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Shu-Mon Chen

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Thy-Sheng Lin

Chang-Zern Hong

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# Subclinical Superficial Peroneal Sensory Neuropathy in Cyclists in South Taiwan

Shu-Min Chen, MD, Jo-Tong Chen, MD, Thy-Sheng Lin\*, MD, Chang-Zern Hong, MD

Departments of Physical Medicine and Rehabilitation, and Neurology\*  
National Cheng-Kung University

The purpose of this study is designed to assess the superficial peroneal sensory nerve conduction of bicyclists since some bicyclists experience tingling or numbness over the dorsum of the foot during prolonged bicycling. Bilateral superficial peroneal nerve conduction studies were performed on 17 bicyclists compared with 20 normal subjects. Compound nerve action potentials were recorded with surface electrodes over the medial and intermediate dorsal cutaneous branches of the superficial peroneal nerves located at the anterior aspect of the ankle. The superficial peroneal nerve (SPN) was stimulated at the anterolateral aspect of the leg 12 cm proximal to the site of active recording electrode. One of our subjects (2.7%, 1/37) failed to show the response of the intermediate branch of the SPN. The distal latency of either branch of the SPN was significantly longer ( $p < 0.01$ ) in the bicyclists ( $3.38 \pm 0.30$  ms for medial branch and  $3.34 \pm 0.35$  ms for intermediate branch) than that in the normal control subjects ( $3.10 \pm 0.23$  ms for medial branch and  $3.08 \pm 0.25$  ms for intermediate branch). Using "mean plus 2 SD" of the control subject as the upper normal limit, 11 bicyclists (64.7%) had abnormal distal latencies either in the medial or in the intermediate branches of the SPN. There was no significant difference ( $p > 0.05$ ) in the amplitude of nerve action potential between bicycle ( $11.54 \pm 4.50$   $\mu$ v for medial branch and  $9.57 \pm 4.39$   $\mu$ v for intermediate branch) and control groups ( $13.58 \pm 4.92$   $\mu$ v for medial branch and  $11.77 \pm 5.97$   $\mu$ v for intermediate branch). All the bicyclists had the habit of excessive ankle motion. Therefore, the repetitive ankle motion in bicyclists may lead to the neuropathy of the SPN. (J Rehab Med Assoc ROC 1997; 25(1): 27-32)

**Key words:** superficial peroneal sensory nerve, nerve conduction, bicyclist

## INTRODUCTION

Bicycling is no longer only for professional athletes or special groups, but is becoming increasingly popular exercise among the general population. The injuries of bicycling include traumatic and nontraumatic injuries. The peripheral nerve injuries in bicyclists have been reported including ulnar nerve compression<sup>[1]</sup> and pudendal nerve compression<sup>[2]</sup>. Some bicyclists experience transient tingling or numbness over the superficial

peroneal nerve supplied territory in the dorsum of the foot during prolonged riding but they obviously have no history of acute trauma to the foot. To our knowledge, there was no study about the correlation between the repetitive ankle movement and the superficial peroneal nerve (SPN) lesion in the bicyclists. The purpose of this study is to assess the SPN conduction of bicyclists to see if there is any subclinical abnormality in bicyclists as compare to the normal control subjects.

## MATERIALS AND METHODS

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Address correspondence to: Dr. Shu-Min Chen, Department of Physical Medicine and Rehabilitation, National Cheng Kung University Hospital, No.138, Sheng Li Road, Tainan, Taiwan

TEL: (06) 2353535 ext. 2666, FAX: (06) 2766106

## Subjects

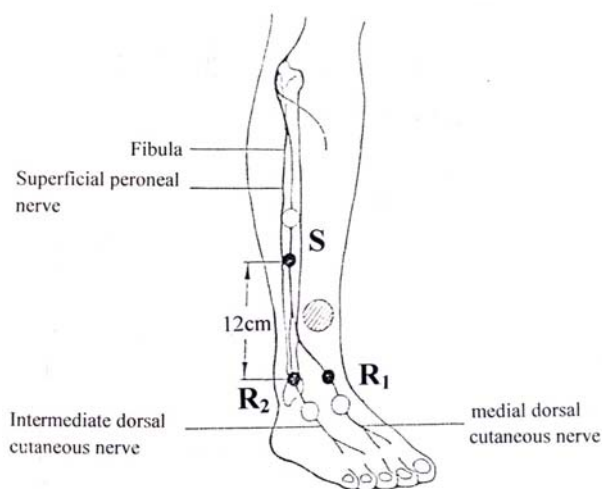
The study population for control data consisted of 20 volunteer, with a mean age of 30.0 years (ranged from 25 to 41 years). The bicyclist population consisted of 17 bicyclists with a mean age of 24.0 years (ranged from 15 to 48 years), who had performed regular bicycle exercise 30-40 hours per week for 1-3 years. All the subjects are males. None of the subjects had a history of alcoholism, diabetes, uremia, or hepatic, malignant, neurologic or psychiatric disease. Every subject received a brief physical examination (including neurological examination).

## Equipment

An EMG machine ( Nicolet Viking ) was used for the nerve conduction studies. The sensitivity was set at 20  $\mu$ V/division. The sweep speed was set at 2 msec/cm. The stimulus was a rectangular impulse with a duration of 0.1 msec at a frequency of 1 Hz.

## Procedure of sensory nerve conduction study

The latency of the evoked potential of the sural nerve was evaluated in each subject to rule out subclinical polyneuropathy. Nerve conduction studies were performed on two separate branches of superficial peroneal sensory nerve of both sides. An antidromic technique, modified from both Izzo's [3] and Jabre's method [4], was used. The compound nerve action potential was recorded with surface electrodes over the medial and intermediate dorsal cutaneous branches of the SPN located at the anterior aspect of the ankle (Fig 1). For the medial branch, the active recording electrode was placed at the midpoint of the line connecting the tips of the medial and lateral malleolus. For the intermediate branch, the active recording electrode is placed at the ankle level about 1 cm medial to the lateral malleolus. The reference electrodes were placed 3 cm distal to the active electrodes following the direction of toe extensor tendon. The nerve was stimulated at a point 12 cm proximal to the active recording electrode. The ground electrode was placed on the anterior aspect of the lower part of the leg between the stimulating and recording electrodes. The exact sites of active recording electrode were further adjusted so that a maximal amplitude of nerve active potential could be best recorded. The distal



**Fig. 1.** The compound action potential was recorded with surface electrodes over the medial and intermediate dorsal cutaneous branches of the superficial peroneal nerve located at the anterior aspect of the ankle. The superficial peroneal nerve was stimulated on the anterolateral aspect of the lower leg, 12 cm proximal to the site of active recording electrode.

latency was taken to the peak. The amplitude was measured for the baseline to the initial negative peak.

## Temperature correction

All studies were performed at an ambient room temperature of 22 to 25 degrees. Skin temperature at the ankle was recorded in all subjects, using the correction formula [5] to correct a skin temperature to 32 °C :

NCV corrected (M/sec)=  $2.0[(32 - \text{skin temp}(^{\circ}\text{C})) \text{ M/sec} + \text{NCV}(\text{M/sec})]$ , and thus,

NCV corrected = Distance/ Latency corrected

Latency corrected

= Distance/ NCV corrected

= Distance/ {2000(mm/sec) x (32 - skin temp) + NCV recorded}

= Distance/ {2000(mm/sec) x (32-skin temp) + Distance/Latency recorded}

Latency corrected (ms)

= Latency recorded(ms) x Distance(mm) / { 2000 mm/sec x [(32- skin temp( $^{\circ}$ C)) x Latency recorded(ms) + Distance(mm) },

## STATISTICAL ANALYSES

We compared mean values of the distal latencies of SPN between the bicyclist and the control groups by using Student's two-sample t test. A value of  $p < 0.01$  was considered as statistically significant.

## RESULTS

All control subjects had no musculoskeletal anomaly or neurological abnormality. All bicyclists were asymptomatic except for one subject. The symptomatic bicyclist had tingling and numbness without decrease in sensation to light touch or pin prick on the foot dorsum over the cutaneous distribution of the nerve. In one of the bicyclists, the left extremity failed to show the response of the intermediate branch of the SPN, but no symptom of the foot was reported. The mean values of the distal latencies of the medial and intermediate dorsal cutaneous branches of the SPN in the control and bicyclists were shown in Table 1.

The distal latencies of the medial and intermediate branches of the SPN in the control group were  $3.10 \pm 0.23$  ms (range: 2.63-3.71) and  $3.08 \pm 0.25$  ms (range: 2.72-3.71), respectively. In bicyclists, the mean distal latencies were  $3.38 \pm 0.30$  ms (range: 2.72-3.89) and  $3.34 \pm 0.35$  ms (range: 2.75-4.15) respectively. The mean distal latency of the either branch of the SPN was significantly higher ( $p < 0.01$ ) in the bicyclists than that in the normal control subjects (Table 1). There were no significant differences between right and left sides, or between medial and intermediate branches. Using "mean plus 2SD" of the

control subject as the upper normal limit, 11 bicyclists (64.7%) had abnormal distal latencies either in the medial or in the intermediate branch of the SPN. The abnormal distal latencies in these 11 bicyclists are listed in Table 2. There was only one subject, the symptomatic bicyclist, who had abnormal distal latencies in all branches (bilateral medial and intermediate branches). This distribution of involvement was not symmetrical. There was no significant difference ( $p > 0.05$ ) in the amplitude of nerve action potential between bicycle ( $11.54 \pm 4.50$   $\mu$ v for medial branch and  $9.57 \pm 4.39$   $\mu$ v for intermediate branch) and control groups ( $13.58 \pm 4.92$   $\mu$ v for medial branch and  $11.77 \pm 5.97$   $\mu$ v for intermediate branch).

## DISCUSSION

The SPN at the dorsum of the foot follows a highly variable anatomic course. In most cases, the nerve penetrated into the crural fascia at the site with an average of  $12.3 \pm 2$  cm proximal to the ankle joint and then divided into a larger medial dorsal cutaneous branch and a smaller, more laterally located, intermediate dorsal cutaneous branch<sup>[6]</sup>. The nerve action potential may be absent in up to 2-9 % of the population<sup>[3,7,8,9]</sup>. The SPN conduction study has been used to assess the SPN lesion in various condition<sup>[9,10]</sup>. Izzo described a technique to measure the superficial peroneal sensory nerve by recording the action potential at the ankle level over the medial and intermediate dorsal cutaneous branches, which were located by inspection and palpation during plantar flexion and inversion of the foot. The nerve was stimulated at 14

**Table 1.** Effect of bicycle exercise on superficial peroneal nerve conduction

Subjects	Number of Subjects	Number of Nerve	Distal Latency (ms)		Amplitude ( $\mu$ v)	
			Med. Br.	Int. Br.	Med. Br.	Int. Br.
Control	20	40	$3.10 \pm 0.23$	$3.08 \pm 0.25$	$13.58 \pm 4.92$	$11.77 \pm 5.97$
Bicyclist	17	34	$3.38 \pm 0.30$	$3.34 \pm 0.35^*$	$11.54 \pm 4.50$	$9.57 \pm 4.39^*$
t			4.40	3.66	-1.85	-1.76
P			$P < 0.01$	$P < 0.01$	$P > 0.05$	$P > 0.05$

Med. Br. : Medial branch of superficial peroneal nerve.

Int. Br. : Intermediate branch of superficial peroneal nerve.

\* : The nerve number is only 33 for the intermediate branch, because one nerve could not be detected.

**Table 2.** Bicyclists with abnormal distal latencies of superficial peroneal sensory nerve

Case #	Right side		Left side	
	MB	IB	MB	IB
2	N	N	3.59	N
4	N	3.59	3.59	N
6	N	3.87	N	3.59
7	N	N	N	4.15
9	3.68	3.87	3.87	N
10	3.87	3.59	3.59	3.59
12	3.89	N	3.76	N
13	N	N	3.65	N
14	N	N	N	4.0
15	3.76	N	3.79	n
17	N	N	3.59	Absent

MB=Medial branch of superficial peroneal sensory nerve.

IB=Intermediate branch of superficial peroneal sensory nerve.

\* Case 10 was the subject with clinical symptom.

cm from the proximal recording electrodes on the anterior lateral aspect of the leg [3]. Sridhara used a similar technique to assess the entrapment of the terminal sensory branch of the SPN and revealed an unrecordable evoked response or a prolonged distal latency of the terminal sensory branches of the superficial peroneal nerve [10]. Jabre measured the NCV of the intermediate dorsal cutaneous branch by stimulating the nerve at a distance of 12 cm and claimed that was reliable [4]. In our study, the response of the medial dorsal cutaneous branch was obtainable in all subjects. Nevertheless, in one of our subjects (2.7%), or one of the limbs studied (1.4%), the response of the intermediate dorsal cutaneous branch could not be recorded by using the surface recording technique. This absent rate is similar to that reported previously. The cause of absence in nerve action potential of a "neurologically normal subject" is unknown. It is possible that the main trunk of this nerve is too deep or the subcutaneous tissue is too thick.

In our study, statistically significant differences in distal latencies of the medial and intermediate dorsal cutaneous branches of the SPN between the bicyclist group and the control group ( $p < 0.01$ ) were observed.

Sports injury to sensory nerves in the foot is not uncommon. Dyck found small, but statistically significant differences of nerve conduction in the leg and foot nerves of the runners [11]. Yasusuke described two members of a mountain climbing club had tarsal tunnel syndrome due to persistent and repetitive reciprocal movements of ankle dorsal and plantar flexion [13]. Similarly, the repetitive ankle motion in bicyclists may cause damage (repetitive friction of the nerve against the crural fascia and/or a tight shoe) of the SPN due to its anatomic location being closed to the surface of skin.

The cause of subclinical or clinical neuropathy of the SPN in bicyclists is unknown. The repetitive and excessive ankle movement during pedaling may cause thickening of crural fascia and surrounding soft tissue as in the case of entrapment neuropathy, such as carpal tunnel syndrome [12], or tarsal tunnel syndrome [13]. This type of chronic entrapment is different from the entrapment of SPN resulted from compartment syndrome (acute) as reported by Garfin et al [14] and Sridhara & Izzo [10]. Another possible mechanism is repetitive and excessive stretching of the SPN during pedaling. However, based on our study, there was no remarkable evidence of axonal lesion due to stretch, since the amplitude of nerve action potential was seen within normal limits. Therefore, the slow conduction of SPN in cyclist may indicate a more diffuse demyelinating lesion probably due to extensive friction of the nerve against the crural fascia and/or a tight shoe rather than due to limited focal demyelination due to entrapment from the pressure or due to excessive stretching. A technique named "ankling" has been used by most of professional bicyclists; they fix their ankle during pedaling. This technique may effectively prevent the SPN neuropathy if the "repetitive friction of the nerve against the crural fascia and /or a tight shoe" is the major cause of SPN neuropathy. Due to small sample size in our study, we are unable to compare the difference between the cyclists who had free ankle movement during cycling and those who fixed their ankle.

In conclusion, the repetitive dorsiflexion and plantarflexion of the ankle in bicyclists may result a demyelinating lesion of the SPN although the deficit was subclinical. In view of the pleasures and health benefits derived from the activity, bicycling exercise need not be

curtailed because of the associated mild neuropathic deficit. A large number of case survey is necessary to confirm this conclusion and to clarify the pathophysiology of SPN lesion.

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## 南台灣騎自行車者之潛在性腓淺感覺神經病變

程琬敏 陳若修 林志勝\* 洪章仁

國立成功大學醫學院附設醫院 復健部、神經部\*

自行車是一項簡單、方便、不需要耗油的代步工具，也是一項相當普及的大眾化運動。長時間騎自行車者，有些會有足背刺麻不舒服的感覺。本篇研究是以 17 位自行車騎士為對象，與 20 位控制組作比較，探討其腓淺感覺神經遠端潛期的差異。採用的方法是將表面電極置於兩側足踝前面，來記錄腓淺感覺神經的兩條分枝-足背內側及足背中間皮神經(medial and intermediate dorsal cutaneous branches of superficial peroneal nerve)的複合動作電位，而刺激腓淺感覺神經的位置是在記錄表面電極近端 12 公分處。

結果顯示：控制組其腓淺感覺神經兩分枝-足背內側及足背中間皮神經-的遠端潛期平均值，分別為  $3.10 \pm 0.23\text{ms}$  及  $3.08 \pm 0.25\text{ms}$ ；而自行車騎士此組的平均值則分別為  $3.38 \pm 0.30\text{ms}$  及  $3.34 \pm 0.35\text{ms}$ ；兩組間的差異具有統計學上的意義( $p < 0.01$ )。至於振幅，控制組其足背內側及足背中間皮神經的平均值，分別為  $13.58 \pm 4.92\mu\text{v}$  及  $11.77 \pm 5.97\mu\text{v}$ ；而自行車騎士此組的平均值則分別為  $11.54 \pm 4.50\mu\text{v}$  及  $9.57 \pm 4.39\mu\text{v}$ ；兩組間的差異並無統計學上的意義( $p > 0.05$ )。另外，回顧腓淺感覺神經的神經傳導方法之研究，約有 2-9% 做不出此條神經的複合動作電位(compound action potential)。而此篇研究，實驗組與控制組共 37 人，其中 1 人(2.7%)無法測得足背中間皮神經(intermediate dorsal cutaneous branch)此條分枝，與其他文獻比較，比例差不多。

我們的結論是：自行車騎士，其腓淺感覺神經之遠端潛期比控制組來得長。可見長期騎自行車者，因過多反覆的踝關節動作，可能會導致腓淺感覺神經病變，這可能與造成足背刺麻的原因有關。至於固定腳踝是否可以減緩神經病變，由於實驗組個數不多，無法比較。這需要更進一步的研究。(中華復健醫誌 1997; 25(1): 27-32)

**關鍵字：**腓淺感覺神經(superficial peroneal sensory nerve)，神經傳導(nerve conduction)，自行車騎士(bicyclist)