Tarsal Tunnel Syndrome 24

Patients with pain and numbness in the foot often are referred to the electromyography (EMG) laboratory for evaluation of possible tarsal tunnel syndrome (TTS). TTS results from entrapment of the distal tibial nerve under the flexor retinaculum at the medial ankle. Superficially, it might seem that tibial nerve entrapment under the flexor retinaculum at the ankle would be analogous to median nerve entrapment under the flexor retinaculum at the wrist [i.e., carpal tunnel syndrome (CTS)]. However, in contrast to CTS, which is very common, TTS is exceptionally rare. Although electrophysiology can be useful in demonstrating focal slowing at the tarsal tunnel in those rare cases of true TTS, every electromyographer should be aware that significant technical difficulties are often encountered when studying the distal tibial nerve and the muscles it innervates, especially in older patients.

ANATOMY

As the tibial nerve descends distal to the medial malleolus, it runs beneath the flexor retinaculum on the medial side of the ankle, through the tarsal tunnel (Figure 24–1). The tarsal tunnel is a fibro-osseous tunnel below the medial malleolus with a bony floor and a roof formed by the flexor retinaculum. In addition to the tibial nerve, the tibial artery and tendons of the flexor hallucis longus, flexor digitorum longus, and tibialis posterior pass through the tarsal tunnel. The distal tibial nerve then divides into three or four branches. One or two branches (*medial and lateral calcaneal sensory nerves*) are purely sensory and provide sensation to the heel of the sole (Figure 24–2). The other two branches, the *medial and lateral plantar nerves*, contain both motor and sensory fibers that supply the medial and lateral sole of the foot, respectively. Typically, the medial plantar nerve supplies the first three and a half toes (including the great toe), whereas the lateral plantar nerve supplies the little toe and the lateral fourth toe. Both plantar nerves innervate the intrinsic muscles of the foot. The muscles that are most accessible to study using needle EMG are the abductor hallucis brevis (AHB), flexor hallucis brevis (FHB), and flexor digitorum brevis (FDB) for the medial plantar nerve, and the abductor digiti quinti pedis (ADQP) for the lateral plantar nerve.

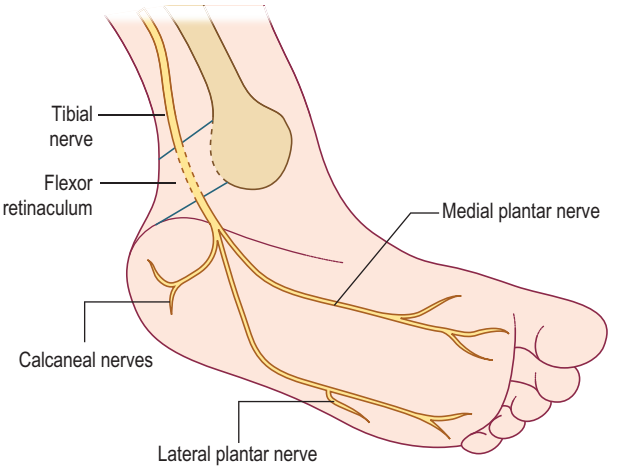

FIGURE 24–1 Anatomy of the distal tibial nerve at the ankle and sole of the foot. The distal tibial nerve runs posterior to the medial malleolus under the flexor retinaculum on the medial side of the ankle (i.e., through the tarsal tunnel), before dividing into the medial plantar, lateral plantar, and calcaneal nerves. The calcaneal nerves are purely sensory and provide sensation to the heel of the sole. The medial and lateral plantar branches both contain motor fibers to supply the intrinsic muscles of the foot and sensory fibers to supply the medial and lateral sole, respectively.

CLINICAL

The most frequent symptom in patients with TTS is perimalleolar pain. Ankle and sole pain often is described as burning and often is worse with weight bearing or at night. Paresthesias and sensory loss involving the sole of the foot may occur due to compression of the plantar or calcaneal nerves (Figure 24–3). There are few other reliable clinical signs. Intrinsic foot muscle atrophy may occur but is not specific to TTS. For example, atrophy of intrinsic foot muscles may occur in L5–S1 radiculopathy, proximal tibial neuropathy, or polyneuropathy. It is extremely difficult to assess strength of the intrinsic foot musculature, because most of the important toe and ankle functions are subserved by the long extensors and flexors in the lower leg, which are innervated by the proximal peroneal and tibial nerves. Finally, many consider a Tinel's sign over the tarsal tunnel to be suggestive of TTS. Unfortunately, like Tinel's

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signs elsewhere, this is a nonspecific sign and may occur in some normal subjects. Significantly, the ankle tendon reflex, which is mediated by the tibial nerve proximal to the tarsal tunnel, is normal in TTS, as is sensation over the lateral foot (sural nerve) and the dorsum of the foot (superficial peroneal nerve).

ETIOLOGY

The incidence of TTS is widely debated. Some podiatrists believe that TTS is rather common, whereas most neurologists and electrophysiologists believe that it is quite rare. Lesions of the medial and lateral plantar nerves most often occur as a result of trauma (including sprain and fracture) or occasionally from degenerative bone or connective tissue disorders. Rare cases of TTS are caused by varicosities or other unusual mass lesions (e.g., lipomas, ganglions, cysts, exostoses). TTS caused by hypertrophy of the flexor retinaculum from repetitive use (akin to CTS) is unusual. One or more of the three nerve branches (calcaneal, medial plantar, and lateral plantar) may be involved.

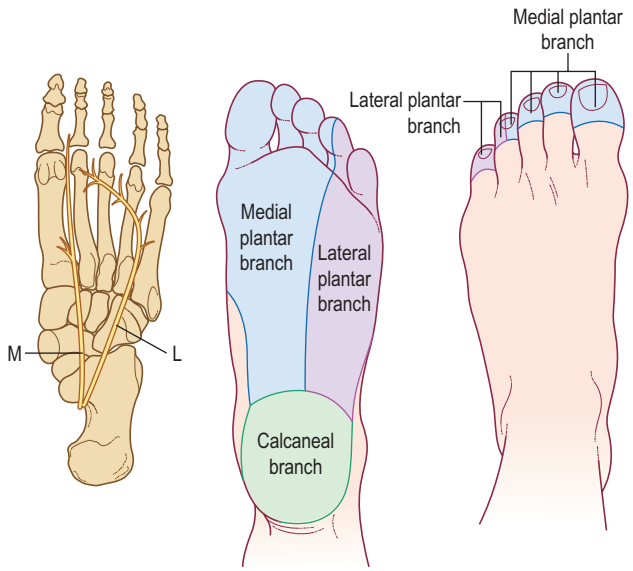

FIGURE 24–2 Tibial sensory innervation of the foot. The distal tibial nerve supplies sensation to the sole of the foot via the medial plantar, lateral plantar, and calcaneal sensory nerves.
(Adapted from Omer, G.E., Spinner, M., 1980. Management of peripheral nerve problems. WB Saunders, Philadelphia.)

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of TTS includes local orthopedic problems of the foot (especially tendinitis and fasciitis), proximal tibial neuropathy, and, especially early on, mild polyneuropathy. S1 radiculopathy or lumbosacral plexopathy may cause sensory loss over the sole, but neither is typically associated with local foot pain. It is not unusual for patients who first present with polyneuropathy to be misdiagnosed with TTS. Most patients studied in our laboratory referred for possible TTS had either a normal electrophysiologic examination (and may have had a local orthopedic problem) or were found to have a mild distal polyneuropathy.

ELECTROPHYSIOLOGIC EVALUATION

Nerve Conduction Studies

Evaluation of suspected TTS is greatly simplified if one side is symptomatic and the other side is normal. This situation allows for side-to-side comparison studies (Box 24–1). The important nerve conduction studies to perform include bilateral tibial distal motor latencies to both the AHB and ADQP, for the medial and lateral plantar nerves, respectively, stimulating the tibial nerve proximal to the tarsal tunnel at the medial malleolus (Figure 24–4). Compound

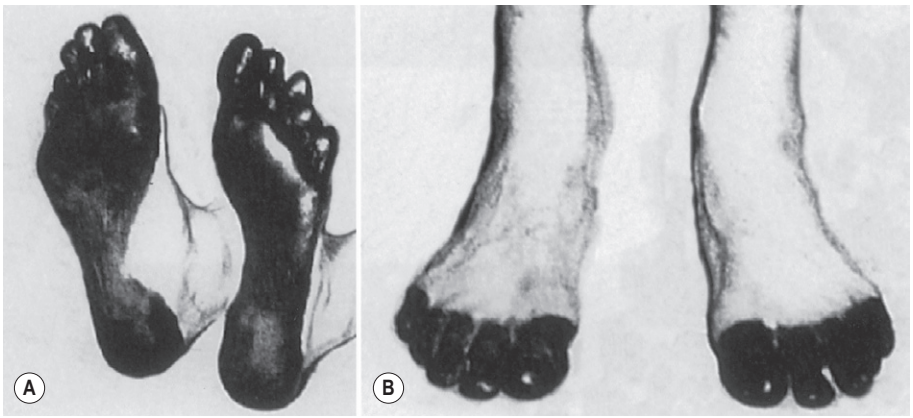

FIGURE 24–3 Sensory loss in tarsal tunnel syndrome. **A:** The first case of tarsal tunnel syndrome was reported by Captain Keck in an army recruit who developed pain in the feet and anesthesia in the distribution of the distal tibial nerve. **B:** Black shading indicates areas of anesthesia from the original case report.
(From Keck, C., 1962. The tarsal tunnel syndrome. J Bone Joint Surg 44, 180.)

Box 24–1. Recommended Nerve Conduction Study Protocol for Tarsal Tunnel Syndrome

Routine studies:

1. Distal tibial motor (medial and lateral plantar) studies, stimulating tibial nerve at medial malleolus and recording abductor hallucis brevis (medial plantar) and abductor digiti quinti pedis (lateral plantar). Comparison with contralateral side is required
2. Routine tibial motor study, recording abductor hallucis brevis, stimulating medial ankle and popliteal fossa
3. Routine peroneal motor study, recording extensor digitorum brevis and stimulating ankle, below fibular neck, and lateral popliteal fossa
4. Medial and lateral plantar mixed or sensory studies (plantar mixed and sensory responses usually require averaging several potentials). For mixed studies, stimulate medial sole and record medial ankle (medial plantar mixed); stimulate lateral sole and record medial ankle (lateral plantar mixed). For sensory studies, stimulate great toe and record medial ankle (medial plantar sensory); stimulate little toe and record medial ankle (lateral plantar sensory). Comparison with the contralateral side is required, using identical distances between the stimulating and recording sites
5. Sural sensory response, stimulating posterior calf, recording posterior ankle
6. Tibial and peroneal F responses
7. H reflexes, bilateral studies (may be abnormal in S1 radiculopathy or polyneuropathy but not in tarsal tunnel syndrome)

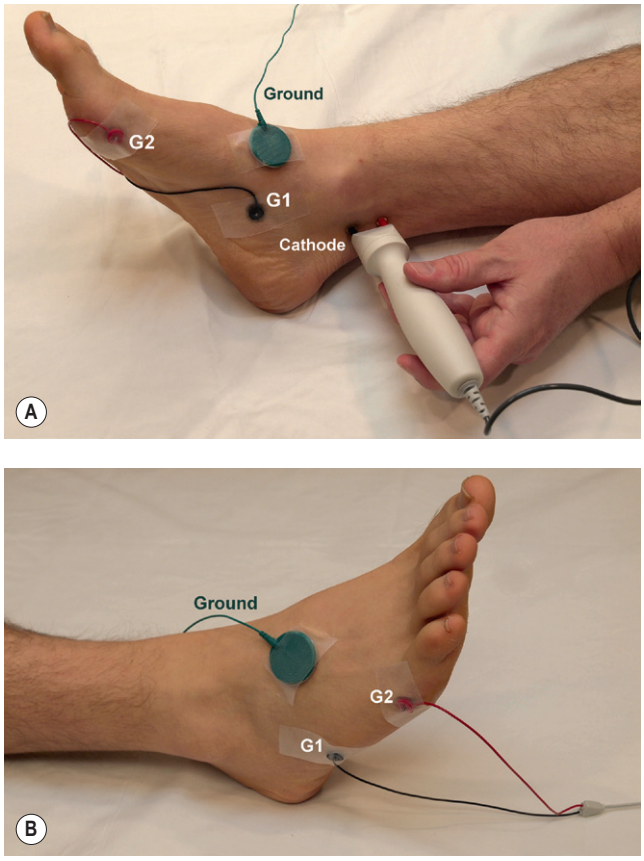

FIGURE 24–4 Distal tibial motor studies. The medial and lateral plantar distal motor latencies can be measured by recording the abductor hallucis brevis (**A**) and abductor digiti quinti pedis (**B**), respectively, and stimulating the tibial nerve behind the medial malleolus.

muscle action potential (CMAP) amplitudes and distal latencies are compared from side to side. Theoretically, if there is demyelination across the tarsal tunnel, the distal latencies on the involved side should be markedly prolonged. In axonal loss lesions, the CMAP amplitudes will be reduced, and the latencies will be normal or only slightly prolonged.

Surface sensory and mixed nerve studies are difficult to perform, even in normal healthy subjects, but they increase the sensitivity of making the electrodiagnosis of TTS. Orthodromic surface sensory studies can be performed stimulating the great and little toes (medial and lateral plantar sensory nerves, respectively) and recording over the tibial nerve at the medial ankle proximal to the tarsal tunnel. The potentials are usually extremely small in amplitude, making it necessary to average many potentials. Antidromic surface sensory studies also can be performed, but they have similar technical limitations. Surface recording of the mixed plantar nerves is slightly easier (Figure 24–5). Both the medial and lateral plantar mixed nerves can be stimulated in the sole, recording over the tibial nerve at the medial ankle (proximal to the tarsal tunnel). Averaging is still required to measure these small potentials, and in older individuals they may be absent. *Often, medial and lateral plantar sensory and mixed nerve potentials are unobtainable even in normal subjects*. Consequently, an absent or low-amplitude potential should not be considered abnormal unless a clear side-to-side difference is found using identical distances between the stimulating and recording sites. No diagnostic significance should be attributed to bilaterally absent plantar mixed or sensory nerve responses, especially in middle-aged or older individuals. It is important to emphasize that the plantar mixed and sensory nerves are the most distal nerves in the lower extremities. As such, their conduction velocities normally are slower than those of more proximal nerves and are more susceptible to the effects of temperature and cooling.

In addition to bilateral plantar motor, sensory, and mixed nerve studies, further nerve conductions should be performed routinely, especially to exclude a polyneuropathy. Routine peroneal and tibial motor studies and their respective F responses should be obtained along with the sural sensory response. If the sural sensory response is abnormal, any abnormalities in the plantar nerves are likely secondary to either a polyneuropathy or, less often, a sciatic or lumbosacral plexus lesion. In some situations, assessment of bilateral H reflexes can yield useful information. H reflexes are normal in TTS but may be abnormal in polyneuropathy, proximal tibial neuropathy, sciatic and lumbosacral plexus lesions, and S1 radiculopathy, all of which clinically may cause sensory abnormalities over the sole of the foot.

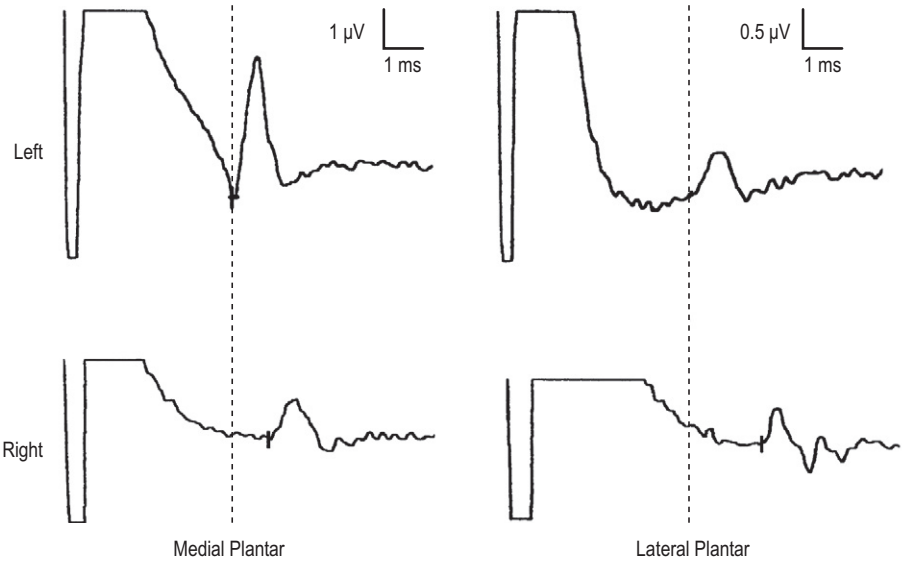

FIGURE 24–5 Medial and lateral plantar mixed nerve responses: value of comparing symptomatic side to asymptomatic contralateral side. The medial and lateral sole are stimulated while recording over the tibial nerve at the medial ankle. Sensory and mixed nerve potentials are very low in amplitude and must be averaged to be discerned from background noise. Although the right medial plantar mixed nerve potential is two to three times lower in amplitude than the left, the absolute difference is only 2 to 3 µV. However, the right medial and lateral plantar mixed nerve potentials are significantly prolonged in comparison to the left.

Box 24–2. Recommended Electromyographic Protocol for Tarsal Tunnel Syndrome

Routine muscles:

1. Abductor hallucis brevis and abductor digiti quinti pedis (must be compared with the contralateral side)
2. At least two distal tibial-innervated muscles proximal to the tarsal tunnel (e.g., medial gastrocnemius, soleus, tibialis posterior, flexor digitorum longus)
3. At least one distal peroneal-innervated muscle in the lower leg (tibialis anterior, extensor hallucis longus)

Special considerations:

- If any muscle proximal to the tarsal tunnel is abnormal, additional muscles must be sampled to determine whether the lesion represents a more proximal tibial or sciatic neuropathy, lumbosacral plexopathy, radiculopathy, or polyneuropathy.
- From a practical point of view, it is nearly impossible to diagnose tarsal tunnel syndrome in the presence of a polyneuropathy.
- Examination of intrinsic foot muscles often is painful for patients and these muscles are difficult to activate. Increased insertional activity and occasionally fibrillation potentials, associated with large, long duration motor unit action potentials, are frequently found in normal subjects without symptoms. Interpreting the electromyographic findings in an intrinsic foot muscle as abnormal requires that (1) the abnormalities be fairly marked or (2) the contralateral asymptomatic muscle is distinctly different on EMG from the symptomatic side.

Electromyographic Approach

EMG often is quite problematic in the assessment of TTS (Box 24–2). An EMG study of the intrinsic foot muscles is fraught with problems. First is the limited ability of patients to tolerate the examination. The sole is quite sensitive, and placement of the EMG needle into the intrinsic foot muscles is painful for most patients. Second, activating these muscles is difficult; therefore, it frequently is difficult to assess a sufficient number of motor unit action potentials (MUAPs). Finally, the interpretation of what is normal may be difficult. Intrinsic foot muscles commonly show increased insertional activity and occasionally fibrillation potentials associated with large, long-duration MUAPs, as one would expect in a neurogenic lesion. *Such findings are not unusual in normal subjects without symptoms*, however, and are thought to be due to everyday wear and tear on the feet. Therefore, interpretation of these abnormalities is problematic. Interpreting the EMG findings in an intrinsic foot muscle as abnormal requires that (1) the abnormalities be fairly marked or (2) the contralateral asymptomatic muscle is distinctly different on EMG from the symptomatic side.

In addition to the plantar-innervated intrinsic foot muscles (AHB, FHB, and ADQP), tibial- and peroneal-innervated muscles in the lower leg should be sampled to exclude a more proximal lesion or polyneuropathy. If abnormalities are found in these muscles, a more extensive evaluation should be performed to sort out whether the changes are due to a proximal tibial neuropathy, sciatic neuropathy, lumbosacral plexopathy, radiculopathy, or polyneuropathy.

EXAMPLE CASE

Case 24–1

History and Physical Examination

A 41-year-old woman was referred for persistent foot pain after an ankle fracture. Four months previously, she sustained a non-displaced fracture of the right ankle and wore a cast for 6 weeks. She continued to experience ankle pain, which worsened with walking.

Examination showed tenderness over the medial ankle. There was mild atrophy of the right intrinsic foot muscles. Toe and ankle flexion and extension were normal. Sensation was intact over the lateral foot and the dorsum of

CASE 24–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = µV			Latency (ms)			Conduction Velocity (m/s)			F Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Tibial (m)	Ankle	AHB	6.5	10.6	≥4	5.3	4.8	≤5.8				54	51	≤56
	Popliteal Fossa	AHB	5.0	8.5		12.1	11.1		44	48	≥41			
Tibial (m)	Ankle	ADQP	4.2	5.3	≥3	5.8	5.2	≤6.3						
Peroneal (m)	Ankle	EDB	4.1		≥2	4.7		≤6.5				55		≤56
	Below fibula	EDB	4.0			10.7			50		≥44			
	Lateral popliteal fossa	EDB	3.9			12.9			51		≥44			
Sural (s)	Calf	Posterior ankle	17		≥6	3.0		≤4.4	52		≥40			
Peroneal (s)	Lateral calf	Ankle	27		≥6	3.2		≤4.4	50		≥40			
Medial plantar (mixed study)	Medial sole	Medial ankle	1	8	≥3	4.1	3.3	≤3.7						
Lateral plantar (mixed study)	Lateral sole	Medial ankle	0.5	4	≥3	4.4	3.5	≤3.7						

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; AHB = abductor hallucis brevis; ADQP = abductor digiti quinti pedis; EDB = extensor digitorum brevis.

Note: All sensory and mixed latencies are peak latencies. All sensory and mixed-nerve conductions are calculated using onset latencies. The reported F wave latency represents the minimum F wave latency.

CASE 24–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right AHB	↑	+1	0	Poor	NL	+1	+1	NL
Right ADQP	↑	0	0	Poor	NL	NL/+1	NL	NL
Left AHB	↑	+1	0	Poor	NL	NL/+1	NL/+1	NL
Left ADQP	NL	0	0	Poor	NL	NL/+1	NL	NL
Right medial gastrocnemius	NL	0	0	Fair	NL	NL	NL	NL
Right tibialis posterior	NL	0	0	NL	NL	NL	NL	NL
Right tibialis anterior	NL	0	0	NL	NL	NL	NL	NL

↑ = increased; NL = normal; AHB = abductor hallucis brevis; ADQP = abductor digiti quinti pedis.

the foot. Hypesthesia to pinprick and temperature was present over the sole of the right foot. The deep tendon reflexes, including the ankle reflexes, were present and symmetric.

Summary

There is little in this patient's history to suggest a nerve injury. Persistent pain following trauma and an ankle fracture could very well be of local orthopedic origin. However, the neurologic examination reveals mild abnormalities. There is mild atrophy of the intrinsic foot muscles on the right, although, with the history of recent casting, this finding could be due to disuse alone. The sensory examination shows normal sensation over the lateral foot and the dorsum of the foot, but pinprick and temperature sensation are decreased over the sole of the right foot. This pattern of abnormal sensation over the sole of the foot with complete sparing of the lateral foot and dorsum of the foot would be unusual for a typical polyneuropathy, in which all the distal fibers are affected first. This finding raises the possibility of a lesion of the plantar nerves. Numbness over the sole may be seen in disorders other than polyneuropathy, including proximal tibial neuropathy, sciatic neuropathy, lumbosacral plexopathy, or lesions of the S1–S2 nerve roots. The bilaterally intact ankle reflex is a helpful piece of information. For example, a lesion of the S1 nerve root, lumbosacral plexus, sciatic nerve, or more proximal tibial nerve may

well result in an abnormal ankle reflex on the symptomatic side.

When reviewing the nerve conduction studies, particular attention must be paid to whether the results correlate with the clinical examination. First, tibial motor studies recording the AHB muscle are performed bilaterally; the results are normal. There is a slight asymmetry, i.e., the amplitude on the symptomatic right side is slightly smaller with a slightly longer distal latency, but the differences would not be considered significant. Note that routine tibial motor studies recording the AHB muscle check only the medial plantar nerve. Tibial motor studies recording the ADQP muscle are next performed bilaterally to assess the lateral plantar nerves. The amplitudes and latencies are normal, although again there is a slight asymmetry, with the right side slightly smaller in amplitude and longer in latency than the left side. Peroneal motor studies are performed next on the symptomatic side; they are normal.

After the motor studies are completed, the sensory studies are performed, including the sural and then the superficial peroneal sensory responses on the right side. Both are entirely normal. The normal sural sensory response correlates with the normal sensation over the lateral foot, and the normal superficial peroneal sensory response likewise correlates well with the normal sensation over the dorsum of the foot. As for the plantar responses, when the medial and lateral plantar mixed nerves are recorded, only small-amplitude responses are obtained from the right side. This finding alone would not necessarily be considered abnormal, because plantar mixed and sensory responses often are very small or difficult to obtain in normal subjects. When these responses are compared with the asymptomatic contralateral side, however, the amplitudes are clearly and significantly asymmetric (>50% difference from side to side). In addition, the latencies are somewhat prolonged on the right side compared to the left. The degree of prolongation is not in the unequivocally demyelinating range and may be consistent with axonal loss and dropout of the fastest-conducting fibers.

When the nerve conduction studies are completed, there is strong evidence for a lesion affecting the distal tibial nerve and involving the medial and lateral plantar nerves. Polyneuropathy seems less likely, given the intact and robust sural and superficial peroneal sensory responses, and the asymmetry of the plantar mixed nerve studies from side to side. However, the reduced amplitude of the medial and lateral plantar mixed nerve responses and the borderline prolonged latencies are well within the range that would indicate axonal loss. Thus, there still is the possibility of a proximal tibial neuropathy in the calf. However, the fact that the sural sensory response, which is derived proximally from the tibial and peroneal nerves in the popliteal fossa, is normal argues against a proximal lesion of the tibial nerve. The needle EMG examination should be helpful in confirming the location of the lesion; particular attention should be paid to tibial muscles in the calf above the level of the tarsal tunnel.

Moving on to the needle EMG findings, fibrillation potentials are present in the right AHB muscle. There is poor activation of MUAPs, which is not unusual even in normal subjects. The few MUAPs seen appear to be of slightly increased duration and amplitude. These findings usually are associated with neuropathic lesions. However, one must always be cautious in assessing the intrinsic foot muscles. Normal subjects without any complaints may have mild active denervation or reinnervation (or both) in the intrinsic foot muscles. Indeed, when the contralateral AHB muscle is checked, there are also sparse fibrillation potentials with borderline large and long MUAPs. Therefore, although we might have initially interpreted the right AHB as abnormal, after examining the contralateral side, we determine that the findings on the right side are of dubious significance. A similar lack of asymmetry is seen in the ADQP muscles; both sides are slightly abnormal. Next, two tibial-innervated muscles that arise above the tarsal tunnel are sampled (the medial gastrocnemius and the tibialis posterior), and both are entirely normal. Finally, the tibialis anterior muscle is sampled. This is a peroneal-innervated muscle, and it is completely normal.

At this time, we are ready to formulate our electrophysiologic impression.

IMPRESSION: *The electrophysiologic findings are consistent with a distal tibial neuropathy affecting the medial and lateral plantar nerves.*

An important question should be addressed at this point.

How Does One Localize the Lesion to the Plantar Nerves?

The electrophysiologic abnormalities are limited to the distal tibial nerve, that is, the medial and lateral plantar nerves. Both plantar mixed nerve responses are low compared with the contralateral side, with mild prolongation of peak latency. This type of abnormality can be seen in TTS, but it also can be seen in proximal tibial neuropathy, sciatic neuropathy, or lumbosacral plexopathy. The fact that the EMG study does not demonstrate any abnormality of peroneal- or tibial-innervated muscles proximal to the tarsal tunnel argues against a lesion of the proximal tibial nerve, sciatic nerve, or lumbosacral plexus. Note that the asymmetric abnormalities in the mixed nerve responses seen in this case would not be expected in a sacral radiculopathy, because sensory potentials (which make up the majority of mixed nerve potentials) are spared in lesions proximal to the dorsal root ganglion. The clinical findings of intact sensation over the lateral and dorsal foot also argue strongly against a polyneuropathy, sciatic neuropathy, or lumbosacral plexopathy. These findings are later substantiated on the nerve conduction studies, which show normal sural and superficial peroneal

sensory responses. Therefore, although the electrophysiology fails to definitively localize the lesion, the weight of the evidence favors a lesion of the distal tibial nerve at the ankle (medial and lateral plantar nerves), especially considering the site of the trauma and the site of the persistent pain.

Suggested Readings

Almeida, D.F., Scremin, L., Zuniga, S.F., et al., 2010. Focal conduction block in a case of tarsal tunnel syndrome. Muscle Nerve 42, 452–455.

Cimino, W.R., 1990. Tarsal tunnel syndrome: review of the literature. Foot Ankle 11, 47.

Dawson, D.M., Hallett, M., Millender, L.H., 1990. Entrapment neuropathies, second ed. Little, Brown, Boston.

Felsenthal, G., Butler, D.H., Shear, M.S., 1992. Across tarsal tunnel motor nerve conduction technique. Arch Phys Med Rehabil 73, 64–69.

Keck, C., 1962. The tarsal tunnel syndrome. J Bone Joint Surg 44, 180.

Oh, S.J., Arnold, T.W., Park, K.H., et al., 1991. Electrophysiological improvement in tarsal tunnel syndrome following decompression surgery. Muscle Nerve 14, 407.

Oh, S.J., Meyer, R.D., 1999. Entrapment neuropathies of the tibial (posterior tibial) nerve. Neurol Clin 17, 593–615.

Oh, S.J., Sarala, P.K., Kuba, T., et al., 1979. Tarsal tunnel syndrome: electrophysiological study. Ann Neurol 5, 327.

Patel, A.T., Gaines, K., Malamut, R., et al., 2005. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence-based review. Muscle Nerve 32, 236–240.