AAEM CASE REPORT #2:
THE CARPAL TUNNEL SYNDROME

Mark A. Ross, M.D.
Jun Kimura, M.D.

Department of Neurology
University of Iowa College of Medicine
Iowa City, Iowa

and

Department of Neurology
Kyoto University School of Medicine
Kyoto, Japan

Reviewed and accepted by:
Education Committee of
American Association of Electrodiagnostic Medicine

Lawrence R. Robinson, M.D., Chair
Elaine S. Date, M.D.
Allen B. DevleschHoward, M.D.
Dale J. Lange, M.D.
Joseph Y. Matsumoto, M.D.
Jeffrey B. Palmer, M.D.
Ghazala Riaz, M.D.
Michael H. Rivner, M.D.
David K. Ryser, M.D.
Mohammad A. Saeed, M.D.

Copyright © May 1995
American Association of Electrodiagnostic Medicine
21 Second Street S.W., Suite 103
Rochester, MN 55902

The ideas and opinions contained in this case report are solely those of the authors and do not necessarily represent those of the AAEM.
CME STUDY GUIDE

AAEM CASE REPORT #2:
THE CARPAL TUNNEL SYNDROME

Mark A. Ross, M.D.
Jun Kimura, M.D.

CERTIFYING ORGANIZATION

The American Association of Electrodiagnostic Medicine (AAEM) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education (CME) for physicians and certifies that this CME activity was planned and produced in accordance with ACCME Essentials.

The AAEM has determined that the estimated study time for completing this CME activity is one hour and designates this CME activity for one credit hour in Category 1 of the Physician’s Recognition Award of the American Medical Association.

EDUCATIONAL OBJECTIVES

This case report provides a review of the clinical and electrophysiologic features of carpal tunnel syndrome (CTS) and describes the electrodiagnostic protocol used in one laboratory for evaluation of suspected CTS. Studying this case report should familiarize the reader with the electrodiagnostic techniques used for assessment of CTS, the common electrodiagnostic abnormalities found in CTS, and the clinical significance of such abnormalities for patients with CTS.

INSTRUCTIONS

1. The reader should carefully and thoroughly study this case report. If further clarification is needed, the references should be consulted. Do not neglect illustrative material.

2. Read the CME questions at the end of the case report. Choose the correct answer to each question and record it on the CME Registration form on the last page. Retain a copy of your answers for your records.

3. Complete the Evaluation Form on the reverse side of the CME Registration form.

4. After completing the CME Registration and Evaluation forms, mail with a stamped, self-addressed envelope to the AAEM office as indicated.

5. Correct answers to the CME questions and a certificate of CME credit earned will be mailed to you.

6. Review those parts of the article dealing with the question(s) you answered incorrectly, and read the supplemental materials on this aspect of the subject listed in the references.
AAEM CASE REPORT #2:
THE CARPAL TUNNEL SYNDROME

Mark A. Ross, M.D.
Jun Kimura, M.D.

Department of Neurology
University of Iowa College of Medicine
Iowa City, Iowa
and
Department of Neurology
Kyoto University School of Medicine
Kyoto, Japan


Median nerve entrapment in the wrist-to-palm segment produces a clinical condition known as the carpal tunnel syndrome (CTS). Patients with CTS typically complain of numbness in any of the lateral four digits of the involved hand; however, all digits may be affected. Pain is a common symptom which usually involves the wrist and hand but may also be perceived more proximally in the forearm, arm, or shoulder. Pain and numbness are often aggravated by use of the hands, and patients characteristically awaken at night complaining of these symptoms. Sensory loss, when present, usually affects the volar and the distal dorsal aspect of the lateral three-and-one-half digits, although variations are common. More severe cases develop weakness, and advanced cases have atrophy of median-innervated thenar muscles. The present case demonstrates the utility of electrophysiologic evaluation of suspected CTS.

She was awakened by pain in her left wrist and paresthesias involving the lateral three digits. She denied weakness or loss of sensation. Her past medical history was unremarkable. Her referring physician diagnosed CTS and requested evaluation with electrodiagnostic studies when conservative therapy with a wrist splint and nonsteroidal antiinflammatory medication failed to improve her symptoms.

Physical Examination. The general physical examination was normal. There was no weakness or atrophy of the thenar muscles. Muscle stretch reflexes were normal. Sensation to pinprick was diminished on the volar surface of the second and third digits of the left hand but was otherwise intact. On her left side, Phalen's sign was present, and Tinel's sign was elicited over the median nerve at the wrist.

ELECTRODIAGNOSTIC EXAMINATION

Methods. The following is a description of the protocol used in our laboratory for evaluation of suspected CTS. Nerve conduction studies (NCS) are performed with the subject supine in a warm room. Forearm skin temperature is measured and, if below 32°C, the limb is warmed to this level, or above, before beginning the study. The filter bandpass settings are 2 Hz to 10 kHz for motor
studies and 20 Hz and 2 kHz for sensory studies. For motor and sensory responses, latency is measured to the initial negative response and amplitude is measured from baseline to negative peak. Median motor and sensory fibers are routinely studied bilaterally to allow comparison of the nerve conduction parameters between the two sides.

The compound muscle action potential (CMAP) is recorded from the thenar eminence with the active recording electrode placed over the abductor pollicis brevis (APB) muscle belly, at the proximal one third of the distance between the carpometacarpal and metacarpophalangeal (MCP) joints of the thumb. The reference electrode is placed over the APB tendon at the MCP joint. The antidromic sensory nerve action potential (SNAP) is recorded from the second digit with ring electrodes around the proximal (active) and distal (reference) interphalangeal joints.

We use surface electrodes to stimulate the median nerve at the wrist, palm, elbow, and axilla in this order. The cathode is placed 2 cm distal to the anode at all stimulus sites except for palm motor stimulation which is discussed below. The wrist stimulus site is located 5 cm proximal to the distal wrist crease between the tendons of flexor carpi radialis and palmaris longus muscles. The palm stimulus site for sensory fibers is in the middle, 8 cm distal to the wrist stimulation site.

To stimulate median motor fibers in the palm, that is, the recurrent thenar nerve, the cathode is initially placed at the same site as for palm sensory fibers. Unlike other stimulus sites, however, the anode is directed distally toward the base of the fifth digit. This is done to avoid depolarizing the recurrent thenar nerve beneath the anode. When stimulating the median nerve in the palm, one must carefully observe the nature of the thenar twitch, as stimulation of the deep branch of the ulnar nerve can also generate a motor response recorded over the thenar eminence. If an adduction thenar twitch occurs due to ulnar nerve stimulation, the stimulating electrodes are repositioned in roughly millimeter increments toward the thenar eminence until an abduction twitch is achieved, indicating median nerve stimulation. In addition to analysis of thenar twitch, the median CMAP waveforms from palm and wrist stimulation sites are also compared. A change in the CMAP area or waveform configuration between these sites indicates a different population of nerve fibers has been stimulated. This may occur for a variety of reasons summarized in Table 1.

The elbow stimulus site is on the anterior sur-
<table>
<thead>
<tr>
<th>CMAP area or amplitude*</th>
<th>Possible causes</th>
<th>Confirmation</th>
</tr>
</thead>
</table>
| Palm = wrist            | 1. Normally the CMAP amplitudes with palm and wrist stimulation are approximately equal.  
2. Axon loss also causes equal amplitudes with palm and CMAP wrist stimulation. | Compare CMAP amplitudes between sides. In unilateral carpal tunnel syndrome, axon loss causes reduced amplitude on affected side. Needle exam may reveal fibrillation potentials or abnormal motor unit potentials. |
| Wrist > palm            | 1. Submaximal median nerve stimulation in palm.  
2. Coactivation of median and ulnar nerves at wrist. | Adjust location of palm stimulus or increase intensity of stimulation in palm. Move stimulating electrodes laterally away from ulnar nerve or decrease stimulus intensity at wrist. |
| Palm > wrist            | 1. Submaximal median nerve stimulation at wrist.  
2. Coactivation of the deep ulnar nerve and recurrent median nerve in the palm. | Increase stimulus intensity at wrist. When technical factors (e.g., edema, scarring) at wrist prevent supramaximal nerve stimulation, the amplitude with elbow stimulation should be comparable to palm. |
|                         | 3. True conduction block                                                        | Pathologic conduction block is confirmed by an abrupt change in amplitude or area over a short nerve segment with incremental stimulation. Prolonged latency and/or temporal dispersion of wrist and more proximal CMAP waveforms provide supportive evidence. |

*CMAP area provides the best measure of the number of muscle fibers contributing to the response. Amplitude may be used, provided CMAP waveform configuration and duration are similar at each stimulus site.

overlap. Furthermore, several series have shown coexistence of CTS and cervical radiculopathy.¹³,¹²

Nerve Conduction Studies. Nerve conduction study results are shown in Table 2. The amplitude of median CMAPs are normal bilaterally at all stimulus sites. The 4-ms left median CMAP latency with wrist stimulation was within normal absolute limits, but the 0.8-ms prolongation relative to the right side exceeded the upper limit of normal. Median SNAPs with palm stimulation were 50 μV bilaterally, but with wrist stimulation, the left SNAP amplitude was reduced by 25 μV compared to the right. The 8.3-ms left median SNAP latency with wrist stimulation was within normal absolute limits, but excessively prolonged relative to the right side. Conduction velocities of the left median nerve wrist segment were slowed to 39 m/s for motor fibers and 41 m/s for sensory fibers. An inching study revealed a SNAP latency increase exceeding 0.5 ms/cm, associated with a 50% amplitude reduction at the “—3” level (Fig. 1A). Nerve conduction studies of the left ulnar nerve were normal.

**Needle Electromyography.** Needle examinations of the left abductor pollicis brevis, first dorsal interosseous, flexor carpi radialis, and biceps muscles were performed. No fibrillation potentials were seen. Motor unit action potentials and recruitment were normal.

**Interpretation.** The electrodiagnostic studies revealed a left median mononeuropathy involving the wrist segment. The inching study precisely localized the site of abnormality to the “—3” level, corresponding anatomically to the distal edge of the transverse carpal ligament. The clinical features suggested primarily median sensory nerve involvement. This was verified by prolongation of the left median SNAP wrist latency relative to the right, focal slowing of the left median sensory conduction velocity across the wrist segment, and the inching study. The amplitude of the left median SNAP with palm stimulation was normal and equal to the unaffected side, speaking against axonal loss of sensory fibers. In contrast, the reduced amplitude median SNAP with wrist stimulation sug-
FIGURE 1. (A) Preoperative inching study. Sensory nerve action potentials (SNAPs) are recorded antidromically from the index finger with stimulation of the median nerve in 1-cm increments across the wrist. The zero level is marked at the distal wrist crease. The bar graph shows the latency difference in milliseconds between each stimulus site. Between the -3 and -4 levels, an abnormally large latency difference is seen as well as a reduction in SNAP amplitude at the -3 level. (B) Postoperative inching study. Inching study repeated 10 months after carpal tunnel release surgery revealed normal SNAP latency difference between each stimulus site and normal SNAP amplitude at each site.

suggested conduction block of approximately 50% of left median sensory fibers. This finding, in the context of chronic stable symptoms, suggested a demyelinating nerve injury.

The normal left median CMAP amplitude and normal needle examination of median-innervated muscles argued against axonal degeneration of median motor fibers. The relatively prolonged left median motor wrist latency, and the relative focal slowing of left median motor conduction velocity across the wrist segment, suggested a partial demyelinating nerve injury of left median motor fibers.

<table>
<thead>
<tr>
<th>Fiber type</th>
<th>Stimulus site</th>
<th>Normal* (mV or µV)</th>
<th>Patient Latency</th>
<th>Latency difference†</th>
<th>Conduction velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Normal† (ms)</td>
<td>Patient Right</td>
<td>Patient</td>
</tr>
<tr>
<td>Motor</td>
<td>Palm (P)</td>
<td>&gt;3.5 mV</td>
<td>8.0</td>
<td>7.5</td>
<td>&lt;2.4</td>
</tr>
<tr>
<td></td>
<td>Wrist (W)</td>
<td>&gt;3.5 mV</td>
<td>8.0</td>
<td>7.5</td>
<td>&lt;4.2</td>
</tr>
<tr>
<td></td>
<td>Elbow (E)</td>
<td>&gt;3.5 mV</td>
<td>8.0</td>
<td>7.5</td>
<td>&lt;6.6</td>
</tr>
<tr>
<td>Sensory</td>
<td>Palm (P)</td>
<td>&gt;19 µV</td>
<td>50</td>
<td>50</td>
<td>&lt;1.9</td>
</tr>
<tr>
<td></td>
<td>Wrist (W)</td>
<td>&gt;19 µV</td>
<td>50</td>
<td>25</td>
<td>&lt;3.5</td>
</tr>
<tr>
<td></td>
<td>Elbow (E)</td>
<td>&gt;16 µV</td>
<td>30</td>
<td>15</td>
<td>&lt;7.9</td>
</tr>
</tbody>
</table>

Table 2. Median nerve conduction study results.

*Lower limit normal (based on distribution of normative data).
†Upper limit of normal (mean ± 2 SD).
‡Lower limit of normal (mean ± 2 SD).
§Latency difference between right and left sides.
within the carpal tunnel, producing slowing of conduction velocity without conduction block.

**CLINICAL COURSE**

Because the patient's symptoms limited her ability to work, and conservative treatment failed, she underwent surgical sectioning of the left transverse carpal ligament. When reevaluated 2 weeks after surgery, she was asymptomatic. Median nerve conduction velocity across the wrist was 30 m/s for motor fibers and 44 m/s for sensory fibers. At 10 months after surgery, she remained free of symptoms. Median nerve conduction velocity across the wrist was normal at 57 m/s for motor fibers and 62 m/s for sensory fibers. An ensuing study at this time was also normal (Fig. 1B).

**DISCUSSION**

The electrophysiology of CTS has been extensively studied since the first description by Simpson in 1956. Many techniques have been developed for identifying abnormal median nerve function within the carpal tunnel. These include measuring distal motor and sensory latencies of the median nerve, calculating conduction velocity across the wrist segment; comparing the distal motor latency of the median and ulnar nerves; comparing the sensory latency of the median nerve with that of the radial ulnar or median palmar cutaneous nerve; serial recording of median SNAP latencies in 1-cm increments across the wrist; comparing the median nerve terminal latency with the conduction time in the proximal segment adjusted to equal distance; and comparing the median and ulnar orthodromic sensory latencies with digit or palm stimulation. Presently, there is no consensus regarding which single technique is best for detecting CTS. Studies comparing several techniques have reached different conclusions.

A recent review of the literature concerning electrodiagnostic studies in CTS has provided general recommendations.

It is generally stated that sensory nerve testing is more sensitive than motor in CTS. If CTS is mild, then sensory NCS will be most helpful diagnostically, as sensory fibers are typically involved earlier and to a greater extent than motor fibers. If CTS is severe enough to cause absent median SNAPs, as in a high percentage of cases in some series, the sensory NCS do not localize the median neuropathy to the carpal tunnel, and electrodiagnosis of CTS depends upon abnormal motor conduction studies. Rarely, CTS may selectively involve motor fibers. An anatomical basis exists for this distinctly unusual circumstance, as the motor branch exits the carpal tunnel in a separate channel. Thus, median NCS for CTS should include assessment of both motor and sensory fibers. In our experience, there has been a roughly even distribution of abnormalities between sensory and motor median NCS in patients with CTS, and it is far more common to find abnormalities of both sensory and motor fibers than isolated involvement of one fiber population.

We believe that performing palm stimulation is essential. Palm stimulation increases the sensitivity of detecting CTS compared to using conventional terminal latencies from the wrist. In a series of 172 symptomatic hands previously reported from our laboratory, palm stimulation allowed detection of focal slowing of median nerve conduction velocity across the wrist in 32 hands (19%), which would have been regarded as normal by assessment of conventional terminal latencies. This increased sensitivity results from separation of the relatively normally conducting palm-to-digit segment from the slowly conducting wrist-to-palm segment. Measuring conventional terminal latencies from the wrist combines these segments, allowing the normal palm-to-digit segment to mask a mild abnormality in the wrist-to-palm segment.

By comparing median sensory nerve conduction velocity in the wrist-to-palm and palm-to-digit segments, palm stimulation can also help distinguish CTS from polyneuropathy. The latter condition often has slow conduction in the palm-to-digit segment, whereas this segment usually conducts normally in CTS. Finally, including palm stimulation allows detection of conduction block in the wrist-to-palm segment. Conduction block is suspected when the CMAP amplitude with palm stimulation exceeds that of wrist stimulation. A proximal-to-distal CMAP amplitude ratio of less than 0.7 has generally been said to indicate conduction block. Our laboratory uses a ratio of less than or equal to 0.5 which is a conservative figure. Lesser degrees of block may be accepted as abnormal if accompanied by other features suggesting demyelinating pathology, such as prolonged latency or temporal dispersion. Conduction block is confirmed by finding an abrupt change in amplitude or area over a short nerve segment with incremental stimulation. Conduction block of the median motor fibers at the wrist should never be diagnosed unless inadvertent coactivation of the deep branch of the ulnar nerve in the palm has been excluded. Conduction block of sensory fibers
may be more difficult to confirm because physiologic temporal dispersion results in phase cancellation of some sensory fiber potentials and, consequently, a normal decline in amplitude of antidromic SNAPs with more proximal stimulation.24 Nevertheless, the amplitude of median SNAPs with palm and wrist stimulation is comparable in normal subjects,22,23 and a reduction of wrist SNAP amplitude by 50% or more compared to the palm SNAP suggests conduction block,16 provided technical factors can be excluded.

Reduction of median motor or sensory response amplitudes without evidence of conduction block or temporal dispersion suggests that axon loss has occurred. Significant axon loss may occur despite preservation of amplitude values well above the lower limit of normal. Thus, in unilateral CTS, the median CMAP and SNAP amplitude values should be compared with the uninvolved hand.

Electrodiagnostic studies give an estimation of the severity of the median neuropathy and the contribution of axonal versus demyelinating nerve injury. This information can be used to influence therapeutic decisions. A demyelinating nerve injury generally implies a good prognosis for recovery,31 providing the underlying condition causing neurapraxia can be alleviated. In contrast, electrodiagnostic evidence of axonal loss suggests a more advanced nerve injury, and is often viewed as an indication for surgical intervention. Thetanar atrophy and electrodiagnostic evidence of axonal injury can suggest a relatively poor prognosis,30 however, many patients with these features improve with surgery.17,29 Following surgery for CTS, electrodiagnostic studies typically improve,12,14,17,29 although complete normalization does not always occur.29

We believe electrodiagnostic studies are essential for evaluation of CTS. Documenting median neuropathy within the carpal tunnel and excluding an alternative or concomitant disorder should always be done before contemplating surgical therapy.

REFERENCES

24. Kimura J, Machida M, Ishida T, Yamada T, Rodnitzky RL, Kudo Y, Suzuki S: Relation between size of compound sen-