Comparison of median and ulnar sensory nerve action potentials in the diagnosis of the carpal tunnel syndrome

S. C. LOONG AND C. S. SEAH

From the Medical Unit, Thomson Road General Hospital, Singapore

SUMMARY Recording of median and ulnar digital sensory nerve action potentials in normal subjects showed that the ratio of the median (index finger) to ulnar (little finger) potential amplitude was consistently greater than one. In 15 patients with the carpal tunnel syndrome (seven bilateral) this ratio was found to be less than one for all but two of the 22 clinically affected hands, including three of the four hands with a normal motor latency to threshold stimulation and four of the five hands with a normal sensory conduction. It is concluded that the estimation of the ratio of the median to ulnar sensory potential amplitude is a sensitive test in the diagnosis of the carpal tunnel syndrome and is particularly useful in those patients who show a normal motor latency and sensory conduction.

Since the first description of the carpal tunnel syndrome (Brain, Wright, and Wilkinson, 1947), various techniques have been employed in the diagnosis of this condition. These currently include the measurement of distal motor latency (Simpson, 1956; Preswick, 1963) and the estimation of digital sensory conduction and amplitude (Gilliatt and Sears, 1958). While the diagnostic value of these methods is considerably enhanced by their combined application there still remains a group of patients, particularly those with minor, intermittent symptoms, who escape confirmation (Preswick, 1963; Fullerton and Gilliatt, 1965).

In the course of establishing normal conduction values for the authors' laboratory, it was observed that the amplitude of the 'median' digital sensory nerve action potential recorded at the wrist upon stimulation of the index finger was consistently greater than that of the 'ulnar' potential evoked by stimulation of the little finger of the same hand. This prompted the authors to investigate the probable disturbance of this relationship between the median and ulnar sensory potentials and its diagnostic significance in the carpal tunnel syndrome.

METHODS

The 15 patients used in this study were referred for electromyography with a clinical diagnosis of carpal tunnel syndrome. In seven the condition was bilateral; thus 22 clinically affected hands were examined. All the patients were female with an age range of 31 to 60 years. All of them gave a typical history of the syndrome with intermittent paraesthesia occurring spontaneously at night or after use of the affected hand. None had any underlying disease such as diabetes. One patient (case 9) was in the eighth month of her pregnancy when she presented. The relevant clinical data are summarized in Table 1.

All examinations were performed in a room with a constant ambient temperature of 23 to 25°C. Surface electrodes were used for stimulation and recording and the overall technique was essentially similar to that previously described (Dawson, 1956; Gilliatt and Sears, 1958; Preswick and Jeremy, 1964). The index finger was stimulated for the median nerve and the little finger for the ulnar nerve; great care was taken to prevent spread of stimulus to the adjacent digits by covering them with a non-conducting adhesive plaster, as it was found that a spread of stimulus through contact of stimulating electrodes with the adjacent digits could significantly increase the size of the sensory potential in some individuals. The electrical stimulus, with a duration of 0.1 to 0.4 msec and an amplitude up to 200 V, was delivered by two Devices isolated stimulators. The muscle and nerve action potentials were amplified by a Tektronix type 122 preamplifier and displayed for Polaroid photography on a Tektronix type 564B storage oscilloscope. For the purpose of estimation of sensory conduction velocity, the sensory latencies were measured from the stimulus artefact to the onset of the first negative deflection. However, sensory latencies to the peak of the main (negative) deflection were also recorded. The amplitudes...
TABLE 1

CLINICAL DETAILS AND ELECTROPHYSIOLOGICAL FINDINGS IN PATIENTS

<table>
<thead>
<tr>
<th>Sex and age yrs.</th>
<th>Right</th>
<th>Left</th>
<th>Duration of symptoms (yr.)</th>
<th>Symptoms</th>
<th>Signs</th>
<th>Clinical data</th>
<th>Electrophysiological findings</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Par-aesthesia</td>
<td>Weakness</td>
<td>Sensory (APB)</td>
<td>Median motor latency</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>Threshold stim. (msec)</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>F 40</td>
<td>R</td>
<td>L</td>
<td>2/12</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>6-9</td>
</tr>
<tr>
<td>F 42</td>
<td>R</td>
<td>L</td>
<td>6/12</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>6-7</td>
</tr>
<tr>
<td>F 50</td>
<td>R</td>
<td>L</td>
<td>6/12</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>6-0</td>
</tr>
<tr>
<td>F 59</td>
<td>R</td>
<td>L</td>
<td>2/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>6-4</td>
</tr>
<tr>
<td>F 37</td>
<td>R</td>
<td>L</td>
<td>6/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>5-2</td>
</tr>
<tr>
<td>F 60</td>
<td>R</td>
<td>L</td>
<td>2/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>5-2</td>
</tr>
<tr>
<td>F 35</td>
<td>R</td>
<td>L</td>
<td>3/12</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>6-8</td>
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<tr>
<td>F 37</td>
<td>R</td>
<td>L</td>
<td>3/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>5-0</td>
</tr>
<tr>
<td>F 60</td>
<td>R</td>
<td>L</td>
<td>3/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>F 31</td>
<td>R</td>
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<td>-</td>
<td>+</td>
<td>7-0</td>
</tr>
<tr>
<td>F 42</td>
<td>R</td>
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<td>+</td>
<td>-</td>
<td>4-3</td>
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<tr>
<td>F 45</td>
<td>R</td>
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<tr>
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<td>R</td>
<td>L</td>
<td>3/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<tr>
<td>F 43</td>
<td>R</td>
<td>L</td>
<td>1/12</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>6-1</td>
</tr>
</tbody>
</table>

Mal controls: Mean ± 1 SD

|                  |       |      |                           |       |      |               | 4-36 ±                     | 3-67 ±                    | 25-55 ±                  | 2-82 ±                  | 2-16 ±                  | 52-3 ±                  | 19-98 ±               | 1-51 ±               |
|                  |       |      |                           |       |      |               | (4-4-5-1)                  | (3-1-4-6)                 | (9-48)                  | (2-2-3-6)               | (1-1-2-8)               | (44-59)                 | (7-5-33)              | (1-1-2-4)             |

(Plot (m/sec) | (m/sec) | (μV) | (msec) | (μV) | (m/sec) | (μV) | (m/sec) | (μV) | (m/sec) | (μV) |

of the sensory potentials were measured from peak to peak; at least five successive maximum action potentials were recorded (Figure) and if there was any variation, the average of their amplitudes was taken. Apart from sensory studies, motor responses evoked by threshold as well as supramaximal stimulation at the wrist were recorded from the abductor pollicis brevis muscle for motor latency measurement.

Similar studies were performed on 30 healthy female subjects with an age range of 30 to 60 years.

RESULTS

CONTROL OBSERVATIONS ON HEALTHY SUBJECTS The results for the control hands are given in Table 2. The overall findings for the sensory potentials are in agreement with those reported by others.

The ratio of action potential amplitudes of the median (index finger) to ulnar (little finger) sensory nerves was greater than unity for all control hands, with a range of 1-1 to 2-4.

The motor latencies with threshold stimulation varied in different individuals from 3-4-5-1 msec with a mean of 4-36 msec (SD ± 0-41). With supramaximal stimulation, the range was 3-1 to 4-6 msec with a mean of 3-67 msec (SD ± 0-38). As these values were obtained with surface electrodes no strict comparison could be made with those by Preswick (1963).

PATIENTS WITH CARPAL TUNNEL SYNDROME The results of the patient group are given in Table 1. As is necessary when considering individual cases, the criterion of abnormality is that the value should lie outside the normal range.

Motor latency with supramaximal stimulation was abnormal in only 13 out of 22 hands, whereas that

TABLE 2

RESULTS FOR CONTROL HANDS COMPARED WITH TWO CONTROL SERIES

<table>
<thead>
<tr>
<th></th>
<th>Median sensory velocity (m/sec)</th>
<th>Median sensory latency (msec)</th>
<th>Median sensory amplitude (μV)</th>
<th>Ulnar sensory amplitude (μV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present series</td>
<td>44-59</td>
<td>2-2-3-6</td>
<td>9-48</td>
<td>7-5-33</td>
</tr>
<tr>
<td>Gilliatt and Sears (1958)</td>
<td>52-3 ± 4-1*</td>
<td>2-5-4-0</td>
<td>9-45</td>
<td>8-28</td>
</tr>
<tr>
<td>Preswick (1964)</td>
<td>50-2 ± 4-8</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

*Mean ± 1 SD. †Not available.
with threshold stimulation was abnormal in 18 out of 22 hands.

Median sensory nerve action potentials were absent in three hands. However, in the remaining hands only five had a sensory action potential with amplitude below the lower limit of normal. This clearly showed the limited diagnostic value of measuring absolute amplitude of sensory action potentials.

In the 19 hands in which median sensory action potentials were present, 12 had an abnormally prolonged sensory latency whether measurement was made to the onset or to the peak of the negative deflection. A slightly greater number of hands (14 out of 19) showed an abnormal result when sensory conduction was estimated in terms of metres per second. The amplitude ratio of the median (index finger) to ulnar (little finger) sensory action potential was below the lower limit of 1.1 in 17 out of the 19 hands; these included three of the four hands with a normal motor latency to threshold stimulation (cases 1—L, 7, 9—L, and 13—R) and four of the five hands with a normal sensory conduction (cases 1—L, 3—R, 6, 7, and 9—L). However, only two (cases 1—L and 7) of these 17 hands had both a normal motor latency and sensory conduction. That these two hands did not represent misdiagnoses was supported by their typical clinical picture and by their motor latencies and sensory conduction being at the lower limit of normal. The two hands in which the median/ulnar sensory ratio did not fall below the lower limit of normal were both the clinically less affected left hands of right-handed individuals (cases 9 and 15) with a bilateral syndrome.

The percentage of abnormal results obtained with the various diagnostic techniques is shown in Table 3.

**DISCUSSION**

The measurement of motor latency to supramaximal stimulation in the diagnosis of the carpal tunnel syndrome was first described by Simpson (1956). Although this remains the most convenient diagnostic test, its lack of sensitivity has been a recognized drawback. Simpson (1956) found an increased latency in 11 of 15 subjects with a clinical diagnosis of the syndrome. Similarly, in an unselected series of 95 consecutive cases with a diagnosis of suspected carpal tunnel syndrome, Thomas (1960) was able to demonstrate an abnormal latency to supramaximal stimulation in only two-thirds of them. The modified method with threshold stimulation by Preswick (1963) is a more sensitive diagnostic procedure (Fullerton and Gilliatt, 1965) and this has been further confirmed in the present study employing only surface electrodes.

While sensory conduction is expected to be altered in those cases with predominantly sensory symptoms, the diagnostic value of its estimation...
is lessened somewhat by its wide normal range, while comparison with contralateral median conduction is not possible in patients with a bilateral syndrome. Similarly, owing to considerable variation in sensory amplitude in different individuals—such variation being as great with needle as with surface electrodes (Buchthal and Rosenfalck, 1966)—the measurement of the median sensory action potential amplitude per se cannot be expected to have great diagnostic sensitivity, particularly in the early cases.

Gilliatt and Sears (1958) noted in their controls that sensory action potentials recorded from the ulnar nerve at the wrist were generally smaller than those from the median nerve at the wrist. However, no specific mention was made of their relationship in individual subjects and of prevention of spread of stimulus to adjacent digits. The present study shows that the amplitude of the median sensory action potential recorded at the wrist is consistently greater than that of the ulnar action potential in the same hand of normal subjects when the stimulus is confined to the index and the little finger, respectively. This consistency was observed despite possible experimental errors in peak-to-peak measurement and variation of responses secondary to difference in distance from the nerves. That this median (index) to ulnar (little finger) sensory action potential amplitude ratio is greater than one is to be expected in view of the fact that the index finger is the larger and functionally more important digit and presumably carries more sensory fibres; Buchthal and Rosenfalck (1966), for example, have shown that the sensory threshold is significantly lower for the index finger than for the other digits. It is noteworthy that the median/ulnar sensory action potential amplitude ratio was reduced to below one ("reversed") in all but two of the 22 clinically affected hands, including three of the four hands with a normal motor latency to threshold stimulation and four of the five hands with a normal sensory conduction. This indicates that, as a diagnostic procedure, the estimation of the median/ulnar sensory action potential amplitude ratio has a greater sensitivity than that of motor latency measurement or sensory conduction estimation alone. However, it suffers from the disadvantage that this sensitivity is expected to be reduced when it is performed on patients with an associated ulnar nerve lesion in the same hand.

The recognition of the carpal tunnel syndrome is important as disabling symptoms can be readily relieved by hydrocortisone injection or retinacular section. The diagnosis of the syndrome is easy when clinical involvement is limited to the distribution of the median nerve in the hand but becomes difficult when sensory symptoms are absent or spread to involve ulnar digits or to the elbow, or even up to the shoulder. In the latter instances, electrodiagnostic methods become particularly important. However, any one of the techniques discussed above may give a normal result despite clinical evidence to the contrary. Hence, more than one procedure may have to be employed. The present study indicates that the measurement of the ratio of median to ulnar sensory action potential amplitude is a useful addition to the battery of tests available for the diagnosis of the carpal tunnel syndrome and would be particularly of value in those patients who show a normal motor latency and sensory conduction.

We are grateful to the Lions Club (Host) International, Singapore, for their generosity which made possible the setting up of the EMG laboratory. We wish to thank the orthopaedic surgeons of the Outram Road General Hospital who referred all the patients used in this study.

REFERENCES


The October 1971 Issue

The October 1971 issue contains the following papers:


Mechanical properties of dystrophic mouse muscle. J. B. Harris and P. Wilson.


Serum creatine kinase studies in the detection of carriers of Duchenne dystrophy. R. C. Hughes, Dorothy C. Park, Mary E. Parsons, and M. D. O'Brien.

Myasthenia gravis occurring in twins. T. Namba, M. S. Shapiro, N. G. Brunner, and D. Grob.


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