AANEM Case Report # 1
Ulnar Neuropathy at the Elbow

Robert G. Miller, MD
AAEM CASE REPORT #1: ULNAR NEUROPATHY AT THE ELBOW

Robert G. Miller, M.D.

Department of Neurology
Children's Hospital of San Francisco
San Francisco, California

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

William J. Litchy, M.D., Chair
Richard D. Ball, M.D., Ph.D.
William W. Campbell, Jr., M.D.
James M. Gilchrist, M.D.
John J. Halperin, M.D.
Gerald J. Herbison, M.D.
Susan L. Hubbell, M.D.
John C. Kincaid, M.D.
James J. Rechtien, D.O., Ph.D.
Lawrence R. Robinson, M.D.

Copyright © February 1991
American Association of Electrodiagnostic Medicine
(formerly the American Association of Electromyography and Electrodiagnosis)
21 Second Street S.W., Suite 306
Rochester, MN 55902

The ideas and opinions contained in this case report are solely those of the author and do not necessarily represent those of the AAEM.
CERTIFYING ORGANIZATION

As an organization accredited for continuing medical education, the American Association of Electrodiagnostic Medicine of Rochester, Minnesota, certifies that this continuing medical education activity meets the criteria for one credit hour at the Category 1 level provided it is used and completed as described in this study guide.

EDUCATIONAL OBJECTIVES

By studying the attached case report, the clinical electromyographer should gain a broader understanding of the clinical approach to ulnar neuropathies at the elbow.

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 and record it on the CME Registration form on the last page. Retain a copy of your answers for your records.

3. Fill in all the information requested on the form and mail with a stamped, self-addressed envelope to the AAEM office as indicated.

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

5. 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 #1:
ULNAR NEUROPATHY
AT THE ELBOW

Robert G. Miller, M.D.

Department of Neurology
Children’s Hospital of San Francisco
San Francisco, California

First Printing: June 1980
Revised: February 1991

Ulnar neuropathy at the elbow represents a common condition familiar to every clinical electromyographer. In many cases, the localization of the lesion in the ulnar nerve is difficult. Both clinical and electrophysiologic criteria for precise localization of an ulnar neuropathy at the elbow will be discussed.

CASE REPORT
Clinical History. The patient is a 60-year-old man with a chief complaint of aching pain in the right forearm of 8 months’ duration. The patient reports steady aching pain which involves the right forearm as well as the ulnar aspect of the right hand, which is increased while driving as a traveling salesman. In addition, he has noted numbness and tingling in the small finger of the right hand over the past 6 months. Finally, he has had increasing weakness of the right hand over the past 2 to 3 months.

Some 20 years previously, the patient developed similar symptoms in the left hand and forearm. There was no associated injury to the arm, neck, or shoulder; and the same symptoms developed over a period of 1 to 2 years, resulting in severe weakness of the left hand which has persisted until the present time. No evaluation or treatment was sought for that problem.

The patient denies any prior injuries to either arm, and there is no history of arthritis. The patient takes no medication, drinks only moderately, and denies any symptoms in the feet.

Examination. There was no joint deformity at the elbow. A Tinel’s sign could not be elicited at the elbow, but there was pain on compression at the cubital tunnel (2 to 3 cm distal to the medial epicondyle) on the right. Palpation of the ulnar nerve revealed an enlarged nerve in the ulnar groove bilaterally, which was markedly tender, taut, and immobile at 90° elbow flexion.

There was marked wasting of the hypothenar muscle group and of the first dorsal interosseous on the left, with moderate wasting of these muscles on the right. There was weakness of the first dorsal interosseous and abductor digiti minimi bilaterally (MRP grade 3 on the left and MRP grade 4 on the right) with mild weakness of the left flexor digitorum profundus of the fourth and fifth fingers and normal strength of the right flexor digitorum profundus. The flexor carpi ulnaris was normally strong, as were the remaining muscles of both arms. Sensation was decreased bilaterally in the fifth fingers and in the ulnar aspect of the fourth finger to both touch and pinprick, both dorsally and ventrally.
Laboratory Investigations. All blood and urine tests and screening for diseases associated with polyneuropathy gave normal results.

ELECTRODIAGNOSTIC EXAMINATION

Methods. Muscle sampling was carried out with a monopolar needle electrode. Ulnar nerve conduction studies were performed with the elbow extended and the arm abducted 30° from the trunk. Motor nerve conduction velocities were determined for each segment (forearm, across elbow and above elbow) with normal values provided in Table 1.

Needle Electromyography. The results of muscle sampling with a needle electrode are presented in Table 2. The findings indicate chronic partial denervation limited to ulnar-innervated muscles bilaterally. The abnormalities were mild in the flexor carpi ulnaris and more marked in the ulnar-innervated intrinsic hand muscles.

Nerve Conduction Studies. The results of motor and sensory nerve conduction studies are shown in Table 1. The sensory nerve action potential could not be obtained for either ulnar nerve, while a normal response was obtained for both the right median and sural nerves.

On the right side, the distal motor latency was normal for the ulnar nerve, with a normal motor nerve conduction velocity in the below-elbow and above-elbow segments of that nerve. The motor nerve conduction velocity was markedly slowed in the 10-cm segment across the elbow. The amplitude of the response recorded over the abductor digiti minimi (ADM) with surface electrodes was much smaller when stimulating above the elbow and at the axilla compared with stimulation below the elbow and at the wrist (Fig. 1). The response obtained from proximal stimulation, which was markedly desynchronized, increased in amplitude as the stimulator was inched from 2 to 6 cm distal to the medial epicondyle. While still recording at the ADM with surface electrodes, the median nerve was stimulated at the antecubital fossa, and a 2 mV response was recorded, thus establishing the presence of a median-to-ulnar nerve anastomosis in the forearm. Even after subtracting the contribution from the median nerve, however, the amplitude reduction across the cubital tunnel was still close to 50%.

On the left side, slowing was present in all segments of the ulnar nerve, and a low-amplitude response was obtained over the ADM when stimulating at all points along the nerve. The changes are those of a severe axonal neuropathy. The severe slowing in the forearm is compatible with nerve regeneration. Thus, the precise localization of this

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Amp</th>
<th>Stimulus site</th>
<th>Recording site</th>
<th>Latency (ms)</th>
<th>Distance (cm)</th>
<th>NCV (m/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensory Action Potentials</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R. Median</td>
<td>10 μV</td>
<td>Index finger</td>
<td>Wrist</td>
<td>3.4</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25 μV</td>
<td>Palm</td>
<td>Wrist</td>
<td>2.1</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>R. Ulnar</td>
<td>No Response</td>
<td>No Response</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. Ulnar</td>
<td>12 μV</td>
<td>Calf</td>
<td>Lat. mal</td>
<td>4.0</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>R. Sural</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor Nerve Conduction</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R. Ulnar</td>
<td>3.5 mV</td>
<td>Wrist</td>
<td>ADM</td>
<td>3.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.7</td>
<td>Below elbow</td>
<td></td>
<td>7.1</td>
<td>19.5</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>0.5 disp</td>
<td>Above elbow</td>
<td></td>
<td>11.5</td>
<td>10</td>
<td>23² (37)</td>
</tr>
<tr>
<td></td>
<td>0.5 disp</td>
<td>Axilla</td>
<td></td>
<td>13.6</td>
<td>12</td>
<td>57</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>Wrist</td>
<td>First dorsal int.</td>
<td></td>
<td>4.2</td>
<td></td>
</tr>
<tr>
<td>R. Median</td>
<td>1.5</td>
<td>Antecub. fossa</td>
<td>ADM</td>
<td>4.5³ (3.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L. Ulnar</td>
<td>0.7</td>
<td>Wrist</td>
<td>ADM</td>
<td>4.5³ (3.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.6</td>
<td>Below Elbow</td>
<td></td>
<td>16.2</td>
<td>19</td>
<td>16³ (49)</td>
</tr>
<tr>
<td></td>
<td>0.4</td>
<td>Above Elbow</td>
<td></td>
<td>20.2</td>
<td>10</td>
<td>25³ (37)</td>
</tr>
<tr>
<td></td>
<td>0.4</td>
<td>Axilla</td>
<td></td>
<td>22.8</td>
<td>12</td>
<td>46³ (49)</td>
</tr>
</tbody>
</table>

¹Shock artifact to peak of potential, surface electrodes, orthodromic stimulation.
²Shock artifact to start of potential, surface electrodes.
³Surface electrodes over ADM—normal amplitude >4.3 mV.
⁴Prolonged distal motor latency — upper limit of normal in parentheses.
⁵Slowed maximum motor nerve conduction velocity — lower limit of normal in parentheses.
⁶Evaluation of median-ulnar anastomosis.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Nerve</th>
<th>Root</th>
<th>Insertional activity</th>
<th>Postinsertional activity (PSW Fib Fascic other)</th>
<th>Motor unit action potentials</th>
</tr>
</thead>
<tbody>
<tr>
<td>L. Abductor pollicis brevis</td>
<td>Median</td>
<td>C8 T1</td>
<td>Normal</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>L. First dorsal intersosseous</td>
<td>Ulnar</td>
<td>C8 T1</td>
<td>↑</td>
<td>0</td>
<td>LLMU, ↓ ↓ ↓ ↓ IP, FR</td>
</tr>
<tr>
<td>L. Abductor digiti minimi</td>
<td>Ulnar</td>
<td>C8 T1</td>
<td>↑</td>
<td>0</td>
<td>Few LLMU, ↓ ↓ IP</td>
</tr>
<tr>
<td>L. Flexor carpi ulnaris</td>
<td>Ulnar</td>
<td>C7 8</td>
<td>Normal</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>L. R cervical paraspinai</td>
<td>C5 T1</td>
<td>Normal</td>
<td>0</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>R. Abductor pollicis brevis</td>
<td>Median</td>
<td>C8 T1</td>
<td>Normal</td>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>R. First dorsal intersosseous</td>
<td>Ulnar</td>
<td>C8 T1</td>
<td>↑</td>
<td>1 + Fibr, PSW</td>
<td>LLMU, ↓ ↓ ↓ ↓ IP</td>
</tr>
<tr>
<td>R. Abductor digiti minimi</td>
<td>Ulnar</td>
<td>C8 T1</td>
<td>↑</td>
<td>1 + Fibr, PSW</td>
<td>LLMU, ↓ ↓ ↓ ↓ IP</td>
</tr>
<tr>
<td>R. Flexor carpi ulnaris</td>
<td>Ulnar</td>
<td>C7 8</td>
<td>Normal</td>
<td>0</td>
<td>Few LLMU, ↓ ↓ IP</td>
</tr>
</tbody>
</table>

1Fibrillations, positive sharp waves, and fasciculations. ↑ indicates occasional, unsustained activity.
2LLMU = numerous large amplitude, long duration, polyphasic motor units.
3IP = interference pattern at maximum effort; ↓ ↓ = modest reduction; ↓ ↓ ↓ = marked reduction; ↓ ↓ ↓ ↓ = discrete, with preservation of a portion of the baseline.
4FR = only a few units seen, long at high rates of discharge (20 to 30 imp/s), indicating a good effort.

Lesion is not possible on the basis of the conduction studies.

**Interpretation.** These findings indicate chronic partial denervation of ulnar innervated muscles bilaterally. Ulnar neuropathy is present on both sides, although precise localization to the cubital tunnel was possible only on the right side (Fig. 1). Slowed conduction and a change in the amplitude and configuration of the evoked potential across the elbow indicate demyelination, while a reduced amplitude of the evoked response when stimulating at the wrist indicates axonal degeneration more marked on the left side. Thus, there is evidence on the right of a focal neuropathy, with elements of conduction block and focal slowing at the elbow, as well as chronic axonal loss. On the left, the ulnar neuropathy is mainly an axonal loss lesion and not active, with conduction velocity slowing around the elbow but no conduction block.

**CLINICAL COURSE**

The patient was subsequently operated upon, and the findings at surgery included a swollen hyperemic ulnar nerve extending from 3 cm proximal to the medial epicondyle to 3 cm distal to the medial epicondyle at the point where the heads of the flexor carpi ulnaris formed a tight, thick fibrous band compressing the right ulnar nerve. Below this band and distally for approximately 2 cm, the nerve was quite narrow and pale. Intraoperative stimulation of the ulnar nerve after the fibrous band had been sectioned disclosed a marked change in amplitude and configuration of the response recorded at the ADM, as one inch from just proximal to the area of the band to just distal. Accordingly, simple decompression without transposition was performed. Three weeks following the operation, the pain in the forearm had completely resolved. One year following the operation, there had been definite improvement in strength in the ulnar-innervated hand muscles and sensation had returned to normal on the right side. There was only a rare fibrillation and fasciculation in ulnar-innervated hand muscles on the right with only a mild decrease in the interference pattern. Although the response to ulnar nerve stimulation above the elbow still produced a desynchronized response over the ADM, the amplitude difference between above-elbow and below-elbow stimulation was only 10% when allowing for the anastomosis.
DISCUSSION

This patient has definite evidence of an ulnar neuropathy at the elbow, both clinically and electrophysiologically. He also fulfills all of the features for the diagnosis of cubital tunnel syndrome, which are: (1) no evidence of joint deformity or prior trauma; (2) frequent occurrence of bilateral ulnar neuropathy; (3) a taut, palpably enlarged nerve in the ulnar groove; (4) electrophysiologic localization to the cubital tunnel; and (5) operative findings of a swollen, taut, hyperemic nerve distally limited by the proximal border of the aponeurosis joining the 2 heads of the flexor carpi ulnaris muscle. Although this patient denied habitual elbow leaning, he spends much of his time with his elbows flexed to 90° while driving, and even while sleeping with both hands under his pillow. The fact that the cubital tunnel narrows when the elbow is flexed is probably important in the development of the compression neuropathy.

Localization of the lesion in the right ulnar nerve is possible both on clinical and electrophysiologic grounds. Clinically, the flexor carpi ulnaris was spared, a fact which probably depends upon the internal fascicular anatomy of the nerve as well as the severity of the lesion. The branch to the flexor digitorum profundus muscle of the fifth finger exits the main nerve trunk distal to the aponeurosis, however, and this muscle was weak. Further, the nerve was enlarged and taut on elbow flexion. Operative observations confirmed constriction of the nerve in the cubital tunnel, with distal narrowing and proximal swelling and hyperemia.

Both in the clinical neurophysiology laboratory and intraoperatively, precise localization of abnormal motor nerve conduction was possible based upon electrophysiological evidence of focal demyelination. The presence of a markedly desynchronized, low-amplitude response, with marked slowing of the maximum motor nerve conduction velocity across the cubital tunnel, are all diagnostic of a demyelinating lesion. The slowing of motor nerve conduction velocity is particularly clear-cut in this case even with the elbow extended (normal values are lower with the elbow extended than with the elbow flexed). We now utilize a lower limit of normal of 50 m/s in all segments of the ulnar nerve when the elbow is fully flexed (angle between the humerus and radius = 45°). We believe this position to be the best for performing ulnar nerve conduction studies because it removes the artifactual underestimate of nerve length across the elbow when the arm is extended, which produces factitious slowing across the elbow in normal controls. The shape and size of the compound muscle action potential deserve particularly careful analysis in suspected ulnar neuropathy.

Dispersion occurs in normal peripheral nerve, and some decline in amplitude and increase in duration along different segments of the ulnar nerve is found in normal control subjects. We consider a response abnormal if the difference in amplitude of the evoked muscle action potential comparing above-elbow to below-elbow exceeds 10%, or if the difference in amplitude comparing above elbow with wrist exceeds 25%.

Similarly, a change in shape with dispersion or a marked reduction in amplitude (conduction block) is occasionally observed in the absence of conduction slowing. All of these factors, which reflect focal segmental demyelination, are helpful in the diagnosis of focal ulnar neuropathy. Pathologic changes in human ulnar neuropathy have been observed directly under the tendinous arch joining the heads of flexor carpi ulnaris, consisting of distorted internodes with bulbar swellings at one end of the internode and thinning of the myelin sheath with paranodal demyelination at the other. The displacement of myelin away from the aponeurosis suggests intermittent or recurrent pressure under the edge of that ligament.

On the left side, there was pronounced slowing of conduction in the forearm and across the elbow, compatible with either nerve regeneration or demyelination. There was also evidence of axonal degeneration. The low-amplitude response recorded over ADM on the left indicates a substantial loss of motor units in that muscle, a finding that was confirmed by electromyography. On the right side, the amplitude of the compound muscle action potential over ADM was diminished in response to stimulation at the wrist (5.5 mV, normal greater than 4.3 mV). On the basis of this finding, one can conclude that there has been some degree of axonal degeneration with loss of motor units. In this case, there is evidence of coexistent pathology, segmental demyelination, and Wallerian degeneration on the left and predominant demyelination on the right.

A number of points, with respect to the technique of performing ulnar nerve conduction studies, deserve comment. When the standard motor nerve conduction study with the elbow flexed is normal recording over abductor digiti minimi, it is frequently worthwhile to repeat the study recording over first dorsal interosseous or to record from both muscles simultaneously. It does appear
that clinical evidence of weakness, as well as electromyographic evidence of denervation and conduction block, are more frequent in the first dorsal interosseous muscle. It could be argued that if a single muscle is to be studied, it should be the first dorsal interosseous muscle. Furthermore, sensory conduction studies utilizing antidromic recording further increase the sensitivity of electrodagnosis in ulnar neuropathy. Here again, the lower limit of conduction velocity for the sensory studies is 50 m/s across the elbow. A change in amplitude from above-elbow to below-elbow exceeding 8 µV or 43% of the amplitude of the sensory nerve action potential is considered abnormal. It may also be useful to compare the elbow segment with the forearm segment in motor conduction velocity since a difference exceeding 14 m/s is abnormal. The inching technique may be useful for precise localization of an ulnar neuropathy at the elbow, since some lesions may be either proximal or distal to the cubital tunnel, a point that is important in surgical treatment. Finally, the results of needle examination may be helpful in localization. Abnormalities in flexor carpi ulnaris or flexor digitorum profundus localize the lesion at or proximal to the elbow, although in many patients these muscles are normal, particularly in mild ulnar neuropathy.

Using all of these techniques, it may be possible to approach the sensitivity in electrodagnosis reported by Payan who was able to place the lesion at the elbow in 96% of cases with clinical weakness. The evaluation of this patient was complicated by the presence of a Martin-Gruber anastomosis, said to be present in approximately 15% of the general population. Whenever a lower amplitude response is obtained from proximal compared with distal stimulation, the presence of the anastomosis, as well as the possibility of submaximal proximal stimulation, must be considered before concluding that a demyelinating lesion is present. In this case, both the slowing of maximum motor nerve conduction across the elbow, as well as the dispersed response obtained from proximal stimulation, suggested the presence of abnormal motor nerve conduction in addition to the anastomosis.

The natural history of the mild cubital tunnel syndrome (sensory symptoms and no weakness) with documented spontaneous improvement in 27 of 30 patients, warrants conservative treatment for such patients. On the other hand, when there is progressive weakness, especially in conjunction with electrophysiologic evidence of partial denervation or conduction block, few would oppose surgical treatment. For the cubital tunnel syndrome, simple decompression of the nerve in the tunnel, without transposition, is a logical and effective form of therapy. Whether decompression is also an appropriate treatment for tardy ulnar palsy, as some have suggested, remains to be proven.

REFERENCES