

# Medicine & Science

# The Official Journal of the American College of Sports Medicine

OVID

Wolters Kluwer



AMERICAN COLLEGE of SPORTS MEDICAL WINNING STORES

15580 8185-9121

# **Exercise-related leg pain: neurological perspective**

# **McCRORY PAUL**

Medicine & Science in Sports & ExerciseMedicine & Science in Sports & Exercise. 32:p S11-S14, March 2000.

#### **Author Information**

Olympic Park Sports Medicine Centre, Swan Street, Melbourne, AUSTRALIA 3004; and Department of Neurology, University of Melbourne, Austin & Repatriation Medical Centre, Heidelberg, Victoria, AUSTRALIA 3084

Submitted for publication November 1999.

Accepted for publication December 1999.

Address for correspondence: Dr. Paul McCrory, 31 Grosvenor Parade, Balwyn, Victoria, Australia 3103. E-mail: pmccrory@compuserve.com.

A 22-yr-old elite female distance runner presented with a 2-yr history of exercise-related lateral leg pain. This pain had become progressively worse as her level of training increased and was particularly bad when running in spikes on synthetic tracks. The pain came on after 200 m of sprinting and progressed to the point where she had to stop running after 800 m. Slow jogging did not seem to reproduce her symptoms. She described the pain as "burning," and it radiated down the lateral calf and into the dorsum of the foot. If she continued to run through the pain, she found that the same area became numb to touch and she developed a foot "slapping" gait. Examination at rest was normal. Postexercise examination demonstrated numbness over the dorsum of the foot, including the first web space. The remainder of her neurological exam was normal. Plain x-rays, bone scan, ultrasound, and compartment pressure testing was normal. Nerve conduction studies demonstrated a conduction block at the level of the peroneal tunnel (head of fibula) bilaterally that was worse postexercise.

Surgical exploration of the fibular tunnels demonstrated fascial entrapment of the common peroneal nerve and its branches. Neurolysis was performed with symptom resolution. She now competes at an international level without recurrence of her problem.

# Back to Top

# ANATOMICAL FEATURES OF NERVE ENTRAPMENT SYNDROMES

A summary of the nerves and their motor and sensory distribution in relation to the leg is shown in <u>Table 1</u>. Details of the individual nerves are provided in the section below. It must be emphasized that many nerve entrapment syndromes may present as nonspecific or poorly localized, and furthermore the cutaneous dermatomal distribution is extremely variable among individuals.

Nerve	Main Branches in Leg	Motor Innervation in Leg	Sensory Distribution in Leg
Femoral nerve	Saphenous nerve	Nil	Antero-medial knee and medial le
Common peroneal nerve	Superficial peroneal Deep peroneal, Lateral cutaneous,	Peroneus longus and brevis, tibialis anterior, extensor hallucis longus, extensor digitorum longus, peroneus tertius	Lateral calf
	Sural communicating		
Tibial nerve	Anterior tibial, Posterior tibial, Sural nerve	Gastrocnemius, soleus, tibialis posterior, flexor digitorum longus, flexor hallucis longus	Lateral ankle

Reference 24. Stewart, J. Focal Peripheral Neuropathies, 2nd Ed. New York: Raven Press, 1993, pp. 347-401.

Nerves of the lower leg (adapted from (24)).

In addition, radicular pain arising from compression or irritation of the lumbo-sacral nerve roots, referred pain from innervated structures of the spine, polyneuropathies, myopathies, and other forms of muscle disease, as well as the various chronic regional pain syndromes, must be considered in any neurological differential diagnosis.

#### Back to Top

# GENERAL APPROACH TO NEUROGENIC LEG PAIN

Unless nerve entrapment syndromes produce "hard" neurological signs of motor weakness, sensory loss or change in tendon reflexes, specific

diagnosis may be difficult and often circumstantial. For this reason a regional approach to the likely nerve entrapments may be useful. If the diagnostic suspicion is sufficient, then the appropriate electrophysiological studies are sought ( $\underline{28}$ ).

# Back to Top

# History.

The aim of obtaining the history of a patient with neurogenic leg pain is to determine the location and nature of the pain as well as the presence of specific neurological symptoms. Previous history of back pain and its response to treatment may be particularly important clues in diagnosis.

# Back to Top

# Key symptoms.

A number of important symptoms should be looked for specifically since they may indicate the presence of potentially serious disease. Patients reporting such symptoms should be referred immediately for neurological assessment. These include:

# Back to Top

# Examination.

Details of neurological examination skills are beyond the scope of this paper. Readers are referred to Bickerstaff et al. (2) for further reading. Specific points in the neurological examination may be worth noting; these include:

# Back to Top

# Investigations.

In the management of most cases of exercise-related leg pain in which a neurological diagnosis is suspected then specific confirmatory tests, such as nerve conduction studies or electromyography, should be sought ( $\underline{30}$ ). The main role of radiological imaging modalities, such as isotope bone scanning, ultrasound, and magnetic resonance imaging (MRI), relate to the detection of bone and soft tissue lesions rather than neurological disease.

Nerve conduction studies and electromyography is a specialized diagnostic technique that relies on the electrophysiological response of nerve and muscle. Areas of localized dysfunction of a nerve such as that seen with a nerve entrapment syndrome will be evident as a "conduction block" to electrical impulses. If dysfunction is severe or long standing, then there may be evidence of damage to the nerve axon itself. Needle electromyography may also be helpful in localizing and prognosticating the severity of the nerve lesion. Although such tests are useful, they have a number of limitations, most importantly the lack of correlation between clinical symptoms and electrophysiological abnormality. The sensitivity and specificity of such tests depends on what is being measured and by what techniques. In general terms, detailed discussion between the sports medicine clinician and the electrophysiologist will assist in improving the diagnostic yield.

#### Back to Top

# SPECIFIC NERVE ENTRAPMENT SYNDROMES

#### Back to Top

#### Saphenous nerve.

This is the longest sensory branch of the femoral nerve arising from the L1, L2, and L3 nerve roots. The nerve leaves the femoral triangle to enter the adductor canal (or subsartorial canal of Hunter). The two terminal branches of the saphenous nerve, the infra-patellar branch and the descending branch, arise within the canal (17,24). The infra-patellar branch supplies the sensation to the medial portion of the joint and the overlying antero-medial

skin. The descending branch accompanies the saphenous vein to supply the skin of the medial leg and foot.

The nerve may be injured in the adductor canal by local trauma or inflammatory conditions such as thrombo-phlebitis. In the region of the knee, surgery is the predominant cause. Some authors have speculated that stretching the nerve during repeated knee flexion may be a biomechanical explanation in which cases of saphenous nerve entrapment in body building have been reported (4,6,9,10,32).

The typical presentation is of claudicant medial leg pain that may be easily confused with vascular disorders. Compression of the nerve (or Tinel's sign) in the region of the adductor canal or where the nerve crosses the medial femoral condyle usually produces pain radiating to the medial malleolus. Alteration of sensation in the cutaneous distribution of the nerve may be present (29). Where diagnostic uncertainty exists, a local anesthetic block of the nerve within the canal may help isolate the syndrome. Treatment may either be conservative or involve surgical neurolysis (13,22).

# Back to Top

# **Common peroneal nerve.**

The common peroneal nerve (CPN) arises from the sciatic nerve in the distal thigh above the popliteal fossa. It then runs laterally in an exposed fibro-osseous tunnel passing below the tendinous origin of peroneus longus and enters the peroneal tunnel between the two heads of this muscle. The lateral sural cutaneous nerve branches from the CPN arise proximal to the fibular head. As the nerve enters the peroneal tunnel, the CPN divides into the deep, superficial, and recurrent peroneal nerves.

Injury to the main trunk of the CPN in the region of the peroneal tunnel frequently is caused by an external compressive source. Functional anatomical changes such as repetitive exercise involving inversion and pronation (e.g., runners, cyclists, machine operators) stretch the CPN against the peroneal tunnel, resulting in the clinical syndrome. Many cases

are idiopathic in origin (10,15). Involvement of the CPN or its branches may also be seen in conjunction with compartment syndromes, the DPN with a deep posterior compartment syndrome and the SPN with anterior compartment syndrome.

Few cases of CPN entrapment have been reported in runners (<u>11,14,23</u>). In all reported cases running induced the typical pain and numbness. Examination postexercise confirmed muscle weakness and a positive Tinel's sign at the level of the fibular neck. All patients were treated surgically with a high (90%) success rate after failure of conservative management.

The superficial peroneal nerve (SPN) runs between the fibula and the peroneus longus muscle and distally on the anterior inter-muscular septum. The SPN supplies the peroneal muscles. At the junction of the middle and distal third of the tibia, the nerve pierces the crural fascia and splits into two cutaneous branches. This nerve may be compressed by the crural fascia (the SPN syndrome) or where the terminal branches cross the anterior aspect of the ankle joint subcutaneously (1,12). The terminal branches provide the sensation to the antero-lateral leg, the dorsum of the foot and the dorsal skin of the great, second, third, and medial fourth toes. Clinical signs of the SPN syndrome include pain or anesthesia of the dermatome with resisted dorsiflexion and eversion (26,27). SPN entrapment has been reported in runners, soccer players, jockeys, tennis players, bodybuilders, and dancers (17). In these cases loss of sensation during exercise over the lateral distal calf or dorsum of the foot was noted. Relief by conservative measures was uncommon (25,26).

The deep peroneal nerve (DPN) pierces the anterior intermuscular septum and travels with the anterior tibial blood vessels distally. The nerve enters the foot under the cruciform ligament and supplies the sensation to the skin between the great and first toes and a medial branch to the extensor digitorum brevis muscle. All sports-related cases of DPN entrapment relate to anterior tarsal tunnel syndrome with symptoms referable to the foot rather than to the lower leg (<u>17</u>).

Treatment may be conservative with modification of the precipitating activity or biomechanical correction or if this is unsuccessful, surgical

decompression. If an underlying compartment syndrome is diagnosed with appropriate pressure studies, then surgical decompression is recommended.

# Back to Top

# Sural nerve.

The sural nerve usually originates from two main branches, the medial cutaneous sural nerve from the tibial nerve and the ramus communicans of the common peroneal nerve; however, in up to 20% of cases, based on cadaver studies, the origins and course of the sural nerve were variably absent (15,31).

The sural nerve begins in the popliteal fossa and then runs distally between the two heads of gastrocnemius beneath the crural fascia. Between the middle and distal thirds of the calf, the nerve pierces the fascia and runs distally near the small saphenous vein lateral to the Achilles tendon. The sural nerve then passes behind the lateral malleolus to supply the ankle joint, the posterior calf, and the lateral side of the heel and foot (<u>17</u>).

Although not commonly reported as a cause of exercise-related leg pain, compression of the nerve because of mass lesions, scar tissue, ganglia, surgical trauma, and thrombophlebitis have all been reported. Extrinsic compression from tight ski boots and casts may also induce this problem. The crural fascia may act as either a compression point or a fixation point for the nerve, and athletic activities such as running or track sports may stretch the nerve at this level. Similarly, repetitive ankle inversion injuries may lead to fibrosis and nerve entrapment (3,4,18-20).

Clinically patients usually report shooting pain or dysesthesias in the cutaneous distribution of the nerve, although objective signs may not always be present (21).

Back to Top

Tibial nerve.

The tibial nerve originates from the anterior divisions of L4-S3 via the medial trunk of the sciatic nerve. The sciatic nerve divides into the common peroneal and tibial nerves at the level of the popliteal fossa and the tibial nerve continues distally deep to gastrocnemius. The nerve divides into the anterior and posterior tibial branches.

Injury or entrapments affecting the tibial nerve in the leg are rare. In the popliteal fossa, Baker's cysts, popliteal artery aneurysms, and ganglia can compress the nerve, but no specific sport or exercise-related conditions have been reported ( $\underline{8}$ ). By contrast, entrapment of the distal branches, most notably the posterior tibial nerve as it passes through the tarsal tunnel are extremely common in sport ( $\underline{5}, \underline{16}$ ).

# Back to Top

#### Other neurogenic causes of exercise-related lower limb symptoms.

Apart from focal nerve entrapment syndromes discussed above, patients with other neurological conditions may present with symptoms referable to the lower limb. These may include myopathies, polyneuropathies, exerciserelated cramps, or leg weakness resulting from proximal nerve injury. Muscle pain may occur following unaccustomed or extreme exercise and may be severe and delayed in onset in its presentation. Clinicians need to be alert to the differential diagnostic possibilities in this situation and the patient history is the most important aspect of the overall assessment.

A patient with an underlying myopathic or neurogenic condition may present with a variety of symptoms. A feeling of weakness, fatigue, or tiredness is not unusual. Subjective muscle pain is not commonly reported in adults although muscle tenderness may be noted. Localized muscle wasting may be present, particularly in the inherited muscle diseases. If there is florid muscle weakness, then the patient may present for diagnosis; however, usually a concerned parent or coach notices the weakness that may be more evident following exercise. These symptoms are often bilateral and usually develop slowly. Some possible diagnoses are listed in <u>Table 2</u>.

#### TABLE 2. Causes of neurogenic lower limb weakness other then nerve entrapments.

Myopathic Inflammatory myopathies Endocrine, toxic or drug-induced myopathies Muscle dystrophies Myotonic dystrophy Acid maltase deficiency Benign congenital myopathies Neuromuscular blocking disorders Myasthenic syndromes Demyelinating polyneuropathy Neurogenic Diabetic amyotrophy Root or lumbar plexus lesion Motor neuron disease

Causes of neurogenic lower limb weakness other then nerve entrapments.

#### Back to Top

#### Muscle cramp syndromes.

Muscle cramps represent sudden, involuntary, and often painful shortening of muscle and may be attended by visible "knotting" of the muscle which may in turn lead to abnormal posturing of the affected joint. These are perhaps the most common neuromuscular disorder to affect an athlete and to occur universally. For the most part their origin is benign. The common cause of muscle cramps is muscle fatigue; however, other factors such as dehydration, vascular insufficiency, electrolyte imbalance, or unaccustomed muscle activity may be associated. Rare pathological causes include motor neuron disorders, hypothyroidism, muscle enzyme disorders, or specific movement disorders such as Isaac's syndrome. Treatment is usually symptomatic, and prevention by stretching, exercising in graduated programs, and ensuring adequate hydration before exercise is all that is usually required  $(\underline{7})$ .

#### Back to Top

# Overlap of clinical syndromes.

As previously discussed, there is considerable clinical overlap between vascular entrapment, compartment syndromes, and focal nerve entrapments in cases of exercise-related leg pain. In some situations, e.g., compartment syndrome affecting the peroneal or deep posterior compartments of the leg. exercise will produce neurogenic symptoms secondary to raised intracompartmental pressures. Similarly, popliteal artery entrapment may produce neural ischemia resulting in focal neurological symptoms. The astute clinician must: 1) be aware of the various causes of exercise-related leg pain and their overlap, 2) examine the patients following provocative exercise while the typical symptoms are present, and 3) appropriately investigate suspected cases to establish a pathophysiological diagnosis before definitive management. For most sports medicine clinicians, the common causes of exercise-related leg pain relate to tibial stress fractures and other muscular problems. In cases where the presentation is atypical, where the patient describes prominent neurological symptoms, or where the initial bone investigations are unrevealing, then consideration of the conditions described above may be helpful.

# Back to Top

# REFERENCES

1. Adelman, K., G. Wilson, and J. Wolf. Anterior tarsal tunnel syndrome. J. Foot Surg. 27:299–302, 1988.

Cited Here...

2. Bickerstaff, E. and J. Spillane. Neurological examination in clinical practice. 5th Ed. London: Blackwell Scientific Publications, 1989, pp. 347–401.

Cited Here...

3. Coert, J. and A. Dellon. Clinical implications of the surgical anatomy of the sural nerve. Plast. Reconstr. Surg. 94:850–855, 1994.
<u>Cited Here...</u>
<u>A</u> Deese, L and D. Bayter. Compressive neuropathies of the lower.

4. Deese, J. and D. Baxter. Compressive neuropathies of the lower extremity. J. Musculoskel. Med. 5:678–695, 1988. <u>Cited Here...</u>

5. Donell, S. and D. Barrett. Entrapment neuropathies: Part 2 Lower limb. Br. J. Hosp. Med. 46:99–101, 1991.

Cited Here...

6. Dumitru, D. and R. Windsor. Subsartorial entrapment of the saphenous nerve of a competitive female bodybuilder. Physician Sportsmed. 17:116–125, 1989.

Cited Here...

7. Jordan, B., P. Tsaris, and R. Warren (Eds.). *Sports Neurology*, 2nd Ed. Philadelphia: Lippincott-Raven, 1998, pp. 728–729. <u>Cited Here...</u>

8. Kashani, S., A. Moon, and W. Gaunt. Tibial nerve entrapment by a Baker's cyst. Arch. Phys. Med. Rehabil. 66:49–51, 1985.

Cited Here...

9. Kopell, H. and W. Thompson. Peripheral Entrapment Neuropathies. Baltimore: Williams & Wilkins, 1963, pp. 79–117.

Cited Here...

10. Kopell, H. P. and W. Thompson. Peripheral nerve entrapments of the lower extremity. N. Engl. J. Med. 266:16–19, 1962.

Cited Here...

11. Leach, R., M. Purnell, and A. Saito. Peroneal nerve entrapment in runners. Am. J. Sports Med. 17:287–291, 1989.

Cited Here...

12. Levin, K., J. Stevens, and J. Daube. Superficial peroneal nerve conduction studies for electromyographic diagnosis. Muscle Nerve 9:322–326, 1986.

Cited Here...

13. Luerssen, T., R. Campbell, R. Defalque, and R. Worth. Neurosurgery 13:238–241, 1983.

Cited Here...

14. Moller, B. and S. Kadin. Entrapment of the common peroneal nerve. Am. J. Sports Med. 15:90–91, 1987.

Cited Here...

15. Mumenthaler, M. and H. Schliack. Peripheral Nerve Lesions:Diagnosis and Therapy. New York: Thieme, 1991, pp. 345–76.

Cited Here...

16. Nakano, K. The entrapment neuropathies. Muscle Nerve 1:264–279, 1978.

#### Cited Here...

17. Pecina, M., J. Krmpotic-Nemanic, and J. Markiewitz. Tunnel Syndromes: Peripheral Nerve Compression Syndromes. 2nd Ed. Boca Raton, FL: CRC Press, 1997, pp. 1–218.

Cited Here...

18. Pringle, R., K. Protheroe, and S. Mukherjee. Entrapment neuropathy of the sural nerve. J. Bone Joint Surg. 56B:465–468, 1974.

Cited Here...

19. Schon, L. Nerve entrapment, neuropathy, and nerve dysfunction in athletes. Orthop. Clin. North Am. 25:47–59, 1994.

Cited Here...

20. Schon, L. and D. Baxter. Neuropathies of the foot and ankle in athletes. Clin. Sports Med. 9:489–509, 1990.

Cited Here...

21. Schuchmann, J. Isolated sural neuropathy: report of two cases. Arch. Phys. Med. Rehabil. 61:313–329, 1980.

Cited Here...

22. Senegor, M. Iatrogenic saphenous neuralgia. Neurosurgery 28:295–298, 1991.

Cited Here...

23. Stack, R., A. Bianco, and C. McCarty. Compression of the common peroneal nerve by ganglion cysts. J. Bone Joint Surg. 47A:773–778, 1965. Cited Here...

24. Stewart, J. Focal Peripheral Neuropathies, 2nd Ed. New York: Raven Press, 1993, pp. 347–401.

Cited Here...

25. Styf, J. Chronic exercise-induced pain in the anterior aspect of the lower leg: an overview of the diagnosis. Sports Med. 7:331–339, 1989. <u>Cited Here...</u>

26. Styf, J. Entrapment of the superficial peroneal nerve: diagnosis and results of decompression. J. Bone Joint Surg. 71B:131–135, 1989. <u>Cited Here...</u>

27. Styf, J. and I. Korner. Chronic anterior compartment syndrome of the leg. J. Bone Joint Surg. 68A:1338–1347, 1986.

Cited Here...

28. Tardif, G. Nerve injuries: testing and treatment strategies. Physician Sportsmed. 23:61–72, 1995.

#### Cited Here...

29. Wartenberg, R. Digitalia paraestetica and gonialgia paraesthetica. Neurology 4:106–116, 1953.

Cited Here...

30. Wilbourne, A. Electrodiagnostic testing of neurologic injuries in athletes. Clin. Sports Med. 9:229–245, 1990.

Cited Here...

31. Williams, D. A study of the human medial peroneal nerve, a new name proposed for the peroneal anastamotic nerve. Anat. Rec. 118:415–421, 1954.

Cited Here...

32. Worth, R. M., D. B. Kettelkamp, R. J. Defalque and K. U. Duane. Saphenous nerve entrapment: a cause of medial knee pain. Am. J. Sports Med. 12:80–81, 1984.

Cited Here...

# Keywords:

NERVE ENTRAPMENT SYNDROME; SAPHENOUS NERVE; PERONEAL NERVE; SURAL NERVE; TIBIAL NERVE

©2000The American College of Sports Medicine