SL) is defined as the region hindlimb suspensory ligament (SL) or metatarsophalangeal joint3. Prevent overextension of the plantar aspect of the fourth extending band originating on the hindlimb, the SL originates in injury. It is not possible to pal- often chronic nature of the There are frequently no localis- Clinical examination There are frequently no localis- pat the proximal suspensory ligament (PSL) because of the position between the second and fourth metatarsal bones. In acute injury, distension of the medial plantar vein, localised pain9 and oedema are some- times found. PSL is often accompanied by medialateral foot imbal- ance1,15. Hindlimb PSL may result in secondary back pain, including development of pain from pre-existing dorsal spinous processes5. Horses with metatar- sophalangeal hyperextension or straight hock conformation (Figure 1) are predisposed1,11,12, and in old brood mares1,15, which may indicate age-related degeneration. A positive response to both a proximal limb flexion is not unusual4,5. Many horses present with lameness most evident when the affected limb is on the side of the circle on a soft surface. However, there is no pathognomonic gait abnormality9 or pattern in lameness for hindlimb PSL. Analgesia Ultrasonographic and radio- graphic changes are sometimes subtle, resulting in diagnostic anaesthesia for diagnosis. Pain resulting from PSL can be partially improved with “low-4(6)-point” analgesia9 probably due to proximal suspension of local anaesthe- tic solution. Proximal suspensory liga- ment desensitisation can be achieved by anaesthesia of the proximal medial and lateral metatarsal nerves11,18,19,21; tibial nerve, DBLPb using a nerve block (DBLPb), and local infiltration15. The author uses the DBLPb due to its simplic- ity and option of neuroectomy if response is marked. There is also a reduced risk of radi- 10,15; and tarsal15,16 shear. If the response is unclear, it is useful to perform tibial nerve analgesia, which alleviates PSL pain without significantly influ- encing tarsal pain4. There has been evidence of DBLPb causing desensitisa- tion of the lateral heel bulb to the distal aspect of the fourth metatarsal bone on the plan- tarolateral aspect of the limb, indicating analgesia of the lateral plantar and plantar metatarsal nerves1,15. It is, therefore, advisable that analgesia of the distal aspect of the limb be undertaken first. When there is a significant response (greater than or equal to 75% improvement), following DBLPb, then TMT) analgesia on a separate occasion due to the possible inadvertent entrance of the TMT) plantar pouch17, and results compared. If there is a positive but less than 75% per cent improvement following DBLPb, then TMT) analgesia and/or infiltration of the provonplantar MTIII region can be assessed to any coexistence of distal tarsal or proximal MTIII entheseous pain and PSL respectively. Bilateral DBLPb can be undertaken in horses with poor hindlimb impu- lse. Analgesia of one limb in these cases may not result in visible lameness in the contralateral limb, as would be expected19, yet bilateral analge- sia can result in marked riddenn improvement. Figure 2. Longitudinal ultrasonographic image of the right proximal metatarsal region of a nine- year-old, working hunter pony at Zones 1A and 1A/1B. Proximal is to the left. There is a convex contour of the plantar aspect of the SL reflecting swelling (red arrows), loss of long fibre pattern (white arrow) and mildly irregular bone (green arrow) at the origin of the suspensory ligament. Diagnostic imaging – Ultrasonography For ultrasonography of the PSL, the limb must be approached from the plantaromedial aspect18. The plantar aspect of MTIII must be visualised to ensure SL changes are not artifactual. The size of this “window” can be narrow, affecting image quality. Due to the depth of the ligament and position between the second and fourth metatarsal bones, the medial and lateral margins are not as evident. Use of a convex-array transducer; “virtual convex” application or “stand-off” pad may alleviate these problems. Pathology seen on ultrasonography may include enlargement (Figure 2), poor margin definition, loss of fibre pattern (Figure3), areas of hypoechoegenicity, both cen- trally (Figure 4) and peripherally, hyperchoegenitic (Figure 3), short fibre pattern on longitudinal images (Figures 2 and 3) and MTIII plantar con- tex irregularity (Figure 2). Analgesic techniques can cause artefacts. Because of this it is advisable to leave 24 hours between analgesia and ultrasonography. Presence of muscular tissue and shadowing artefacts15 resulting from fluid- filled structures (such as blood vessels) and round structures (such as overlying flexor ten- dons18) may also complicate interpretation. Longitudinal images highlight short fibre pat- tern and may be more sensitive than radiography at detecting plantar MTIII pathology20. Increased proximal cross sectional area (greater than 1.5cm) of the PSL has been recog- nised in sound horses4,25 and so should be interpreted with care. Radi- ology may underestimate the presence of new bone, which may be more reliably detected using computed tomography19 or MRI21,22. – Scintigraphy Scintigraphy may determine if there is active pathology at the origin of the SL, which is not evident radiographically20. This is especially important if ultra- sonographic changes do not fit the degree of lameness. Anal- gesia of the DBLPb may result in diffusion of local anaesthetic solution to the proximal aspect of MTIII. In the absence of ultrasonographic abnormality and radiographic examination may help to define active osseous lesions as the principal cause of pain, altering prognosis and treatment. Lack of increased radiopharmaceutical uptake (RIU) is a common finding with PSL unless there is an avulsion injury29. In a study of 126 horses, only 12 per cent continued on page 12
with hindlimb PSD had IRU identified subjectively in bone phase images.

- Magnetic resonance imaging

If there is a positive response to analgesia, but ultrasonography is negative or equivocal, MRI should be undertaken of the distal hock and proximal metatarsal regions.

Some horses with osseous injury at the origin of the suspensory ligament where lameness is abolished following DLBLPN, have only been noted on MRI.

TREATMENT

Horses with hindlimb PSD respond poorly to rest alone, although horses with loss of support to the metatarsophalangeal joint need long-term rest regardless of therapy. Only 6/42 (14 per cent) of horses were able to resume work without lameness following DBLPnb, have achieved similar results.

Deconditioning and fasciectomy for core injuries resulted in 87 per cent of 23 horses returning to work.

Bioscaffold therapy (A-Cell) in combination with fasciectomy resulted in 84 per cent of 77 horses with fore or hindlimb PSD returning to full work.

The latter studies had a poorly defined follow-up period. Stem cell therapy and osteotomy undertaken when there is concurrent osseous pathology of the proximal aspect of MTIII has also been used. Stem cell therapy and (biocalcium treatment) may be an advantageous treatment for return of SL structure, but may not alleviate pain and lameness as the result of neural compression and compartment syndrome.

Such treatments may be useful in conjunction with neurectomy and fasciectomy to improve ultrasonographic fibre pattern, which is often not achieved following this surgery. Use of biophosphonates has also been reported as being useful in horses with enthesitis-related pain.

Tibial nerveectomy has been used as a treatment for PSD with 6/8 horses (75 per cent) returning to full athletic function for at least two years post-surgery. 

Neurectomy of the DLBLPN and fasciectomy in combination has resulted in success rates between 62 per cent and 91 per cent, with good long-term follow-up (greater than one year postoperatively) and no loss of sensation or proprioceptive deficits.

In most cases, the author recommends treatment of PSD with a neurectomy and fasciectomy if there is a diagnosis of primary PSD causing pain and lameness, significant response to DLBLPN (greater than equal to 75 per cent) and SL enlargement probably leading to compartment syndrome and neural compression.

Lameness and response to analgesic techniques is sometimes only seen unilaterally. Some horses become lame on the contralateral limb after treatment such as surgery when strenuous exercise resumes.

A histopathology study of the DLBLPN of 16 PSD cases that underwent nerveotomy found myxomatous expansion with Renault bodies in the subperineurium and nerve fascicles, and axonal degeneration.

This was also found in non-lame limbs if surgery was undertaken bilaterally.

In the non-lame limb these findings may indicate the presence of neural compression caused by an enlarged SL. Therefore it may be of benefit to undertake surgery in the non-lame contralateral limb if there are any ultrasonographic abnormalities.

Success of neurectomy and fasciectomy can be assessed four to six weeks following surgery.

Continued lameness at this point carries a guarded prognosis.

Ultrasonographic improvement often lags behind clinical improvement because structural improvement requires more time. Ideally, ultrasonographic improvement is gauged before any increase in exercise, regardless of clinical improvement.

In some cases there is no ultrasonographic improvement, despite soundness. Reduction in cross-sectional area is often seen, which may be resolution of desmitis, or the result of neurogenic atrophy of the SL muscular tissue. Painful neuromas did not occur in a study involving more than 250 horses, suggesting a reduced risk compared with palmar digital neurectomy.

Tarsotomy should also be addressed, such as reducing the dorsal hoof wall angle. This decreases the break-over reducing the requirement for limb flexion and decreases strain within the suspensory ligament. Medial lateral hoof imbalance should be addressed to prevent further SL aggravation.

Hyperextension of the metatarsophalangeal joint can be a sequel to PSD, especially in horses that appear to have progressive degeneration of the SL.

In these horses, the SL may continually degenerate despite surgery. Horses with hyperextension of the metatarsophalangeal joint and straight hock conformation are at greater risk of surgical failure, lameness reoccurrence, SL deterioration and SL breakdown and should not be candidates for neuroectomy and fasciectomy. Horses that undergo surgery for PSD with concurrent local pathology have a reduced prognosis for return to full athletic function.

Surgical repair of tarsal pain is reported with PSD. Surgical management of horses with PSD and tarsal pain may improve the overall prognosis.

The accessory ligament of the SL originates on the plantar aspect of the calcaneus and fourth tarsal bone and merges with the SL in the proximal metatarsal region. As neurogenic atrophy of the muscular tissue within the SL may occur following neuroectomy, it is possible the biomechanics of the distal hock parts may be altered, which may predispose to the development of distal hock joint pain.

References


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SHELLEY DOWN qualified from the University of Bristol in 2004 followed by an internship (based in orthopaedic diagnostics and imaging) under Sue Dyson at the Animal Health Trust (AHT), Newmarket. In 2006, Shelley worked as a large animal assistant in Cambridgeshire before becoming a senior clinical scholar in equine orthopaedics at the University of Cambridge. She then worked as an equine clinician at the AHT and gained her RCVS Certificate in Equine Surgery and Bone Disorders. She later worked as a visiting worker to complete research interests.

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