Sensory Nerve Conduction Studies of the Superficial Peroneal Nerve in L5 Radiculopathy

Ying-Hao Ho, Sui-Hing Yan, Yuh-Te Lin, and Yuk-Keung Lo

Abstract-

Background: Theoradically, sensory nerve action potential (SNAP) of the superficial peroneal nerve (SPN) should be preserved when L5 roots are damaged. Previous study indicated that SNAP of SPN was lost or reduced in amplitude in patiens with L5 radiculopathy. To address this issue, this study compared results of SPN sensory conduction studies between healthy subjects and patients with L5 radiculopathy.

Methods: Ninety-four healthy subjects were enrolled and assigned to two age groups: group I, (≤ 60 years, n=61) and group II (> 60 years, n=33). Forty-one patients with unilateral L5 radiculopathy were enrolled by our electrodiagnostic laboratory between July 2000 and July 2003 and assigned to two age groups: 60 years or below (n=19) and above 60 years (n=22).

Results: SPN response was found to be abnormal in only 1.6% of group I healthy subjects, but absent or abnormal SPN response was noted in 21.1% of patients with L5 radiculopathy of the same age group (p=0.01). However, a greater proportion of (27.3%) our healthy subjects above 60 years had abnormal SPN responses. This proportion was similar to that of patients with L5 radiculopathy and abnormal SPN response (31.8%) (p=0.72).

Conclusions: SPN sensory responses are reliably obtained in most healthy subjects under 60 years of age. Absence of SNAP or reduced SNAP amplitude of SPN on the side of their lesions did not necessarily exclude the diagnosis of L5 radiculopathy in the patients under 60 years of age.

Key Words: Electrodiagnosis, L5 Radiculopathy, Sensory nerve conduction, Superficial peroneal nerve

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INTRODUCTION

Although a standard recording technique used for doing sensory conduction studies of the superficial peroneal nerve (SPN) has been established for years[1-8], the studyof SPN sensory conduction study has not been reported in Taiwan. The SPN is derived from L5 roots[9] and sensory nerve action potential (SNAP) of the SPN should be preserved in L5 radiculopathy. It is commonly thought in electrodiagnosis that the dorsal root ganglion (DRG) resides within the intervertebral foramen, making it distal to the site of compression in radiculopathy.

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However, Levin[10] has reported that 21% (13/62) of the patients with L5 radiculopathy in his study had absence of SNAP or reduced SNAP amplitude of SPN. A series of anatomic studies[11-13] reported that 10-20% of L5 DRG are located in an intraspinal canal and suggested that disk protrusion or spondylotic encroachment may compress L5 DRG. These studies made us wonder whether there was an absence of SPN sensory response in L5 radiculopathy or whether the absence of SPN response can be attributed to normal variability.

In this study, after grouping our patients according to age and excluding those with a history of diabetes, we characterized and determined the reliability of normal SPN sensory responses in healthy individuals and then compared our findings with SPN sensory responses in patients with unilateral L5 radiculopathy.

PATIENTS AND METHODS

Healthy subjects and patients

After ensuring that there were no clinical features of peripheral neuropathy or history of diabetes, we enrolled 94 healthy subjects and assigned them in to two age groups: group I (≤ 60 years), and group II (< 60 years).

In our study, a patient was diagnosed as having unilateral L5 radiculopathy when he or she had (1) a clear history of low back pain radiating in a L5 distribution pattern and/or L5 segmental sensory loss or weakness, (2) needle EMG evidence of active denervation with/without marked neurogenic recruitment in L5 segmentally innervated leg muscles, including distal muscles (tibialis posterior, tibialis anterior, peroneus longus, or extensor digitorum longus) and/or proximal muscles (tensor facia lata or gluteus medius), and (3) either positive sharp waves in the lower lumbar paraspinal muscles or neuroimaging studies of the lumbosacral spine demonstrated moderate-to-severe L4-5 spinal stenosis or surgical identification of L5 root compression. We excluded patients with L5 radiculopathy if they had any one of the following: (1) a history of diabetes, (2) absence of SNAP or reduced SNAP amplitude of sural nerve(s), or (3) a conduction block across the fibular head in the peroneal nerve motor response. There were 41 patients fulfilling the above criteria of active L5 radiculopathy between July 2000 and July 2003. They were assigned to two age groups: group I (≤ 60 years, n=19) and group II (> 60 years, n=22).

Methods for needle EMG screening and nerve conduction studies were performed according to the guidelines of the American Association of Electrodiagnostic Medicine[14]. Nerve conduction studies were performed with surface-stimulating electrodes and a pair of cup surface-recording electrodes to obtain a series of nerve conduction studies with an electrodiagnostic machine (Viking IV D, Nicolet, Madison, Wisconsin, USA).

The site of stimulation was 15 cm proximal to the active ankle electrode, just anterior to the edge of the shaft of the fibula. The active recording electrode was placed at a standard site, a point on the bimalleolar line, which was in the midway between the edge of the tibia and the tip of the lateral malleolus[5-8]. The reference recording electrode was placed 3.0 cm distal to the active electrode on the dorsum of the ankle (Figure).

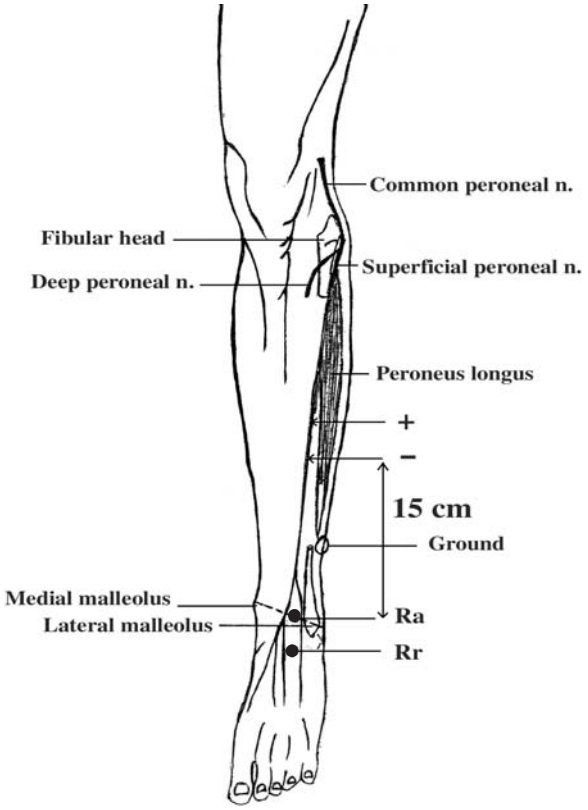

Figure. Sensory conduction study of the superficial peroneal nerve; n: nerve; Ra: active recording electrode; Rr: reference recording electrode.

Skin temperatures at the ankle ranged from 30˚C to 34˚C in all subjects.

Latencies were measured from the stimulus artifact to the first negative defection of the evoked response, and amplitudes were measured from the onset point to the peak. Lower limits of amplitude were based on the range of the normative data. SNAP was considered absent when no constant waveforms could be detected after repeated stimulations by two examiners, and it was considered abnormal when there was a greater than 50% reduction in amplitudes compared to that on the opposite side or it was below the lower limit of normative data.

Statistical analysis

We calculated the mean and standard deviation (SD) for latencies, amplitude, and nerve conduction velocities from the measured values in healthy subjects. The differences between latencies, amplitudes and nerve conduction velocities in the two groups of healthy subjects were analyzed by Student's t test. Absence of SNAP or abnormal SPN of the age-matched groups in healthy subjects were compared with those of patients with L5 radiculopathy using χ^2 test or Fisher's exact test.

RESULTS

Data from healthy subjects

Normal values identified in this study are summarized in Table 1. Mean values for latencies and conduction velocities of healthy subjects in the SPN study were 2.98±0.24 ms and 50.6±4.1 m/s in group 1 and 3.20±0.30 ms and 47.4±4.4 m/s in group 2. Lower limit of amplitude was based on the range of the normative data: 5.7 μV in group 1(under 60 y/o) and 3.5 μV in group II.

There were significant statistical differences in latencies, amplitudes and conduction velocities between group I and group II ($p < 0.001$) (Table 1). Of the total number of healthy subjects, 10.6% were found to have either absence of SNAP or abnormal SPN sensory response at least on one side. Group II subjects had a higher rate (27.3%) of absence of SPN or abnormal SPN response than that of Group I subjects (1.6%) below 60 years old ($p < 0.001$) (Table 2).

Patients with L5 radiculopathy

Of the 41 patients with unilateral L5 radiculopathy, 11 (26.8%) patients were found to have absence of SNAP or abnormal SPN sensory responses on the side of their lesions. Among these patients, 4 of 19 (21.1%) of those were 60 years old or younger and 7 of 22 (31.8%) of those were above 60 years. Electrophysiological findings, neuroimaging studies and clinical symptoms of the 11 patients with absence of SNAP or abnormal SPN sensory response on the side of their lesions are listed in Table 3. In patients 60 years old or younger, a statistical difference ($p = 0.01$) was found when comparing the rates of absence of SNAP or abnormal SPN response rates in patients L5 radiculopathy with those of the healthy subjects. However, no significant difference was found between patients and healthy subjects above 60 years old (Table 2).

DISCUSSION

The method we used to perform sensory conduction study of the SPN followed the guidelines of Sensory Conduction Studies in the American Association of

Table 2. *Comparison of loss or abnormal SPN conduction studies in health control subjects and L5 radiculopathy*

Age	Healthy control	L5 radiculopathy	P
≤ 60 y/o	1/61 (1.6%)	4/19 (21.1%)	0.01
> 60 y/o	9/33 (27.3%)	7/22 (31.8%)	0.72
Total	10/94 (10.6%)	11/41(26.8%)	< 0.05

Table 1. *Results of superficial peroneal nerves conduction studies in healthy control subjects*

Group	n	Age (y)	Latency* (ms)	Amplitude* (μV)	Conduction velocity* (m/s)
≤60 y/o	61	36.7±13.8 (16-60)	2.98±0.24 (2.45-3.80)	10.9±3.5 (5.7&-25.0)	50.6±4.1 (39.5-61.2)
>60 y/o	33	75.5± 7.1 (62-91)	3.20±0.30 (2.60-4.10)	9.0±3.5 (3.5&-17.0)	47.4±4.4 (36.6-57.7)
Total	94	50.4±22.0 (16-91)	3.05±0.28 (2.45-4.10)	10.4±3.6 (3.5&-25.0)	49.6±4.4 (36.6-61.2)

*P< 0.001 between the group of below 60 y/o and above 60 y/o. Data are the mean±SD. Data in parentheses are range. Lower limits of amplitude are based on the range of the normative data.

Table 3. *Results of 11 patients of unilateral active L5 radiculopathy with loss or abnormal SPN sensory responses at the lesion side*

Case	Age	Sex	SNCS (μV)		EMG							Neuroimaging studies	Clinical symptoms
	(y/o)		SPN	Sural	MG	EDB	PL	TA	TP	TFL	PSM		
1	24	M	4/8	17.8/18.6	●	⊗	⊗	●	●	⊙	●	MRI: HIVD L4-5, severe	Radiating pain for 6 months
2	45	M	4/11.9	13.4/14.5	⊙	●	⊗	●	●	●	○	MRI: L4-5, severe stenosis	Radiating numbness over L5 dermatome for 1 month
3	49	M	-/10.5	10.3/10.1	○	⊗	●	●	●	●	●	MRI: L4-5, severe stenosis	Radiating pain and weakness for 1 month
4	60	F	-/-	7.3/8.4	○	⊗	⊗	●	●	⊙	○	MRI: L4-5, severe stenosis	Radiating pain for 2 months
5	65	F	-/8.1	7.8/9.7	○	⊗	●	●	●	⊙	○	MRI: L4-5-S1, severe stenosis; rotatory scoliosis	Radiating pain, weakness and numbness for 3 years
6	66	F	-/-	5/5	○	●	⊗	⊙	●	⊙	●	MRI: L4-5, moderate stenosis	Radiating pain for 3 months
7	71	M	-/6.5	5.2/5.4	●	●	⊗	●	●	⊙	●	MRI: L4-5, severe stenosis	Weakness over L5 myotome for 1 month
8	72	M	-/-	8.7/12.5	○	⊗	●	●	●	⊙	●	MRI: L4-5-S1, severe stenosis	Radiating pain, weakness and numbness for 1 years
9	75	M	-/12	13/15	○	⊗	⊗	●	●	●	⊗*	MRxI, CTM: L4-5-S1, severe stenosis	Radiating pain, numbness and weakness for 2 month
10	77	F	7/16.9	17.6/17.1	●	⊗	⊗	●	●	⊙	○	MRI: L4-5-S1, severe stenosis	Radiating pain and weakness for 6 month
11	80	M	-/5	7/8	●	●	⊗	●	●	⊙	●	Not done	Radiating pain, drop foot and numbness over L5 der matome for 6 months

Involved side/Uninvolved side; SNCS: sensory nerve conduction study; EMG: electromyography; SPN: superficial peroneal nerve sensory response. Sural: sural nerve sensory response; - indicates absent; ●: active denervation with/without marked neurogenic recruitment; ⊙: only marked neurogenic recruitment; ○: normal pattern; ⊗: not performed; ⊗*: not performed due to surgical scar; MG: medial gastrocnemius; EDB: extensor digitorum brevis; PL: peroneus longus. TA: tibialis anterior; TP: tibialis posterior; TFL: tensor facia lata. PSM: paraspinal muscle; MRI: magnetic resonance image; CTM: computerized tomography myelography. HIVD: herniated intervertebral disc.

Electromyography and Electrodiagnosis (AAEE)[8]. The standard location at the ankle, on the bi-malleolar line and at the midway between the edge of the tibia and the tip of the lateral malleolus, gives a higher amplitude and a faster rise-time response than do other sites over the dorsum of the foot or ankle[5]. Although several groups have reported the reliability of SPN nerve conduction results in the healthy individuals[1-5], most of these studies had fewer subjects than ours and they did not group the results by age. The results of our study showed that absence of SNAP or abnormal SPN responses in on least one side in 10.6% of our healthy subjects, a finding similar to the nine percent reported by Levin[5]. Some investigators have reported the rate of absent SPN responses to be 2-5% in healthy subjects[1-3]. Jabre reported normal SPN responses in all subjects[4].

In our study, using the same method of measuring, we found healthy individuals above the age of 60 years to have a 27.3% absent SPN response rate or abnormal SPN response rate. This finding may be related to the anatomic location of SPN over the anteriolateral aspect of the leg, where it is vulnerable to chronically asymptomatic injury in elderly individuals. Healthy individuals over the age of 60 years old had lower SNAP amplitudes and slower nerve conduction velocities than those of persons younger than 60 years in our study, supporting the finding of other studies that advanced age negatively affects amplitude and nerve conduction velocities in nerve conduction studies[15-16]. The SPN sensory response has often a small SNAP and may be hard to elicit in some older individuals. Therefore, very low or absent SPN sensory responses in patients of advanced age must be interpreted with caution. They should not necessarily be considered abnormal without clinical or laboratory confirmation. However, the healthy subjects 60 years old or younger only had an abnormal SPN response rate of 1.6% (1 of 61) and higher reliability of SPN sensory response.

There is no widely accepted standard definition for nerve root compression without surgical confirmation[17-18]. However, the surgical identification of L5 root lesion could not be performed in every patient because of the invasiveness of surgical procedures. Therefore, in addition to using typical clinical pictures and neurological

deficits for diagnosis, we also defined the acquired factors with anatomic evidence of L4-5 spinal stenosis in neuroimaging studies or with the presence of positive sharp waves in the lower lumbar paraspinal muscles for diagnosing L5 radiculopathy. The presence of positive sharp waves in paraspinal muscles is also a strong electrophysiological evidence of motor axonal loss at the root level[19].

In our study, the patients with absent or abnormal SPN sensory responses were excluded if they had possible peroneal neuropathy, sciatic neuropathy and/or lumbosacral plexopathy by clinical history, electrophysiological and neuroimaging studies. None of our patients with radiculopathy had diabetes. On the basis of results from the Rochester Diabetic Neuropathy Study cohort[20], most patients with diabetic lumbosacral radiculoplexopathy are not expected to have developed a preexisting distal diabetic polyneuropathy. Thus, it is unlikely that our patients with L5 radiculopathy had proximal diabetic neuropathy, plexopathy, or radiculoplexopathy, which may affect the SPN sensory response.

In our study, 26.8% of our patients with L5 radiculopathy had lost or reduced SNAP of SPN on the side of their lesions, a finding similar to the 21% reported by Levin[10]. There was a significant statistical difference in the absent or abnormal SPN response in the conduction reliability studies of the healthy patients and the patients 60 years of age or below with L5 radiculopathy. Therefore, our results reconfirm those of Levin[10] who reported that lost or reduced SNAP of SPN may be noted in some younger patients with L5 radiculopathy if L5 DRGs are located in an intraspinal canal and disk protrusion or facet encroachment compressed at or distal to the L5 DRG. Our findings and Levin's are further supported by a series of anatomic studies of L5 DRG location. In a cadaver study, Sato[11] found 11% of L5 DRG to be located within the spinal canal. Hamanishi[12] noted that 13% of L5 DRG in patients with low back pain or sciatica were located in an intraspinal canal by axial views of magnetic resonance imaging. Kikuchi[13] found 19.2% of the L5 DRGs to be located in an intraspinal canal in 77 cadavers and 38.9% of the L5 DRG in an intraspinal canal in a radiographic study of 131 patients with L5 radiculopathy.

In conclusion, the SPN sensory responses are reliably obtained in most healthy subjects under 60 years of age. An absent or reduced SNAP of SPN on the side of their lesions does not necessarily exclude the diagnosis of L5 radiculopathy in the patients under 60 years of age if L5 DRGs are located in an intraspinal canal.

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