

# REVIEW

## Clinical Neurophysiology in the Diagnosis of Peroneal Nerve Palsy

Yoshihisa Masakado, Michiyuki Kawakami, Kanjiro Suzuki, Leon Abe, Tetsuo Ota, and Akio Kimura

*Keio University Tsukigase Rehabilitation Center, Shizuoka, Japan*

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**Abstract:** Peroneal neuropathy is one of the common focal mononeuropathies in the lower extremities occurring in both adults and children. Foot drop due to weakness of ankle dorsiflexion is the most common presentation of a peroneal neuropathy. It may also result from other causes involving the upper or lower motor neurons. Disorders that must be distinguished from peroneal neuropathy include sciatic mononeuropathy, lumbosacral plexopathy, motor neuron disease, polyneuropathy, and an L5 radiculopathy. To establish a diagnosis, electrodiagnostic studies have been used to localize the level of the abnormality and to establish prognosis. The most common site of injury is the fibular head, but focal neuropathies have also been reported at the level of the calf, ankle, and foot. In this article, we overviewed the peroneal nerve palsy, and its diagnosis by neurophysiologic evaluation, conduction study and needle EMG. The neurophysiologic information gives us the underlying pathophysiology and its prognosis. Therefore the neurophysiologic evaluation must be performed not only for the differential diagnosis, but also for planning the treatment strategy. (Keio J Med 57 (2) : 84–89, June 2008)

**Key words:** peroneal neuropathy, neurophysiological evaluation, motor conduction study, sensory conduction study, needle EMG

### Introduction

Peroneal nerve palsy is the most common entrapment neuropathy in the lower extremity. Most often, peroneal nerve palsy occurs at the fibular neck, where the nerve is superficial and vulnerable to injury. Patients usually present with a foot drop and sensory disturbance over the lateral calf and the dorsum of the foot. However patients with sciatic neuropathy, lumbosacral plexopathy, or L5 radiculopathy may reveal similar clinical symptoms. In addition to establishing a diagnosis, electrodiagnostic (EDX) studies have been used by some authors to localize the level of the abnormality and to establish prognosis.<sup>1,2</sup> The most common site of injury is the fibular head as mentioned above, but focal neuropathies have also been reported at the level of the calf, ankle, and foot.<sup>3</sup> In addition, electrophysiology can localize the level of the nerve palsy, reveal the underlying pathology, and establish the prognosis. This review addresses the importance and limitation of EDX techniques in the evaluation of peroneal neuropathy.<sup>4–6</sup>

### Anatomy and its Disorders

The peroneal nerve is derived from the L4-S1 nerve roots, which travel from the lumbosacral plexus and eventually the sciatic nerve. Within the sciatic nerve, the fibers forming the peroneal nerve run separately from those that become the tibial nerve. In the posterior thigh, the peroneal fibers within the sciatic nerve innervate the short head of the biceps femoris, the only muscle that peroneal fibers innervate above the level of the fibular neck. More distally, the sciatic nerve bifurcates above the popliteal fossa into the common peroneal and tibial nerves. The common peroneal nerve first gives rise to the lateral cutaneous nerve of the knee, which supplies sensation to the lateral knee before winding around the neck of the fibula. Then it passes through the attachment of the superficial head of the peroneus longus muscle, and divides into the deep and superficial peroneal nerves. The former innervates the peroneus tertius muscle and the dorsiflexors of the ankle and toes, including the tibia-

**Table 1** Causes of peroneal neuropathy

1. External compression (casts, immobilization)
2. Direct trauma (fracture)
3. Traction injuries (forcible ankle inversion)
4. Masses (ganglion, tumor)
5. Entrapment (fibular tunnel)
6. Vascular conditions
7. Diabetes mellitus
8. Leprosy
9. Idiopathic

lis anterior (TA) muscle, extensor digitorum longus and extensor hallucis longus, and extensor digitorum brevis (EDB). It supplies sensation to the web space between the first and second toes. The superficial peroneal nerve innervates the ankle evertors (peroneus longus and brevis) and then supplies sensation to the mid and lower lateral calf. It passes over the dorsum of the foot, supplying sensation to the dorsum of the foot and to the dorsal medial three or four toes. Although the EDB is usually innervated only by the deep peroneal nerve, occasionally (in about one third of the population)<sup>4,6</sup> it derives additional innervation from an accessory peroneal nerve, a branch of the superficial peroneal, which curves around the lateral malleolus and usually supplies the lateral portion of the EDB. When the peroneal nerve is stimulated at the ankle, the response obtained has a lower amplitude than that obtained from the knee. This difference is due to the fact that at the ankle only the deep peroneal nerve is stimulated but at the knee both deep and superficial branches (the latter with its accessory peroneal branch) are stimulated. If the accessory peroneal nerve is stimulated behind the lateral malleolus, a response is obtained from the EDB, with an amplitude that is equal to the difference between the amplitudes of the responses obtained from knee and ankle stimulation.<sup>5</sup>

The common peroneal nerves are vulnerable to external compression in its course around the head and fibula (Table 1). Acute peroneal neuropathy often follows trauma or compression from prolonged immobilization. This occurs often postoperatively in patients who have received anesthesia or sedation. Slow progressive lesions often suggest a mass, such as a ganglion or nerve sheath tumor.

Several circumstances predispose a subject to peroneal neuropathy. Habitual leg crossing may repetitively injure the peroneal nerve at the fibular neck, where it is quite superficial. Similarly, repetitive stretching from squatting has also been associated with peroneal neuropathy.

It has been reported that traction injuries are the most common (usually during sport) followed by penetrating and iatrogenic injuries. On the other hand, traction is presumed to be the mechanism of injury to the superficial peroneal nerve in an inversion ankle sprain, but it is not

known whether the amount of strain caused by nerve traction is sufficient to cause nerve injury. Peripheral nerves may be injured when they are stretched beyond their physiologic limits. A previous report has suggested that the superficial peroneal nerve may be at risk during actual ankle sprain.<sup>7</sup> In 2000, Garozzo *et al.* reported 5 cases of common peroneal nerve palsy,<sup>8</sup> which was associated with inversion sprains of the ankle. Common peroneal nerve palsy associated with inversion sprains of the ankle has been mentioned in the literature on a case presentation basis only and is consequently regarded to be a rare complication. The etiologic mechanisms responsible for the nerve impairment have not been clearly identified. Some of the cases reported in the literature eventually required surgical treatment, which was always followed by complete recovery. In Garozzo's report they presented 5 additional cases of this peculiar neuropathy: all the patients were surgically treated, but the author observed neurological recovery in only 2 cases.

### Clinical Evaluation

Peroneal nerve lesions at the region of the knee or distal thigh usually result in patient reports of altered ambulation secondary to paretic or paralyzed ankle dorsiflexors. The loss of sensation in the cutaneous distribution of the superficial and deep peroneal nerves may be noted, but the ankle dorsiflexion weakness is most important. Pain is often related to the specific cause of a compromised common peroneal nerve. For example, a nerve compromised secondary to traumatic injury from blunt trauma will likely result in pain secondary to soft tissue swelling and inflammation, whereas chronic compression secondary to habitual leg-crossing is often not related with any pain. Tapping of the nerve at the fibular head may produce pain and tingling (Tinel sign) in the sensory distribution of the peroneal nerve.

Examination often reveals a variable pattern of weakness, with the EDB muscle being most profoundly affected. Ankle and toe dorsiflexion can be significantly affected. Dorsiflexion is best tested by having the patient place the ankle in the neutral position and then dorsiflex the foot and invert it to optimally test the TA muscle. Often, ankle eversion is normal, as patients can have relative sparing of these muscles. In a pure common peroneal neuropathy, plantar flexion should be spared. Most importantly, ankle inversion is spared, mediated by the tibialis posterior (L5, sciatic nerve, tibial nerve innervation). If the ankle is tested in a dropped position, ankle inversion may appear weak. To test ankle inversion in a patient with a footdrop, the ankle should be passively dorsiflexed to avoid the mistaken impression that the tibialis posterior is weak.<sup>4</sup>

Observation of the patient's gait is useful in diagnosing ankle dorsiflexion weakness. The patient often displays a

steppage gait pattern in which the affected foot is lifted excessively from the ground during the swing phase of ambulation in order to clear the foot. This results in excessive hip and knee flexion, and the appearance is as if the patient is stepping over an object in his or her path. In addition, a foot slap may be heard on foot strike, as the ankle dorsiflexors cannot provide a controlled descent of the foot toward the floor. The patient might also stumble when walking, secondary to the toes on the affected side dragging or catching on the floor during the swing-through phase of ambulation.

In fibular neck fractures, complete absence of sensation is possible along the anterodistal portion of the leg and entire dorsum of the foot. Lateral calf sensation may be spared if the lesion is below the nerve branch to this region. When the neural insult occurs at the knee, the short head of the biceps femoris is often spared.

Patient history and physical examination are the most helpful initial clinical tools in arriving at then initial diagnosis of a strongly suspected common peroneal nerve injury. Plain radiographs may be helpful in excluding underlying traumatic injuries, such as a proximal fibular head fracture or osseous tumors, or in assessing the severity of angular deformities about the knee. CT scans and MRI are helpful in finding a compressive lesion along the course of the nerve in cases in which this is suspected. Metabolic and hematologic studies may be helpful in conditions such as diabetic peripheral polyneuropathy, alcoholic polyneuropathy, polyarteritis nodosa, and hyperthyroidism. A nerve biopsy, although largely unnecessary, may confirm the disorder. It, however, is better to evaluate the palsy through neurophysiological procedures, which can localize the level of the nerve palsy, reveal the underlying pathology, and establish the prognosis.

### Electrodiagnostic Evaluation

#### *Nerve conduction study*

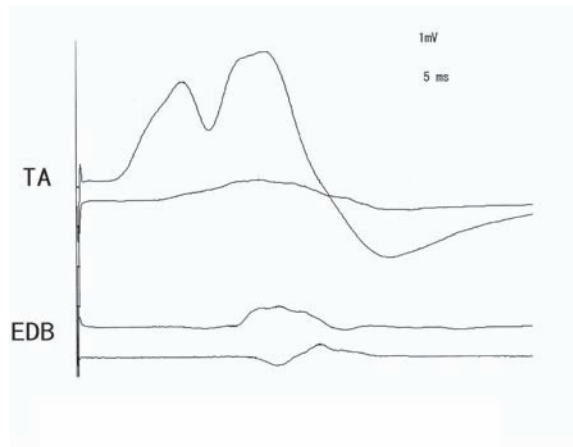
A peroneal motor nerve conduction study (NCS) should be performed first. The usual method for an NCS is to record from the EDB muscle while stimulating the nerve first at the ankle and then above and below the head of the fibula. Superficial peroneal sensory NCSs should also be performed to localize the site of injury and to assess the underlying pathology, axonal loss, demyelination, or both; in demyelinating lesions at the fibular neck, focal slowing or a conduction block are demonstrated across the fibular neck with a motor NCS. If axonal loss predominates, the compound muscle action potential (CMAP) will be reduced in amplitude at all stimulation sites along the nerve. The amount of degeneration of the axon can be approximately estimated by comparing the distal CMAP amplitude of the involved

side with that of the contralateral non-injured side, always assuming that the contralateral side is normal and not affected. If the contralateral limb responses are normal, one can estimate the amount of axonal loss by expressing the CMAP on the affected side as a percentage of the nonaffected side. This method is independent of the location of the active recording electrode and is valid in both circumstances.

The superficial peroneal sensory nerve action potential (SNAP) is important, and an abnormality of the sensory evoked response implies that the lesion is distal to the dorsal root ganglion, although this does not completely rule out the possibility of an L5 radiculopathy. A loss in amplitude of this response implies some axonal loss affecting either the common peroneal nerve or its superficial branch. The particular portion of the nerve that is injured cannot be determined if only a superficial peroneal nerve sensory study is performed. Comparison of the latency and amplitude of the superficial peroneal nerve SNAP with the contralateral limb is required to define the approximate degree of axonal loss. The amplitude of the SNAP of the superficial peroneal nerve will also be reduced. Axonal loss is provided by the decrease in the SNAP and the EMG findings of the peroneally innervated muscles, but it is known that the number of fibrillation potentials or positive sharp waves correlate poorly with the degree of axonal loss.<sup>9</sup> Therefore the best way to quantify axonal loss is to compare the distal CMAP amplitude on the symptomatic side with one of the contralateral asymptomatic normal side, or normal control values. Additionally, CMAP recording from the TA is more useful than the routine study in patients with a foot drop, because in patients with a foot drop, it is the weakness of the TA that accounts for the clinical deficits. If the recording the EDB does not localize the lesion by demonstrating focal slowing or conduction block, the peroneal motor study should be repeated recording the TA (Figure 1). A tibial motor NCS and F wave study, and sural sensory NCS must be performed to exclude a more widespread lesion and systemic neuropathy. Moreover a comparison with the contralateral side, contralateral peroneal MCS and superficial sensory NCS had better be performed as mentioned above.

#### *Needle EMG*

The role of needle EMG is to confirm the localization revealed by NCS, to assess the severity of the lesion, and to exclude other causes. The muscles to be sampled first are those innervated by the deep and superficial peroneal nerves (TA, extensor hallucis longus, peroneus longus or brevis). If any of these muscles are shown to be abnormal, non-peroneal-innervated muscles supplied by the L5 root (for example, tibialis posterior, flexor hallucis longus) must be sampled. Muscle sampling of the short



**Fig. 1** Usefulness of recording the tibialis anterior in peroneal neuropathy. When performing peroneal conduction studies, recording the tibialis anterior is more informative than routine studies. In these traces, the tibialis anterior and extensor digitorum brevis are co-recorded while the peroneal nerve is stimulated below the fibular neck (upper trace) and at the lateral popliteal fossa (lower trace). The conduction block is only detected in the recording from the tibialis anterior, but not evident in the extensor digitorum brevis recording.

head of the biceps femoris may be important, because it is the only muscle supplied by the peroneal division of the sciatic nerve that originates above the deep peroneal nerve. Abnormalities in this muscle, therefore, imply that the lesion is proximal to the deep peroneal nerve, that is to say, injury at the level of the sciatic nerve or higher.

If any abnormalities are found in the hamstrings or distal tibial-innervated muscles, more extensive needle EMG must be performed, including sampling the paraspinal and gluteal muscles (Table 2).

Information gained by needle EMG is derived from spontaneous and voluntary activity.<sup>10</sup> The presence of abnormal spontaneous activity represents neurogenic disorders and is most important in needle EMG. The evaluation of voluntary activity includes an assessment of the shape of individual MUPs and MU recruitment (interference pattern: IP). Each of these assessments can be performed by both quantitative and qualitative analyses. However, quantitative analyses of MUP recruitment and interference pattern are not widely used in clinical settings, because of their low sensitivity.<sup>11,12</sup> Therefore the quantitative analysis of individual MUP parameters has been the main aim of quantitative EMG. However, there are fundamental limitations to quantitative MUP analyses that rely on MUP parameter measurements. In MUP parameters, the duration has been considered a cardinal feature that can specifically differentiate between normal and neurogenic changes,<sup>13</sup> as confirmed by simulation studies.<sup>14</sup> Those studies revealed that MUP duration was determined mainly by the number of muscle fi-

bers belonging to a MU that lay within a rather wide (2.5 mm in radius) uptake area and was expected to differentiate between normal and neurogenic grouping.

The most important characteristics of neurogenic changes manifest themselves as a recruitment pattern in the IP, rather than as a change in the MUP waveforms. Neurogenic disease typically produces a reduced number of functioning MUs. At a given level of force, the remaining MUs fire at a higher rate. To differentiate between normal and neurogenic changes, the relationship between the recruitment of MU and how the patient produces a force must thus be examined. In contrast, MUP shapes in neurogenic disease reflect the stage of reinnervation, acute or chronic, ongoing denervation or reinnervation, and inactive denervation. This can range from normal MUPs, when there has been a loss of MUPs from partial denervation in an acute phase, to “so called” giant MUPs in a chronic process, in which collateral sprouting is occurring at the time of assessment, has almost finished or has already completely occurred. That is the significance of MUP waveform analysis, which helps determine the time course.

The current form of individual MUP analysis usually fails to resolve two important factors that can affect MUP sampling: focusing and level of contraction.<sup>10</sup> Focusing, adjusting the electrode position to acquire sharp MUPs with the highest possible amplitude, is attempted differently among different examiners. The different degree of focusing affects the MUP amplitude values. For example, the upper limit of 1.6 mV in the tibialis anterior described by Bischoff *et al.*<sup>15</sup> is quite different from that used in our laboratory. Fortunately, simulation studies suggest that the MUP duration is theoretically uninfluenced by focusing.<sup>13</sup> However, in actual observations, the duration of MUPs from one MU varies considerably. The level of contraction is also expected to influence the MUP parameter according to the size principle. However Ertas *et al.*<sup>16</sup> did not find evidence of the size principle for the few earliest recruited MUPs. Our investigation over a wider range of contraction did clearly reveal the size principle in needle EMG.<sup>17</sup> That is, MUPs recruited with stronger contractions had higher amplitudes and longer durations. The measurement of the MUP duration also has several serious problems.<sup>18</sup> The terminal portion of the MUP waveform returns very gradually to the baseline. Visual assessment of the MUP duration, therefore, is difficult. The MUP duration measured visually is affected by the amplifier gain, and duration values so measured are comparable only with those recorded with the same gain. Inter- and intraoperator reproducibility of duration measurements is quite low,<sup>19</sup> even with automated measurements, and the cursor positions for duration measurements made by automated techniques often require operator correction. A quiet baseline is required for accurate duration measurements, so very weak contractions are suit-

**Table 2** EDX localizing the lesion site

	Peroneal nerve	Sciatic nerve	Lumbosacral plexus	L5 root lesion
NCS				
Peroneal nerve				
conduction block or temporal dispersion at fibular head	×			
CMAP low amplitude	×	×	×	×
Superficial peroneal SNAP abnormal	×	×	×	
Sural SNAP abnormal		×	×	
Tibial nerve				
CMAP low amplitude		×	×	
Needle EMG sampling				
Tibialis anterior	×	×	×	×
Extensor hallucis longus	×	×	×	×
Peroneus longus	×	×	×	×
Tibialis posterior		×	×	×
Short head of the biceps femoris		×	×	×
Tensor fascia latae			×	×
Paraspinal muscles				×

x means abnormality might be detected.

able for analysis. The sensitivity of duration measurements in discriminating between normal and neurogenic is not very high, probably because of the aforementioned factors.<sup>20</sup>

When performing needle EMG, detection of the abnormal spontaneous potential at rest is therefore most important for diagnosing neurogenic diseases. The recruitment pattern and interference pattern are also important when understanding how the patient produced the force. However the value of the quantitative analysis of MUP is quite limited for diagnosis. If the abnormal spontaneous activity was not detected at rest and no obvious abnormal recruitment pattern was seen in needle EMG, care must be taken when evaluating the lesion only by quantitative MUP analysis.

### Pathophysiology and Prognosis

Determining the underlying pathophysiology is very important in assessing the prognosis. In general, the prognosis for a demyelinating lesion is much more favorable than for an axonal loss lesion. By nerve conduction studies, we can explore whether demyelination or axonal loss is the responsible pathophysiology.

In demyelination, there is evidence of conduction block and slowing across the lesion. The number of blocked fibers can be approximated by comparing the CMAP amplitude above and below the lesion. To approximate the number of fibers that have undergone axonal degeneration, the distal CMAP amplitude on the involved side is compared with that on the contralateral side, again always provided that the contralateral side is

normal.<sup>9</sup> In demyelination, the underlying axon remains intact, and the repair process consists only of remyelination. Remyelination usually occurs over several weeks. In contrast, recovery from axonal loss lesions requires the regrowth of the terminal axon or collateral sprouting from unaffected axons. These processes are usually quite slow and may be incomplete. It will take many months to a year or more to recover function in a patient with severe axonal loss, which may not even then recover sufficiently. In contrast, a patient with a pure demyelinating neuropathy may recover completely over a month or two.<sup>6</sup>

### Summary and Future Research

The American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM)<sup>21</sup> reported that from the point of an evidence-based review, in patients with suspected peroneal neuropathy, the following EDX studies are possibly useful, to make or confirm the diagnosis:

#### 1. NCS

- Motor NCSs of the peroneal nerve recording from the TA and EDB muscles, including an assessment of peroneal conduction through the leg and across the FH (Fibula Head) (Level C recommendation, Class III evidence);
- Orthodromic and antidromic superficial peroneal sensory NCS (Level C recommendation, Class III evidence);
- At least one additional normal motor and sensory NCS in the same limb, to assure that the peroneal neuropathy is isolated, and not part of a more wide-

spread local or systemic neuropathy (Expert opinion).

2. Data are insufficient to determine the role of needle EMG in making the diagnosis of peroneal neuropathy (Data inadequate or conflicting, Class IV evidence). However, abnormalities on needle examination outside of the distribution of the peroneal nerve should suggest alternative or additional diagnoses (Expert opinion).

3. In patients with confirmed peroneal neuropathy, EDX studies are possibly useful in providing prognostic information, with regards to recovery of function (Level C recommendation, Class III and IV evidence).

This report reviewed the utility of electrodiagnostic techniques in evaluating patients with suspected peroneal nerve palsy. Due to the widespread utilization of EDX testing in the evaluation of patients with suspected peroneal neuropathy (suggesting that clinicians have found EDX testing to be useful in this setting) most studies were published prior to the development of more rigorous standards for study design and assessment of the literature. Consequently, available studies only provided Class III and IV evidence, resulting in a conservative assessment of their utility. In particular, classifying of needle EMG data as Class IV evidence because the examiner is not masked to clinical data, results in understatement of its utility. It is important that future studies to evaluate the usefulness of EDX examine not only suspected peroneal neuropathy, but also other entrapment neuropathies.

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