AAEM MINIMONOGRAPH #31:
THE ELECTRODIAGNOSIS OF ULNAR NEUROPATHY AT THE ELBOW

John C. Kincaid, M.D.
CME STUDY GUIDE

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EDUCATIONAL OBJECTIVES

The objectives of this report on the electrodiagnosis of ulnar neuropathy at the elbow are:
1. To familiarize the reader with the pertinent anatomy of the nerve in the elbow region.
2. To define the known pathology and pathogenesis of elbow lesions.
3. To review the available electrodiagnostic techniques.
4. To review the pattern of electrophysiologic abnormalities encountered in patients with elbow lesions.
5. To discuss the differential diagnosis of ulnar lesions.

INSTRUCTIONS

1. The reader should carefully and thoroughly study this minimonograph. 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 minimonograph. 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 minimonograph dealing with the question(s) you answered incorrectly, and read the supplemental materials on this aspect of the subject listed in the references.
Patients are frequently referred to the EMG laboratory for evaluation of suspected ulnar neuropathy at the elbow. In performing this evaluation the electromyographer undertakes to determine whether a lesion is present, to localize it along the nerve, and to gauge its severity. Localization is important, because the ulnar nerve is liable to injury at the wrist, elbow, and in the upper arm. Lower trunk plexus and root level lesions also may present similarly and must be differentiated.

**Anatomy.** The ulnar nerve is derived from the C8 and T1 nerve roots which form the lower trunk and medial cord of the brachial plexus. C7 also contributes a component through the middle trunk and lateral cord. The nerve descends into the upper arm in proximity to the brachial artery and median nerve. At about the midhumeral level it deviates dorsally and pierces the medial intermuscular septum to continue superficially on the medial head of the triceps. At the elbow, the nerve enters a groove formed by the medial epicondyle of the humerus and the olecranon of the ulna (Fig. 1). The flexor carpi ulnaris overlies this groove and itself arises as two heads: one from the medial epicondyle and the other from the medial margin of the olecranon. The lateral border of the groove is formed by the ulnar collateral ligament, which bridges the medial epicondyle and both the olecranon and coronoid process of the ulna. This groove has been termed the cubital tunnel by Feindel and Stratford. The nerve then continues along the medial aspect of the forearm and, proximal to the wrist, assumes a more ventral location as it enters the hand. The ulnar branches to the flexor carpi ulnaris (FCU) and flexor digitorum profundus (FDP) usually arise distal to the medial epicondyle, but in 2 of Sunderland’s 20 dissections, the FCU branches arose at, or proximal to, the epicondyle. The fascicles for these muscles may have defined themselves intraneurally for several centimeters, however, before actually forming an external branch.

There are several features of anatomy which may enhance the nerves’ susceptibility to injury at the elbow and also account for the frequently observed clinical pattern of hand muscle involvement with sparing of the ulnar forearm muscles. The number of fascicles tends to be reduced in the elbow region, as is the percentage of the total cross-sectional area of the nerve occupied by epineural tissue. These features may render the nerve more compression sensitive. The axons innervating the FCU and FDP tend to be located posterolaterally at that level, while the sensory and

Acknowledgments: The assistance of Drs. J. Goodman, D. Cooper, H. Feuer, M. Barr, M. Turner, and T. Horner of the Indianapolis Neurosurgical Group for referring patients is gratefully acknowledged, as is the assistance of Dorothy Eddy in manuscript preparation.
motor axons for the hand tend to be located anteromedially. The latter are said to be potentially more susceptible to compression in the cubital tunnel.37 The branches to the forearm muscles, being smaller than the main nerve, may be less compression sensitive as well.

The dimensions of the cubital tunnel change with elbow position, so that minimum tunnel size is present when the elbow is flexed. This is said to occur because the ulnar collateral ligament and the aponeurosis between the two heads of the flexor carpi ulnaris become taut when the elbow is flexed.

Pathology. Neary and Eames examined the ulnar nerves in three autopsy cases.27 One had obvious clinical evidence of ulnar neuropathy. A firm nodular swelling was noted in the clinically affected nerve arising just proximal to the arch of the two heads of the flexor carpi ulnaris. Histologic abnormalities were confined to the nerve segments extending 5 cm proximal and distal to the FCU arch. At the lesion’s center, increased perineural thickness and endoneural connective tissue were noted. The numbers of larger-diameter axons were decreased at that level, and both thinly myelinated fibers and clusters of regenerating fibers were noted. Bulbar myelin swellings were noted in the internodes situated centrifugally from the lesion. Even in the more severe of the material they examined, the fiber diameter histogram was normal distal to the lesion, suggesting that the predominant pathology in their patients was demyelination rather than axonal loss. In situations where the electromyographer encounters fibrillation potentials, enlarged motor unit potentials, slowed velocity, and reduced amplitude evoked responses distal to a focal lesion, varying degrees of axonal injury must have also occurred.

Pathogenesis. A number of pathogeneses have been suggested for elbow lesions. The tardy ulnar palsy which is said to follow an elbow fracture or dislocation by many years is probably best known.32 Alteration of the carrying angle of the elbow and limitation of full extension are factors postulated for the nerve being subjected to traction around the elbow. The concept of compression in the cubital tunnel was suggested by Osborne31 and Fiendel and Stafford35 to account for patients who developed idiopathic lesions and also as another explanation for tardy lesions. This is likely the most common cause of ulnar elbow lesions.12,29 Lesions that occur during general anesthesia are also well known. Although a compressive lesion is suspected, the predisposing factors are not known.26 Hypermobility of the nerve, with a tendency to sublux over the medial epicondyle, is another possible pathogenesis. The appearance of ulnar mononeuropathy may also herald the onset of a more generalized neuropathy.
ELECTROPHYSIOLOGIC EVALUATION

The evaluation of suspected ulnar neuropathy at the elbow has been the subject of a number of reports. The length of a nerve segment which includes the elbow portion of the nerve may also be a factor in the ability of conduction studies to identify a lesion. A very short nerve segment which includes a lesion that produces local slowing should have a high chance of being abnormal. When conduction velocity is calculated over a progressively longer segment that may include normal along with the abnormal nerve, any slowing present in the abnormal segment may be diluted and normalized. Short-length segments, though, may be susceptible to considerable error in velocity calculation due to inaccuracies in measuring latency and distance. Most authors have used a 10–12 cm distance between the below and above-elbow stimulation sites, but segments as short as 2 cm or as long as 19 cm have been reported. Longer, overlapping segments such as elbow-wrist and axilla-wrist may be used for velocity comparison with the forearm as an attempt to limit the inaccuracies encountered with shorter-length ones.

If the elbow segment velocity alone is used as the primary identifier of an ulnar elbow lesion, then the angle of the elbow may not be a consideration, since direct comparison to normal values alone is required. Reported lower limits of normal conduction velocity for that segment are shown in Table 1.

When attempting to localize a lesion by comparison of velocities in the elbow and adjacent segments, the tendency for elbow segment slowing, encountered with an extended elbow position, may limit the study’s sensitivity, and therefore a flexed position seems preferable. Even with the elbow flexed, some segment-to-segment conduction

<table>
<thead>
<tr>
<th>Table 1. Elbow segment motor velocity.</th>
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<tbody>
<tr>
<td>Angle</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>0°</td>
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<tr>
<td>0°</td>
</tr>
<tr>
<td>0°</td>
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<tr>
<td>0°</td>
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<td>0°</td>
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<tr>
<td>35°</td>
</tr>
<tr>
<td>45°</td>
</tr>
<tr>
<td>135°</td>
</tr>
<tr>
<td>90°</td>
</tr>
<tr>
<td>90°</td>
</tr>
<tr>
<td>110°</td>
</tr>
</tbody>
</table>

Note: 0° = full extension.

*Two standard deviations or lower range (R) if the latter is higher.
†Denotes intramuscular needle recording.
velocity variation will still exist due to experimental error in distance and latency measurement. A difference of up to 11 m/sec for the elbow segment velocity as compared with the forearm was found in studies of normal subjects done at 45° and 60° of flexion, while another reported that this velocity should be no more than 7.6% slower than the forearm when an 110° flexion was used. Differences of greater than 20 m/sec could be encountered if this comparison is done with the elbow in extension. The normal values used in the author’s laboratory are shown in Table 2, and recording methods are described in the Appendix.

Lesions may be encountered in which the forearm velocity is also slowed. In such a situation, inappropriate elbow segment slowing might not be encountered, but comparison to a proximal segment such as axilla-elbow could be valuable. Reported normal values for axilla-elbow velocities are shown in Table 3. A very mild lesion might produce only relative slowing but leave the absolute velocity or latency normal. Such findings can be seen in mild carpal tunnel syndromes.

Several reports have demonstrated the actual types of motor conduction abnormalities that can be encountered. Pavan found elbow segment motor velocity slowing in 85% of his patients. In 54%, comparison of the elbow segment and forearm velocities localized the lesion to the elbow. In patients with both sensory and motor clinical findings, Tackmann found that 65% had elbow segment slowing, and 49% localized to the elbow by slowing relative to the forearm. Fifty patients with the clinical diagnosis of an ulnar elbow lesion were studied in the Indiana University EMG Laboratory. Among these patients, 40% had both motor and sensory deficits, and of these, 86% had elbow segment motor slowing and 48% could be localized to the elbow by comparison with the forearm velocity. The remainder failed to localize because the forearm conduction was also slow. Comparing velocities between the longer elbow-wrist segment and that of the forearm was slightly less sensitive in localizing lesions. Ninety-four percent of those with a slow elbow segment velocity had elbow-

Table 2. Normal ulnar conduction values. 135° flexion.

<table>
<thead>
<tr>
<th>Segment</th>
<th>CMAP amplitudes (mV)</th>
<th>SNAP amplitudes (μV) antidromic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>x</td>
<td>SD</td>
</tr>
<tr>
<td>Above-elbow</td>
<td>11.7</td>
<td>2.2</td>
</tr>
<tr>
<td>Above-shoulder</td>
<td>11.2</td>
<td>2.1</td>
</tr>
<tr>
<td>Above-neck</td>
<td>10.8</td>
<td>2.1</td>
</tr>
<tr>
<td>Above-lower</td>
<td>10.4</td>
<td>2.0</td>
</tr>
</tbody>
</table>

Maximum change between below-elbow and elbow: 1.2 mV or 10% to 8 μV or 43%.

Table 3. Axilla-elbow segment velocity.

<table>
<thead>
<tr>
<th>Elbow angle</th>
<th>Low-normal* velocity</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>0°</td>
<td>50</td>
<td>14</td>
</tr>
<tr>
<td>35°</td>
<td>46R</td>
<td>2</td>
</tr>
<tr>
<td>90°</td>
<td>52R</td>
<td>19</td>
</tr>
<tr>
<td>135°</td>
<td>50</td>
<td>21</td>
</tr>
</tbody>
</table>

*Two standard deviations below the mean or lower range (R) if the latter is higher.
wrist slowing. Eighty-eight percent of those with a slow elbow segment velocity, both absolute and relative to the forearm, had a greater-than-normal difference between the elbow-wrist and forearm. Seventy-six percent of this group with sensory and motor symptoms had low CMAP amplitude with elbow stimulation.

In a group of patients who had sensory symptoms only, Payan still found elbow segment slowing in 71%. Tackmann found slowing in 33%, and 20% could be localized by comparison to the forearm. Twenty-three percent had a slowed forearm velocity. In a similar group of patients studied in our lab, 48% had elbow segment slowing and a greater-than-normal difference when that velocity was compared with the forearm. Another 16% had normal elbow segment velocity but a greater-than-normal difference with the forearm. None had forearm slowing. The elbow segment velocity was again more often abnormal than was the elbow-wrist one. Only 40% of those with a slow elbow segment velocity had elbow-wrist slowing. Six percent of this entire group had a low CMAP with elbow stimulation. Four percent had an abnormally slow elbow segment motor velocity but had normal sensory conduction studies.

Sensory Conduction Studies. Sensory conduction studies may also be valuable in evaluating ulnar neuropathy. Two studies have shown that assessment of sensory conduction velocity and action potential configuration could be useful in identifying elbow lesions. Orthodromic recording with near-nerve needle electrodes were used in both reports. Elbow segment slowing was present in the normal subjects reported by Payan using the extended-elbow position. Surface recording of orthodromic SNAPs at the wrist and more proximal sites has also been reported. In our experience, orthodromic surface-recorded SNAPs may be lower in amplitude at the below-elbow site than at the elbow, even in thin subjects. This is likely due to greater distance between the nerve and the surface electrodes at the former site because of the nerve being deep to the flexor carpi ulnaris there. Antidromic recording obviates this problem, but rarely antidromic SNAPs may be difficult to distinguish from the closely trailing volume-conducted CMAP. Although Harding found segment-to-segment velocity variation of a sufficient degree to cast doubt on the utility of such studies, others found antidromic recording to be easily, reliably accomplished. Our normal values for sensory velocities and SNAP amplitude are shown in Table 2.

The types of sensory conduction abnormalities actually encountered have been reported by several authors. Payan found elbow segment slowing in 86% of his patients, 83% had forearm slowing, and 63% could be localized to the elbow. Seventy-eight percent had a low amplitude SNAP at the wrist. In a group of patients with both sensory and motor symptoms, Tackmann found elbow segment slowing in 70%, whereas 32% had forearm abnormality, and 54% could be localized to the elbow. Sixty percent had low amplitude or nonresponsive wrist SNAPs. With antidromic surface recording, 71% of our group of patients with both sensory and motor symptoms showed a nonresponsive SNAP at the wrist, and 15% more had a low-amplitude response. Of those 15%, one-third showed elbow segment slowing, one-third had no detectable response with elbow stimulation, and one-third showed normal velocities.

Our patients with sensory-only symptoms had no detectable SNAP with wrist stimulation in 5%, and 26% had low amplitude. Thirty-two percent had elbow segment slowing, and 29% had greater-than-normal slowing compared with the forearm. Another 32% had normal elbow segment velocity but showed a greater-than-normal difference when compared with the forearm. Thirteen percent were normal by all electrical criteria.

Proximal Segment Motor Latencies. Latency from an above-elbow stimulus site to the FCU or the ulnar-innervated FDP has been assessed in several reports and appears to be a valuable parameter. Conduction distances of 12–15 cm have been used for the FCU, and normal latencies of less than 3.7–4.0 msec have been reported for subjects 68 years or younger. One report specifies placing the recording electrode 10 cm distal to, and stimulating 2 cm proximal to, the medial epicondyle. From 44 to 82% of patients have shown abnormalities in this parameter. This may be abnormal while the elbow segment motor velocity is normal. Intramuscular needle recording has been utilized. Whether surface recording could be done has not been specified.

Amplitude Change Between Stimulus Sites. Change in the amplitude (Fig. 2) or configuration (Fig. 3) of the CMAP or SNAP may be useful in localizing a lesion site along the nerve. For surface electrode-recorded CMAPs, Checkles found that normals had elbow amplitudes no
more than 36% smaller than that of the below-elbow site. Brown found no more than a 5% difference in peak-to-peak amplitude, whereas another study reported a maximum decrease of 10.5% or 1.2 mV between these sites for baseline-to-negative-peak measurements. The incidence of such CMAP amplitude changes in a group of patients with ulnar neuropathy has not been specified, but we found these in 10% of patients, all of which had elbow segment slowing. The magnitude of these amplitude changes ranged from 2 to 5.6 mV.

When a greater-than-normal amplitude difference is identified between standard stimulation points such as below-elbow and elbow, then serial stimulation between those sites at intervals of 1 or 2 cm, “inching,” may precisely localize a lesion by showing a point of abrupt change in the response’s amplitude or additionally an abrupt prolongation of latency. This technique can usually be easily applied to ulnar stimulations in the elbow segment. Amplitude change due to a lesion must not be confused with that which can be seen in variations of normal arm innervation. Median-to-ulnar nerve cross-overs may cause a greater-than-normal difference in CMAP amplitude between the elbow and wrist stimulation sites but should not affect the elbow and below-elbow sites.

Change in SNAP configuration or amplitude may also be useful in identifying lesions. Orthodromic potentials recorded with near-nerve needle electrodes could show decreases of up to 40–50% in peak-to-peak amplitude between the below-elbow and elbow sites. Antidromic surface-recorded potentials could change by up to 53%, or 8 μV. Tackmann found that greater-than-normal amplitude change was always accompanied by slowing of sensory conduction velocity. Three percent of a group of patients evaluated in our laboratory showed a greater-than-normal SNAP amplitude change, while all other sensory parameters were normal. Tackmann noted that change in SNAP configuration was useful in localizing 13% of patients with sensory symptoms alone but only 2% of those with motor and sensory lesions. Payan found these parameters helpful in 10% of cases.

**Needle Examination.** Needle electrode examination is useful in establishing whether axonal interruption has occurred, gauging the chronology of the lesion, determining whether reinnervation is occurring, and localizing the lesion site. In concert with conduction studies it can also be very helpful in differentiating a radicular or plexus lesion from one of the ulnar nerve. The examination should include the FDI and hypothenar muscles, a forearm muscle innervated by the ulnar nerve, and the abductor pollicis brevis.

Mild lesions with sensory loss as the primary symptom may result in no needle exam abnormalities. Lesions characterized by conduction block may show reduced motor unit potential (MUP) recruitment, but little else. When axonal interruption occurs, positive sharp waves and fibrillations can be observed. Enlarged MUPs suggest that reinnervation has taken place, whereas polyphasic MUPs demonstrating moment-to-moment variation in shape indicate that reinnervation is taking place. FCU or FDP abnormalities indicate a lesion arising at or proximal to the elbow, although these muscles may be normal in the presence of elbow lesions.

Pavan found hand muscle fibrillation in 57% of his cases. Eisen noted fibrillations or positive waves in the FDI in 50%, hypothenar in 37%, and FCU in 6% of patients with sensory and motor deficits. MUP abnormalities were present in hand muscles in all patients and in the FCU in 27%.

In our group of patients 47% showed needle
exam abnormalities in hand muscles. Sixty-three percent of those had fibrillations. Forty-one percent of the total group also had abnormalities in the FCU. Sixty-five percent of those with FCU abnormalities had enlarged motor unit potentials, whereas another 24% had both fibrillations and enlarged MUPs. The frequency of FCU abnormality on both needle exam and proximal segment latency testing is contrasted with the infrequent involvement of the forearm muscles detected on clinical examination.1,13

Patients in our group who demonstrated needle exam abnormalities tended to have more severe lesions. Ninety-three percent of those with both fibrillations and enlarged MUPs in hand muscles had lesions producing both motor and sensory symptoms.

**Cumulative Results.** Using the combined results of the individual evaluations, Tackmann was able to localize the lesion in 95% of patients with motor and sensory deficits and 82.5% of those with sensory-only deficits.59 Payan could place the lesion at the elbow in 96% of his cases.53

In our patients with both motor and sensory symptoms, 48% could be localized by the finding of an elbow segment motor velocity that was slowed both absolutely and relative to the forearm. Another 5% with normal elbow segment velocity localized by a greater-than-normal difference relative to the forearm. Nineteen percent more could be localized by comparing the elbow segment and axilla-elbow velocities when the forearm was also slowed. Sensory studies did not localize any additional patients. Needle exam abnormalities in forearm muscles helped to place lesions at or proximal to the elbow in an additional 14%. Fourteen percent showed slowed elbow segment and forearm velocities, but did not have axillary stimulation done; nor did they show needle exam abnormality in proximal ulnar muscles.

In the patients with sensory-only symptoms, 48% could be localized by elbow segment motor slowing both absolute and relative to the forearm. Another 16% with a normal elbow segment motor velocity showed a greater-than-normal velocity difference compared with the forearm. Three percent showed elbow segment slowing without a

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**FIGURE 4.** Electrode and ground placement for ulnar (hypothenar) motor recording.
significant difference to the forearm but had a normal axilla-elbow velocity. Three percent additionally localized by absolute and relative sensory velocity slowing, whereas another 13% had normal elbow segment sensory velocity but a greater-than-normal difference relative to the forearm. Three percent more showed only an abnormal change in SNAP amplitude between the below-elbow and elbow stimulus sites. Needle examination did not provide any additional data aiding in elbow localization. Thirteen percent of these patients, 8% of the combined groups, were normal in all parameters.

**Electrophysiologic Differential Diagnosis.**

*Cervical Radiculopathy.* Cervical radiculopathy at the C8 or T1 level could produce symptoms and signs similar to ulnar neuropathy at the elbow. Ulnar sensory conduction studies should be normal. The ulnar and median CMAP could be low and motor conduction velocity mildly slowed if axonal injury has occurred. Both ulnar and median F

![FIGURE 5](image)

*FIGURE 5.* Flexed elbow position showing wrist, below-elbow, and elbow stimulus sites. Axillary site is 10–15 cm proximal to elbow site.
wave latencies could be prolonged. If needle examination abnormalities are present, they should be seen in a combination of ulnar, median, and radial innervated muscles of those myotomes and may also be present in the cervical paraspinal muscles.

Lower Trunk Plexopathy. Involvement of the lower trunk or medial cord of the brachial plexus can present similarly to ulnar neuropathy at the elbow. Because the lesion is distal to the sensory ganglion cell, ulnar sensory nerve conduction studies are often abnormal. The classic conduction study findings are an absent or low-amplitude ulnar SNAP and reduced CMAP amplitude of both the ulnar and median innervated hand muscles. Mild motor velocity slowing may be present, and prolongation of both median and ulnar F wave latencies might be seen. Again in such cases, needle exam abnormalities should be present in both median and ulnar territories that share C₃ and T₁ innervation. Cervical paraspinals should be spared in this lesion.¹⁵,⁴⁰

Wrist Lesion. The ulnar nerve may suffer local trauma at the wrist as it enters the hand through Guyon’s canal. Although both hypothenar and interossei involvement may occur, preferential involvement of the FDI is said to favor a wrist lesion. Distal latency to the FDI may be disproportionately prolonged as compared with the hypothenar group. The ulnar SNAP may or may not be abnormal, depending on the nature of the lesion. The dorsal cutaneous ulnar action potential should be normal, since this nerve branch arises proximal to the wrist.¹⁰,³⁰

SUMMARY

Electrodiagnostic testing is useful in evaluating ulnar nerve elbow lesions. A flexed elbow seems preferable for conduction studies, since it eliminates the elbow segment slowing found in normals done in the extended position. Slowing of the motor velocity in the elbow segment was the most frequent abnormality in this study. Sensory conduction studies and needle examination each provided additional helpful data. Latency to ulnar forearm muscles and “inching” stimulations around the elbow are techniques that also deserve to be included in our standard armamentarium.

FIGURE 6. Electrode and ground placement for antidromic ulnar sensory recording.
APPENDIX

This protocol is used in our laboratory for the evaluation of suspected ulnar neuropathy at the elbow. Ulnar motor conduction studies are performed with recording by surface electrode. The electrode placement is shown in Fig. 4. Surface temperature measured over the first dorsal interosseus must be at least 33°C. The elbow is flexed to 135° and forearm supinated (Fig. 5). Stimulation sites are at the wrist 6.5 cm proximal to the recording electrode, below the elbow 3–4 cm distal to the medial epicondyle, above the elbow 8–12 cm proximal to the below-elbow site, and in the axilla. Responses are measured from photorecorder records. CMAPs are recorded at a 5 mV per division gain and a 1.6 Hz to 16 KHz bandwidth. Onset latency and baseline-to-negative-peak amplitude are determined at each site. F wave latency is measured from the wrist. Antidromic SNAPs are recorded from the fifth digit. The recording electrode placement is shown in Fig. 6. Stimulation sites are the same as motor, except the wrist site is adjusted for an 11 cm distance to the active recording electrode. Responses are recorded at 10 μV/division gain and a 32 Hz to 3.2 KHz bandwidth. Three traces are superimposed on the photorecorder record. Electronic averaging is not used. Action potentials are considered non-responsive when no reproducible response is observable above baseline noise. Velocity calculations are done from onset latencies. The baseline-to-negative-peak amplitude of each response is also measured. Our normal values for amplitudes, segmental velocities, and difference in velocity between segments are shown in Table 2. Median nerve conduction studies are also done. These should include at least assessment of the amplitudes of CMAP and SNAP, motor and sensory distal latency, motor conduction velocity between elbow and wrist, and the F wave latency. Needle examination is done on the FDI, hypothenar, and FCU muscles as well as the abductor pollicis brevis.


REFERENCES