inferior check ligament); c, deep branch of the lateral palmar nerve; d, lateral palmar vein; e, lateral palmar nerve; f, deep digital flexor tendon; and g, superficial digital flexor tendon.

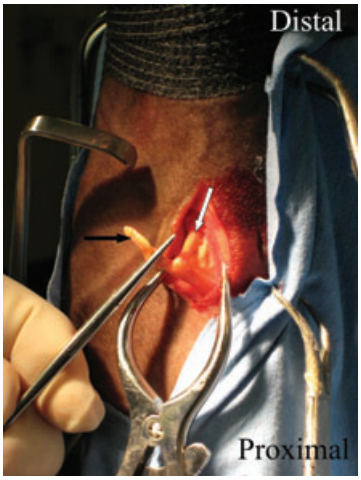

Figure 4 The DBLPaN (black arrow) is severed distally and exteriorized using a Halstead mosquito hemostat. The lateral palmar nerve (white arrow) lies superficial to it. Tissues are retracted using a Gelpi retractor.

middle of the antebrachium was applied. The bandage was removed the day after surgery and replaced with a bandage that extended from the foot to the carpus. The limb was maintained in a bandage for 2 weeks, and the dressing and bandage were replaced every 3-4 days.

All horses were administered gentamicin sulfate (6.6 mg/kg intravenously [IV]), procaine benzylpenicillin (20,000 U/kg intramuscularly [IM]) and tetanus antitoxin (5000 IU intramuscularly [IM]) before surgery. Phenylbutazone (4.4 mg/kg IV) was administered preoperatively and every 12 hours the day after surgery, and then orally (2.2 mg/kg) every 12 hours for 3 days. Skin sutures were removed 12–14 days after surgery. Horses were confined to a stall for 6 weeks. During weeks 3 and 4, the horses were walked in hand or on a walker for 10 minutes twice daily. During weeks 5 and 6, to initiate muscular conditioning, they were walked in hand or on a walker for 10 minutes and trotted in hand for 10 minutes twice daily.

Six weeks after surgery, horses were examined while they walked in a straight line on a hard surface and trotted in a straight line and on each rein on soft and hard surfaces. Horses were allowed to return to the level of exercise at which they performed before the onset of lameness (ie amateur show-jumping) if they were sound at a trot. All horses were examined for soundness 6 months after surgery. Owners were questioned by telephone interview 12 months after surgery about the horse's soundness.

RESULTS

Four, mixed-breed, mature geldings, weighing 510–585 kg (mean, 545 kg), 7 (n = 2) and 12 (n = 2) years old that were used for amateur show-jumping and were lame because of chronic PSD in one or both thoracic limbs were treated by resection of part of one (n = 2) or both (n = 2) DBLPaNs.

Table 1 Lameness Grades Before and After Medical Treatment

Horse	Age	Duration of Lameness Before Medical Treatment (Weeks)	Primary Affected Thoracic Limb	Lameness Grade Before Medical Treatment Primary Limb	Contralateral Lameness Grade Before Medical Treatment	Anesthesia Technique	Lameness Grade of Primarily Affected Limb After Medical Treatment	Lameness Grade of Contralateral Limb After Medical Treatment
1	7	2	Right	2/5	0/5	Local infiltration OSL	2/5	0/5
2	12	4	Left	3/5	0/5	Lateral palmar nerve	2/5	0/5
3	7	4	Left	2/5	1/5	Lateral palmar nerve	2/5	1/5
4	12	4	Right	2/5	2/5	Local infiltration OSL	2/5	2/5

OSL, origin of suspensory ligament.

Horses were initially presented for examination 2–4 weeks after onset of lameness (Table 1). Two horses were lame unilaterally (Horse 1: right thoracic limb, grade 2/5; Horse 2: left thoracic limb, grade 3/5).[20] Two horses were lame bilaterally (Horse 3: affected primarily on left thoracic limb, grade 2/5; Horse 4: affected primarily on right thoracic limb, grade 2/5).[20] After lameness of the primarily affected limb was abolished with local or regional anesthesia, lameness of the contralateral thoracic limb in Horses 3 and 4 became apparent (Horse 3: contralateral lameness on right thoracic limb, grade 1/5; Horse 4: contralateral lameness on left thoracic limb, grade 2/5).[20] The site of pain causing lameness was localized to the palmaroproximal aspect of the metacarpus when lameness was abolished by anesthetizing the lateral palmar nerve proximal to the DBLPaN as it coursed medial to the accessory carpal bone as described by Castro et al[21] using 3 mL 2% mepivacaine hydrochloride (3 limbs, 2 horses) or by instilling 3 mL 2% mepivacaine hydrochloride at the origin of the suspensory ligament (3 limbs, 2 horses), after observing a negative response to a low, palmar nerve block, performed using mepivacaine hydrochloride, as described by Moyer et al[19] Methods of anesthesia were based on clinician preference.

No abnormalities were noted on radiographic examination of the carpus and the proximal aspect of the metacarpus of each affected limb. The proximal aspect of the suspensory ligament of the affected limb(s) was determined to be enlarged on ultrasonographic examination by comparing the size of the proximal aspect of the suspensory ligament to normal reference values for cross-sectional area (CSA) and dorsopalmar thickness[22] for all horses and also by comparing the size of the proximal aspect of the suspensory ligament of the affected limb to that of the sound limb in unilaterally affected horses, but exact measurements of CSA and thickness of the suspensory ligament were not recorded. No evidence of fiber disruption or focal mineralization was observed during ultrasonography of the enlarged suspensory ligaments.

The lameness of 1 unilaterally lame horse (Horse 2; see Table 1) improved by 1 grade (from grade 3/5 to grade 2/5) after it received the nonsurgical treatment described above, but the owner, who considered this degree of improvement to be unsatisfactory, requested that the horse be treated by neurectomy of the DBLPaN. No substantial improvement in lameness was noticed in the other 3 horses (Horses 1, 3, and 4) during or after the 12 weeks of nonsurgical treatment.

The site of pain causing lameness was reconfirmed to be the proximal, palmar aspect of the metacarpus when lameness failed to resolve after conservative treatment, by desensitizing the proximal aspect of the suspensory ligament, using the same method of anesthesia as when the horse was first presented for evaluation, after observing no improvement in lameness after administering a low, palmar nerve block. For each of these 4 horses, radiographic and ultrasonographic findings were similar to those observed before medical treatment.

At the time of suture removal, a seroma was observed at the surgical site of 1 horse that had unilateral neurectomy. The owner reported that the seroma resolved after firm compressive bandaging of the area for 1 week. No other horse experienced a complication from surgery.

All 4 horses were sound at walk in a straight line on a hard surface and sound when trotted in straight line and on each rein on hard and soft surfaces when examined at 6 weeks and at 6 months after neurectomy. All 4 horses had returned to their previous use.

For each horse, the site(s) of surgery appeared unblemished at 6 months. Each owner stated during a telephone conversation 12 months after surgery that his or her horse had remained sound on the operated limb(s). One horse (Horse 3) had developed lameness of a pelvic limb, but the cause of lameness had not been determined.

DISCUSSION

We found that chronic lameness caused by proximal suspensory desmitis of one or both thoracic limbs can be abolished, without serious complication, by neurectomy of the DBLPaN when desmitis is not accompanied by ultrasonographically detectable disruption of fibers in the suspensory ligament or by radiographic abnormalities at the proximal aspect of the third metacarpal bone.

Proximal suspensory desmitis of a thoracic limb is suspected if pain is localized to the palmaroproximal aspect of the metacarpus using local or regional analgesia.[1–4] The proximal aspect of the suspensory ligament of a thoracic limb can be desensitized by anesthetizing the lateral palmar nerve before it gives off its deep branch, either on the medial aspect of the accessory carpal bone[21] or as it courses adjacent to the distal border of the accessoriometacarpal ligament.[23] Alternatively, local anesthetic solution can be

infiltrated around the site of insertion of the proximal aspect of the suspensory ligament on the palmaroproximal aspect of the third metacarpal bone.[23] Structures other than the proximal aspect of the suspensory ligament may be desensitized, however, using regional or local anesthesia, and therefore, the diagnosis of PSD is usually confirmed by the presence of ultrasonographic abnormalities of the proximal suspensory ligament. Other imaging modalities, such as radiography, nuclear scintigraphy, computed tomography, and magnetic resonance imaging may also be useful in confirming its diagnosis.[4]

Ultrasonographic abnormalities associated with PSD of a thoracic limb include enlargement of the cross-sectional area of the suspensory ligament, poor demarcation of the margins of the SL, focal or diffuse areas of reduced echogenicity, focal hypoechogenic or anechogenic core lesions, focal mineralization, and entheseous new bone on the proximal aspect of the palmar cortex of the third metacarpal bone.[4]

Part of the DBLPaN was excised from the lame limb(s) of these 4 horses after determining that the suspensory ligaments were devoid of ultrasonographically identifiable fiber disruption. We believe neurectomy of the DBLPaN is an inappropriate treatment for horses with chronic PSD when disruption of fibers in the proximal aspect of the suspensory ligament has been identified during ultrasonographic examination because removing sensory innervation to the weakened suspensory ligament may increase the risk of the horse exacerbating the injury or even incurring catastrophic disruption of the suspensory ligament. The results of the procedure should be evaluated in larger number of horses to determine the risk of the procedure resulting in damage to the desensitized portion of the suspensory ligament.

The suspensory ligaments of these 4 horses were not reexamined ultrasonographically after being denervated and consequently, we have no information about the effect of denervation on the integrity of the proximal aspect of the suspensory ligament of these horses. Pauwels et al demonstrated that after neurectomy of the DBLPlN, the skeletal muscle fibers in the proximal aspect of the suspensory ligament of the pelvic limbs undergo neurogenic atrophy,[24] and similarly, neurectomy of the DBLPaN of the thoracic limbs likely results in neurogenic atrophy of muscle fibers of the proximal aspect of the suspensory ligament. We can find no reports that describe the effects of denervation of the proximal aspect of the suspensory ligament of the pelvic or thoracic limb on the strength of the denervated suspensory ligament.

A complication caused by transection of a palmar digital nerve is formation of a painful neuroma.[25] None of the 4 horses that had transection of the DBLPaN had signs of formation of a painful neuroma, such as lameness of the treated limb or signs of pain during palpation of the surgical site,[26] and we can find no report that describes formation of a painful neuroma as a complication of transection of the DBLPaN.

The lateral palmar nerve and its deep branch lie superficial to the carpal sheath, which extends from the level of the distal aspect of the middle third of the antebrachium to the proximal aspect of the middle third of the metacarpus.[27] Although inadvertent penetration of this synovial structure is a possible complication of neurectomy of the DBLPaN, we saw no evidence during surgery that the carpal canal was penetrated, and we know of no adverse consequences that might occur from inadvertent penetration of it, provided that neurectomy is performed under sterile conditions.

Trauma to the lateral palmar artery or vein resulting in avascular necrosis is a potential complication of neurectomy of the DBLPaN because these vascular structures lie in close proximity to the peritendinous fascia and to the DBLPaN. Inadvertent transection of one or both of these vessels may lead to acute hemorrhage during surgery or to avascular necrosis of tissue distal to the surgical site after surgery. We did not observe such complications during or after neurectomy of the DBLPaN, and we can find no reports that describe complications associated with trauma to the vasculature adjacent to the DBLPaN.

Horses that had neurectomy of the DBLPaN had no radiographically evident osseous abnormalities at the proximal aspect of the third metacarpal bone. Radiographic changes associated with chronic PSD include increased opacity caused by sclerosis of the trabeculae of the palmar cortex of the third metacarpal bone at the site of insertion of the suspensory ligament.[4,28] We do not know the effect of DBLPaN neurectomy on prognosis for soundness of horses lame because of chronic thoracic PSD accompanied by radiographically apparent osseous changes of the third metacarpal bone, and we can find no reports describing the effect.

Persistence of lameness in horses with PSD of one or both pelvic limbs may be caused by compression of the plantar metatarsal nerves, which innervate the proximal aspect of the suspensory ligament.[4,29,30] These nerves, which branch from the DBLPlN, become compressed between the suspensory ligament, enlarged from injury, and dense fascia plantar to the ligament. Tóth et al examined the DBLPlN of horses determined to be lame on one or both pelvic limbs because of PSD and found histological changes suggestive of chronic compression in these nerves.[30] These investigators theorized that chronic compression of the plantar metatarsal nerves may be the cause of pain causing lameness rather than pain originating in the proximal aspect of the suspensory ligament.[30] Chronic neural compression may also be a cause of chronic lameness of some horses with PSD of a thoracic limb. During dissection of the proximal palmar aspect of the metacarpal area, connective tissue fibers have been observed coursing transversely across the distal aspect of the DBLPaN (R Henry, J Schumacher, unpublished data, 2012); these fibers may contribute to chronic neural compression of the DBLPaN in horses with an enlarged proximal suspensory ligament. Studies involving histologic examination of the DBLPaN of the lame thoracic limb of horses lame because of chronic PSD are warranted to determine if the DBLPaN has lesions suggestive of compression neuropathy that might be

a source of pain in horses that are refractory to medical treatment for PSD.

Summarily, neurectomy of the deep branch of the lateral palmar nerve should be considered as a treatment for resolving lameness of horses caused by chronic, proximal suspensory desmitis of the thoracic limb(s) when horses are refractory to more conservative therapies, provided that proximal suspensory desmitis is not accompanied by ultrasonographically identifiable disruption of fibers.

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