Subclinical Superficial Peroneal Sensory Neuropathy in Cyclists in South Taiwan

Shu-Mon Chen
Jo-Tong Chen
Thy-Sheng Lin
Chang-Zern Hong

Follow this and additional works at: https://rps.researchcommons.org/journal

Part of the Rehabilitation and Therapy Commons

Recommended Citation
Chen, Shu-Mon; Chen, Jo-Tong; Lin, Thy-Sheng; and Hong, Chang-Zern (1997) "Subclinical Superficial Peroneal Sensory Neuropathy in Cyclists in South Taiwan," Rehabilitation Practice and Science: Vol. 25: Iss. 1, Article 3.
DOI: https://doi.org/10.6315/3005-3846.2017
Available at: https://rps.researchcommons.org/journal/vol25/iss1/3

This Original Article is brought to you for free and open access by Rehabilitation Practice and Science. It has been accepted for inclusion in Rehabilitation Practice and Science by an authorized editor of Rehabilitation Practice and Science. For more information, please contact twpmrscor@gmail.com.
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 ± 0.30 ms for medial branch and 3.34 ± 0.35 ms for intermediate branch) than that in the normal control subjects (3.10 ± 0.23 ms for medial branch and 3.08 ± 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 ± 4.50 µv for medial branch and 9.57 ± 4.39 µv for intermediate branch) and control groups (13.58 ± 4.92 µv for medial branch and 11.77 ± 5.97 µv 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[1] and pudendal nerve compression[2]. 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

Submitted for publication: Aug 9, 1996
Resubmitted in revised form: Nov 13, 1996
Accepted in revised form: Jan 17, 1997
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
μV/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 Izso’s [9] and Jambre’s
method [14], 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 mid-point 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
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 [9] to correct a skin temperature to 32 °C:
NCV corrected (M/sec) = 2.0[(32 - skin temp(°C)) M/sec
+ NCV(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(°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±0.23 ms (range: 2.63-3.71) and 3.08±0.25 ms (range: 2.72-3.71), respectively. In bicyclists, the mean distal latencies were 3.38±0.30 ms (range: 2.72-3.89) and 3.34±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 ± 4.50 µV for medial branch and 9.57 ± 4.39 µV for intermediate branch) and control groups (13.58 ± 4.92 µV for medial branch and 11.77 ± 5.97 µV 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 ±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. The nerve action potential may be absent in up to 2-9 % of the population. The SPN conduction study has been used to assess the SPN lesion in various conditions. Izso 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>
<thead>
<tr>
<th>Subjects</th>
<th>Number of Subjects</th>
<th>Number of Nerve</th>
<th>Distal Latency (ms)</th>
<th>Amplitude (µV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>20</td>
<td>40</td>
<td>3.10 ± 0.23</td>
<td>3.08 ± 0.25</td>
</tr>
<tr>
<td>Bicyclist</td>
<td>17</td>
<td>34</td>
<td>3.38 ± 0.30</td>
<td>3.34 ± 0.35*</td>
</tr>
<tr>
<td>t</td>
<td></td>
<td></td>
<td>4.40</td>
<td>3.66</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td></td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Int. Br.</td>
<td></td>
<td></td>
<td>11.54 ± 4.50</td>
<td>9.57 ± 4.39*</td>
</tr>
<tr>
<td>P</td>
<td></td>
<td></td>
<td>P &gt; 0.05</td>
<td>P &gt; 0.05</td>
</tr>
<tr>
<td>Med. Br.</td>
<td>: Medial branch of superficial peroneal nerve.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Int. Br.</td>
<td>: Intermediate branch of superficial peroneal nerve.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*</td>
<td>: The nerve number is only 33 for the intermediate branch, because one nerve could not be detected.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1. Effect of bicycle exercise on superficial peroneal nerve conduction
Table 2. Bicyclists with abnormal distal latencies of superficial peroneal sensory nerve

<table>
<thead>
<tr>
<th>Case #</th>
<th>Right side</th>
<th>Left side</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MB</td>
<td>IB</td>
</tr>
<tr>
<td>2</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>4</td>
<td>N</td>
<td>3.59</td>
</tr>
<tr>
<td>6</td>
<td>N</td>
<td>3.87</td>
</tr>
<tr>
<td>7</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>9</td>
<td>3.68</td>
<td>3.87</td>
</tr>
<tr>
<td>10</td>
<td>3.87</td>
<td>3.59</td>
</tr>
<tr>
<td>12</td>
<td>3.89</td>
<td>N</td>
</tr>
<tr>
<td>13</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>14</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>15</td>
<td>3.76</td>
<td>N</td>
</tr>
<tr>
<td>17</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

MB—Medial branch of superficial peroneal sensory nerve. IB—Intermediate branch of superficial peroneal sensory nerve.

*Case 10 was the subject with clinical symptom.

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 [31]. Yasusuke described members of a mountain climbing club had tarsal tunnel syndrome due to persistent and repetitive reciprocal movements of ankle dorsal and plantar flexion [33]. 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 [33]. 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.

REFERENCES

南台灣騎自行車者之潛在性腓淺感覺神經病變

程璇敏  陳若佟  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運動。本研究是以 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各條分枝(包括中間及遠端中間真皮神經的複合動作電位)而刺激腓淺神經的部位是在記錄表面電極遠端 12 公分處。

結果顯示：控制組各腓淺感覺神經兩分枝-足背內側及足背中間真皮神經的遠端潛期平均值，分別為 3.10±0.23ms 及 3.08±0.25ms；而自行車騎士此組的平均值則分別為 3.38±0.30ms 及 3.34±0.35ms；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真皮神經的平均值，分別為 13.58±4.92μV 及 11.77±3.97μV；而自行車騎士此組的平均值則分別為 11.54±4.50μV 及 9.57±4.39μV；兩組間的差異並無統計學上的意義(p>0.05)。另外，回顧腓淺神經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%)無法測得足背中間真皮神經(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。至於固定腳踏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)