

ABSTRACT: The aim of the study was to assess the diagnostic value of short-segment nerve conduction studies (NCS) at 2-cm intervals from 4 cm above to 4 cm below the medial epicondyle in a large group of patients with ulnar neuropathy at the elbow (UNE). Furthermore, we wanted to compare electrodiagnostic and clinical findings. We evaluated 73 arms in 70 patients with UNE and observed the following abnormalities on short-segment NCS: focal conduction block (CB) in 1, focal CB with increased latency change in 34, and increased latency change alone in 25. Short-segment NCS had an additional localizing value in 28 arms of the 37 patients (76%) with motor conduction velocity (MCV) slowing across the elbow only or with nonlocalizing electrodiagnostic findings. The lesion was located above the elbow in 32 arms (53%), at the epicondyle in 16 arms (27%), and below the epicondyle in 12 (20%) of the 60 arms with focal CB or increased latency change on short-segment NCS. Patients with CB on routine and short-segment NCS had muscle weakness significantly more often than patients without CB. Thus, short-segment NCS are useful in localizing the lesion in patients with UNE and CB on routine NCS and have additional diagnostic value in patients with MCV slowing across the elbow or with nonlocalizing signs on routine nerve conduction studies. We recommend its use in all patients in whom UNE is suspected.

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SHORT-SEGMENT NERVE CONDUCTION STUDIES IN ULNAR NEUROPATHY AT THE ELBOW

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The diagnosis of ulnar neuropathy at the elbow (UNE) is straightforward when motor nerve conduction studies (NCS) show a conduction block (CB) across the elbow. However, in many patients, the only localizing electrodiagnostic abnormality is motor nerve conduction velocity (MCV) slowing across the elbow. Unfortunately, the 95% confidence interval of the mean motor nerve conduction velocity across the elbow may vary considerable due to accumulation of distance and latency measurement errors,¹³ and isolated differential slowing across the

elbow is anyway of limited value as a sole criterion.^{3,13} Furthermore, routine NCS cannot discriminate between a lesion at the retroepicondylar groove and the humero-ulnar arcade. Precise localization becomes especially important when surgery is contemplated. Therefore, additional tests are required to make a reliable diagnosis.

In 1999, the American Association of Electrodiagnostic Medicine suggested the use of short-segment NCS, previously called “inching,” when routine motor conduction studies are inconclusive.¹ Short-segment NCS may make more precise localization possible.^{6–8,16} However, the literature concerning its diagnostic value in patients with UNE is limited, especially in patients without CB. Further, the results of studies are not consistent, e.g., normal values differ considerably between various studies and it is not always clear how normal values were obtained.^{2,7,11}

The aim of this study was to assess the sensitivity and additional value of short-segment NCS in a clin-

Abbreviations: ADM, abductor digiti minimi; CB, conduction block; CMAP, compound muscle action potential; FCU, flexor carpi ulnaris; FDI, first dorsal interosseous; FDP, flexor digitorum profundus digit IV and V; MCV, motor conduction velocity; MRC, Medical Research Council (rating score); NCS, nerve conduction studies; UNE, ulnar neuropathy at elbow

Key words: clinical characteristics; diagnostic value; elbow; short-segment nerve conduction studies; ulnar nerve

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Table 1. Clinical characteristics and baseline electrophysiological characteristics of 70 patients (73 arms) with UNE.

Characteristics	Routine NCS			Total
	Localizing		Not localizing	
	CB plus slowing	Slowing only		
A. Normal neurological examination	0	2	0	2 (3%)
Sensory signs only				
B. Volar cutaneous branches	2	3	1	6 (8%)
C. Volar and dorsal cutaneous branches	5*	10	0	15 (20.5%)
Sensory and motor signs				
D. Volar cutaneous branches with weak ADM or FDI	2	2	1	5 (7%)
E. Volar and dorsal cutaneous branches with weak ADM or FDI	9	9	2	20 (27%)
F. As group D but also with weak FCU or FDP	1	1	0	2 (3%)
G. As group E but also with weak FCU or FDP	17	4	2	23 (31.5%)
Total	36*	31	6	73

ADM, abductor digiti minimi; CB, amplitude reduction of >17% across the elbow; FCU, flexor carpi ulnaris; FDI, first dorsal interosseous; FDP, flexor digitorum profundus digit IV and V; MCV, motor conduction velocity; NCS, nerve conduction studies; Slowing, motor velocity across the elbow <46 m/s; UNE, ulnar neuropathy at elbow.

*One patient with CB without MCV slowing.

ically relevant spectrum of patients with UNE and to study the correlation with clinical findings. For this purpose, we prospectively performed routine and short-segment NCS in a cohort of 70 patients.

METHODS

Patients. Between May 1998 and September 2003, we prospectively performed clinical, electrodiagnostic, and sonographic studies in patients with UNE and performed short-segment NCS in 73 arms of 70 patients: 45 men and 25 women with a mean age of 50 years (range, 20–80 years). Six patients (9%) had bilateral UNE and, in three of these patients, short-segment NCS were performed on both sides. All patients underwent standard clinical and electrodiagnostic evaluations. The study was approved by the local medical ethical committee.

Diagnosis of UNE. A diagnosis of UNE was made on clinical grounds alone when there were signs that localized the ulnar neuropathy to the elbow, i.e., weakness of the proximal ulnar muscles or weakness of the distal ulnar muscles accompanied by a sensory disturbance in the area of the dorsal cutaneous sensory branch (groups E, F, and G, Table 1). Clinical patterns that were compatible with UNE but did not localize the lesion to the elbow (i.e., weakness of the distal ulnar muscles with a sensory disturbance only in the area of the volar cutaneous branch, sensory signs alone, or symptoms without signs) needed further confirmation by electrodiagnostic studies to make a definite diagnosis of UNE (groups A–D, Table 1). Especially in the patients with nonlocaliz-

ing electrodiagnostic signs, clinical follow-up and sometimes magnetic resonance imaging of the brachial plexus or cervical spine were used to exclude conditions mimicking UNE (lower brachial plexopathy, radiculopathy, and ulnar neuropathy at the wrist).⁴

Patients with a history of ulnar nerve surgery, acute traumatic origin of UNE, a history of polyneuropathy or genetically proven hereditary neuropathy with liability to pressure palsies, or signs of polyneuropathy were excluded from the study.

Clinical Assessment. In all patients, the following items were systematically assessed: (1) pinprick sensation in the area of the ulnar digital, palmar cutaneous, and dorsal cutaneous sensory branches; and (2) strength of the first dorsal interosseous (FDI), abductor digiti minimi (ADM), flexor carpi ulnaris (FCU), and flexor digitorum profundus (FDP) muscles using the Medical Research Council rating scale (MRC score). To assess the distribution and severity of muscle weakness, the strength of two proximal (FCU and FDP) and distal (FDI and ADM) muscles were quantified by determining the distal, proximal, and total MRC sumscore. The distal or proximal MRC sumscore ranged from 0 to 10 (10 = normal) and total MRC sumscore from 0 to 20 (20 = normal). For the analysis, we used two groups: patients with sensory loss and muscle weakness, and patients with sensory loss alone.

Routine Nerve Conduction Studies. Ulnar sensory and motor nerve conduction studies were per-

Table 2. Reference values for short-segment NCS of the ulnar nerve across the elbow obtained in 53 healthy controls.

Site	Latency change (ms)				Amplitude change (%)			
	ADM		FDI*		ADM		FDI*	
	Mean ± SD	ULN	Mean ± SD	ULN	Mean ± SD	ULN	Mean ± SD	ULN
4 cm to 2 cm above elbow	0.36 ± 0.17	0.63	0.31 ± 0.16	0.57	4 ± 5.4	13	3.5 ± 4.6	12
2 cm above elbow to midpoint	0.46 ± 0.23	0.84	0.53 ± 0.21	0.87	3.4 ± 5.3	12	2.5 ± 2.6	8
Midpoint to 2 cm below elbow	0.45 ± 0.18	0.74	0.47 ± 0.13	0.68	3 ± 4.3	10	3 ± 3.6	9
2 cm to 4 cm below elbow	0.22 ± 0.13	0.43	0.22 ± 0.2	0.55	5.4 ± 9.2	20	4.9 ± 4.5	12

ADM, abductor digiti minimi; FDI, first dorsal interosseous; midpoint, midpoint of the ulnar groove determined by drawing a line between the medial epicondyle and olecranon; NCS, nerve conduction studies; SD, standard deviation; ULN, upper limit of normal (= mean + 1.64 SD; see text for details).
*Tested in 24 subjects.

formed with the elbow flexed at 90°. When the skin temperature measured at the wrist was less than 32°C, the arms were warmed to >32°C using hot packs. Surface stimulation was performed at the proximal wrist crease, 4 cm distal to the midpoint of the medial epicondyle and 4 cm proximal to this level. Compound muscle action potentials (CMAPs) were recorded from the ADM and FDI muscles using surface electrodes in a belly-tendon montage. Recording from the ADM was performed in 72 arms (99%) and from the FDI in 56 arms (77%). Both recordings were made in 55 of the 73 arms (75%).

Sensory nerve action potentials were obtained antidromically using ring electrodes placed over the fifth digit with surface stimulation at the proximal wrist crease.

Using concentric needle electromyography, one or more of the ADM, FDI, FCU, and FDP muscles were studied for spontaneous muscle-fiber activity (fibrillation potentials and positive sharp waves), motor unit potential configuration, and recruitment pattern.

In a previous study, we obtained normative values for the nerve conduction studies by examining 29 volunteer subjects.⁴ For this study, we increased the sample size to 52 by testing one arm at random from 52 volunteers (normal subjects and patients with other neurological diseases not involving the peripheral nerves). All provided informed consent to participate. The controls were questioned and examined for ulnar motor or sensory symptoms and confounding factors to assure that even mild UNE was excluded. The reference values were as follows: CB, >17% amplitude reduction across the elbow; MCV across the elbow, <46 m/s; low-amplitude distal ulnar SNAP, <12 µV; and low-amplitude ADM CMAP, <7.3 mV.

Short-Segment NCS. Short-segment NCS were performed in each subject with the elbow flexed at 90°.

First, the midpoint of the ulnar groove was identified by drawing a line from the medial epicondyle to the olecranon. From this level, markers were placed along the course of the ulnar nerve at 2-cm intervals from 4 cm proximal to 4 cm distal to the level of the medial epicondyle. The ulnar nerve was stimulated with the cathode at each marker. Before measurements were performed, we ensured that supramaximal stimulation was achieved and adequate pressure to the stimulating electrodes was applied to enable focal stimulation without spread. Latencies were measured from stimulus to CMAP onset and amplitudes from baseline to negative peak.

In order to obtain upper limits of normal for latency change and CMAP amplitude change over each of the 2-cm segments across the elbow, we studied 53 randomly selected asymptomatic arms of 53 healthy subjects and recorded the ADM. In 24 controls, we also recorded from the FDI muscle. There were 25 men and 28 women with a mean age of 42 years (range, 17–72 years); all provided informed consent to participate. After randomization, 24 subjects were examined on the left and 29 on the right side. We did not find any significant differences in reference values between men and women or between the left and right sides. The reference values obtained by recording the ADM did not differ from those obtained by recording the FDI. The mean, standard deviation (SD) and upper limits for normal latency and amplitude changes for each 2-cm segment across the elbow (mean latency change + 1.64 SD) are shown in Table 2.

Statistics. Normative data were determined using mean + 1.64 SD when the upper boundary (95th percentile value) was needed and using mean – 1.64 SD when we were interested in the lower boundary (5th percentile value). The clinical and electrodiagnostic characteristics of the different groups were compared using the chi-square test for comparison

Table 3. Comparison of routine and short-segment NCS in 73 arms of 70 patients with UNE.

Routine NCS	Short-segment NCS			Total
	Focal CB plus ↑ latency	↑ latency alone	No abnormalities	
CB plus MCV slowing	32*	0	2	36 (49%)
MCV slowing	3	23	4	31 (43%)
Nonlocalizing signs	0	2	4	6 (8%)
Total	35 (48%)	25 (34%)	10 (14%)	73

CB, amplitude reduction of >17% across the elbow; MCV, motor conduction velocity; NCS, nerve conduction studies; UNE, ulnar neuropathy at elbow; ↑ latency, increased latency change.

*One patient with only focal CB without increased latency change or MCV slowing across the elbow.

of proportions. For comparison of two groups, we used the Wilcoxon rank-sum two-sample test and, for comparison of means of ordinal variables between more than two groups, the Kruskal–Wallis test (non-parametric one-way analysis of variance) was used.

RESULTS

Routine NCS and Clinical Characteristics. The clinical findings and baseline electrophysiological characteristics of our patients are presented in Table 1. Thirty-six arms (49%) showed CB and 35 of them also had MCV slowing. The mean percentage of CB was 50% [95% confidence interval (CI) 40%–59%] when recording from the ADM and 44% (95% CI 32%–56%) when recording from the FDI.

Thirty-one arms (43%) had MCV slowing across the elbow without CB. Six arms (8%) had only nonlocalizing signs, such as a small sensory nerve action potential, small CMAP, or abnormalities during concentric needle electromyography in one or two of the distal ulnar muscles (Table 1).

The mean age for the patients with CB was significantly younger (46 years) than the patients with MCV slowing alone (54 years; $P = 0.01$). Among the women, 74% had CB in comparison to 43% in men ($P = 0.03$). The patients with CB had a shorter duration of symptoms (2 months vs. 4 months; $P = 0.03$) and more often muscle weakness (81% vs. 52%; $P = 0.02$) than the patients with MCV slowing alone. Concentric needle electromyography was performed in 35 arms with CB and in 29 arms with MCV slowing. Spontaneous muscle activity was more often present in the arms with CB ($n = 23$, 66%) than the arms without CB ($n = 8$, 27%; $P = 0.002$).

We also reanalyzed the data comparing the clinical characteristics of the group of patients with >50% CB (19 arms) on routine electrodiagnostic testing and the 47 patients who had MCV slowing without such a CB. The 19 patients with >50% CB

had a significantly shorter onset of symptoms ($P = 0.01$), more often had muscle weakness ($P = 0.005$), and had more severe weakness of the distal ($P = 0.0003$), proximal ($P = 0.0002$), and all four of the ulnar-innervated muscles ($P = 0.0001$) than the 47 patients without such CB. Also, the MCV across the elbow (recording from ADM) was slower in this group: 30 m/s (95% CI 24–37 m/s) in comparison to 38 m/s (95% CI 34–42 m/s) in the group without >50% CB ($P = 0.04$) and they more often had spontaneous muscle activity on needle electromyography [14/18 (78%) vs. 16/46 (35%); $P = 0.005$].

Short-Segment NCS. The results of short-segment NCS and routine NCS are shown in Table 3. In 35 arms, focal CB was present. Focal CB occurred together with an increased latency change in 34 arms. A focal CB without increased latency change was found in only one arm. This patient was also the only one with CB without MCV slowing on routine nerve conduction studies.

In three cases, it was not possible to make an accurate interpretation of short-segment NCS: in one, because of inconsistencies in CMAP waveform and, in the other two, because of the presence of increased latency change at two different segments (2–4 cm above midpoint of elbow and also 4–2 cm below elbow).

MCV slowing during routine NCS with or without CB was the most common localizing finding, occurring in 66 arms (90%; Table 3, total of rows 1 and 2, except for 1 patient who had CB without MCV slowing), followed by focal CB or increased latency change on short-segment NCS in 60 arms (82%; Table 3, columns 1 and 2), and CB on routine NCS in 35 arms (48%).

Short-segment NCS had additional localizing value in 28 arms of the 37 patients (76%) who had MCV slowing alone or nonlocalizing electrodiagnostic findings.

Table 4. Clinical characteristics of 59 patients (60 arms) with focal CB and increased latency change, increased latency change only, or no abnormalities on short-segment NCS.*

Characteristics	Short-segment NCS			P-value
	Focal CB	Increased latency change	No abnormalities	
No. of arms	35	25	10	
Age (mean, range)	48 (20–80)	53 (22–76)	51 (36–77)	ns
Sex				
Men	19 (54%)	23 (92%)	5 (50%)	0.004
Women	16 (46%)	2 (8%)	5 (50%)	
Affected side				
Left	24 (69%)	15 (60%)	3 (30%)	ns
Right	11 (31%)	10 (40%)	7 (70%)	
Duration of symptoms (months)	3 (2–4)	3.5 (2.8–6.4)	2.3 (1.9–15)	ns
Clinical deficits				
Motor-sensory	30 (86%)	10 (40%)	8 (80%)	0.0006
Sensory only	5 (14%)	15† (60%)	2 (20%)	
Muscle weakness				
Proximal muscle involvement	17 (49%)	2 (8%)	5 (50%)	0.003
Total distal MRC sumscore	8 (7–8)	10 (8–10)	9 (6–10)	0.002
Total proximal MRC sumscore	9 (9–10)	10 (10)	9.5 (9–10)	0.001
Total MRC sumscore (proximal + distal)	17 (16–18)	20 (18–20)	18 (16–20)	0.0003

CB, conduction block; MRC, Medical Research Council; NCS, nerve conduction studies.

*Median values unless specified otherwise; percentage or confidence interval is provided in parentheses.

†Two patients had only symptoms.

Comparison of the Clinical and Electrodiagnostic Characteristics of the Three Groups on Short-Segment NCS.

As can be seen from Table 4, patients with focal CB more often had involvement of the distal as well as the proximal ulnar muscles than patients having increased latency changes alone or no abnormalities on short-segment NCS.

There were no significant differences between the three groups regarding the distal sensory nerve action potential amplitude, the distal CMAP amplitude, and CMAP amplitude elicited 4 cm distal to the midpoint of the elbow and MCV in the forearm segment.

Recording from the ADM, the median MCV across the elbow was significantly lower ($P = 0.002$) in arms having a focal CB (31 m/s, 95% CI 27–35) than in arms with increased latency change without focal CB (39 m/s, 95% CI 35–43) or arms without abnormalities during short-segment NCS (50 m/s, 95% CI 26–57). For the FDI recording, these values were, respectively, 31 m/s (95% CI 27–36), 39 m/s (95% CI 35–43), and 50 m/s (95% CI 26–57) ($P = 0.003$). Spontaneous muscle-fiber activity was also found more frequently in arms with focal CB (22 of 34, 65%) than in arms with increased latency change only (8 of 24, 33%) or arms without abnormalities during short-segment NCS (4 of 9, 44%; trend to significance $P = 0.06$).

Localization of the Lesion on Short-Segment NCS.

Short-segment NCS localized the lesion proximal to the medial epicondyle in 32 (53%), at the level of the medial epicondyle in 16 (27%), and distal to the medial epicondyle in 12 (20%) of the 60 arms with abnormal short-segment NCS (Table 5). Eleven arms had abnormal short-segment NCS over two segments of 2 cm and two arms over three segments (Table 5). In most arms with focal CB and increased latency change, the focal CB and increased latency change occurred in the same segment, but there were some arms with an increased latency change in the first segment with concurrent significant amplitude reduction in the next segment of 2 cm.

The median duration of symptoms tended to be shorter in the patients with a lesion above the medial epicondyle (3 months) compared to patients with a lesion at (4.5 months) or below (4 months) this level ($P = 0.08$).

DISCUSSION

We found that short-segment NCS can be performed accurately in almost all patients with UNE. A focal CB was found mainly in the patients who also had CB on routine NCS. In these cases, short-segment NCS helped to localize the lesion exactly. Although its diagnostic value is less than measuring MCV across

Table 5. Site of focal CB with increased latency change or increased latency change alone in 60 arms with abnormal short-segment NCS.

Localization	CB and increased latency change (n = 35)*	Increased latency change (n = 25)
Above midpoint at elbow		
2–4 cm above midpoint	3	4
0–2 cm above midpoint	12	10
0–2–4 cm above midpoint (2 segments abnormal)	1	2
At midpoint of elbow		
2 cm below to midpoint	7	5
2 cm below–midpoint–2 cm above elbow (2 segments abnormal)	4	
Below midpoint at elbow		
4–2 cm below midpoint	3	3
4–2 cm below–midpoint (2 segments abnormal)	3	1
4–2 cm–midpoint–2 cm above midpoint (3 segments abnormal)	2	

CB, amplitude reduction of >17% across the elbow; n, number of arms; NCS, nerve conduction studies.

*One arm with focal CB without increased latency change.

the elbow, short-segment NCS may provide additional evidence for a lesion at the elbow by demonstrating a focal CB or increased latency change. These additional localizing findings are especially important when routine electrodiagnostic studies leave uncertainties about the diagnosis of UNE, e.g., presence of isolated MCV slowing across the elbow or presence of nonlocalizing signs only. Sonography and short-segment NCS are, therefore, additional instruments to localize the lesion in patients with UNE.⁴

When analyzing the results of short-segment NCS, it is important to determine the upper limits of normal conduction times and amplitude changes over each 2-cm segment separately. Amplitude reduction or latency change differs over the various 2-cm segments across the elbow. The highest values for latency changes were obtained from the segments between the midpoint at the medial epicondyle to 2 cm above or below the elbow (Table 2). This implies that motor conduction of the ulnar nerve around the midpoint of the medial epicondyle is normally slower in comparison to the more proximal or distal areas. Therefore, the upper limits of normal should be determined according to the specific 2-cm segments studied across the elbow. Until now, only Azrieli et al.² have specified the upper

limits of normal for each 2-cm segment along the elbow; other investigators have only reported upper limits of normal of 1- or 2-cm segments without a subdivision of the specific area tested. Kanakamedala et al.¹¹ found a maximum latency difference in a 2-cm segment of 0.60 ms on the left side and 0.63 ms on the right side with a maximal distal-to-proximal reduction in the amplitude of the CMAP of 6% and 4.2%, respectively. Campbell et al. reported (number of controls not mentioned) a latency change over a 1-cm segment of more than 0.40 ms to be abnormal.⁷ Although our upper limits of normal for latency change are similar to these findings, our data emphasize the different upper limits of normal across successive 2-cm segments.

Azrieli et al.² reported that short-segment NCS is a very sensitive and specific diagnostic test for UNE, more sensitive than the presence of MCV slowing across the elbow. This conflicts with our findings, perhaps because of the reference values that these investigators used. In fact, they reported much lower upper limits for normal latency changes over different 2-cm segments across the elbow than others.^{7,11} In addition, they studied a small, relatively young control group of 15 subjects with 25 asymptomatic arms. This may further explain the high sensitivity of short-segment NCS in that study. In our control group, these upper limits of latency change of 0.4 to 0.5 ms over 2-cm segments were often exceeded, so it is likely that the values used by Azrieli et al. have a low specificity.

In all but two patients with CB on routine NCS, short-segment NCS showed a focal CB. The normal short-segment NCS in these two patients may be explained by the fact that both had CB on routine nerve conduction studies with values just above normal (one patient, 18% when recording from the ADM and 16% from FDI; the other patient, 21% from FDI and 4% from ADM). In some patients, MCV slowing is diffuse across the elbow. Thus, in 4 of the 31 patients with MCV slowing alone, no local increased latency change could be detected.

A potential weakness of our study is that we did not perform short-segment NCS in all patients with an UNE seen during the study period.³ There could have been a selection bias towards performing short-segment NCS more often in patients with CB on routine electrodiagnostic studies, because in this study 45% of patients had CB, whereas in a group of 102 patients whom we have studied this percentage was 34%.³

The site of focal CB or increased latency change was above the medial epicondyle in 52% of the patients, a finding which is in accordance with the

study of Herrmann et al.¹⁰ In the past, the humero-ulnar aponeurotic arcade has been the most common place of ulnar nerve entrapment,¹⁶ but this could not be confirmed by the data of Herrmann et al.¹⁰ or by our findings. A disadvantage of our study is that we did not make a comparison with intraoperative findings. However, Campbell et al.⁸ have shown a good (but not perfect) relationship between percutaneous and intraoperative short-segment studies. Studying a large group of patients, Kim et al. found that intraoperative electroneurography also located the lesion in most of the patients above the medial epicondyle.¹²

What can our data teach us about the possible pathogenic mechanisms in UNE? Nerve compression syndromes, such as UNE, may be caused by acute or chronic nerve injury. The electrophysiological consequences of these injuries are CB, MCV slowing, and axonal damage. In acute compression neuropathies, the physiological response is CB.⁹ Chronic injury due to stretch and traction of the nerve leads to demyelination, remyelination, and thickening of the perineurium and endothelium, with nerve enlargement as an end result.¹⁴ The electrophysiological hallmark in these patients can be MCV slowing across the elbow, with or without axonal damage on needle electromyography.

Our findings underline the fact that different pathogenic mechanisms occur in UNE, resulting in clinical and electrophysiological heterogeneity. Patients with CB had a shorter duration of symptoms, were significantly younger, and more often had muscle weakness than patients without CB. Although CB clearly correlates with weakness, this has not been documented before in patients with UNE, at least to our knowledge.

UNE occurred more often in men than women, as has been reported in two other studies.^{15,17} Richardson et al.¹⁷ suggested that external compression at the elbow is a more important cause of UNE among women than men. Matev¹⁵ recently reported that, in men, the ulnar nerve is more mobile and therefore more sensitive to gliding impairment at the medial epicondyle. External compression can cause CB and gliding impairment may result in MCV slowing, mechanisms which can explain our finding that women presented more often than men with CB, whereas men often had MCV slowing across the elbow without CB.

Herrmann et al.¹⁰ reported a correlation between the site of the lesion and clinical presentation of UNE. Their results suggested that patients with a block above the level of the medial epicondyle had a more subacute onset.¹⁰ Our data confirm that pa-

tients with CB had a significantly shorter onset of symptoms and the data showed a trend towards earlier onset of symptoms in patients with CB above the midpoint of the elbow. Incidental external compression at the superficial location of the ulnar nerve above the elbow may be the cause of UNE in these patients.¹⁰

The presence of muscle weakness is usually an indication for operation. However, when CB is a sign of demyelination occurring after external compression, it is possible that the outcome for such a lesion is favorable, especially when electromyography does not show abnormal spontaneous activity in the weakened muscles. Our recent follow-up study of 69 patients with UNE showed, using multivariate analysis, that signs of demyelination (CB or MCV slowing across the elbow) are independent prognostic factors indicating a favorable outcome.⁵

We conclude that short-segment NCS have additional diagnostic value in UNE, especially when measurement of latency changes is performed. Short-segment NCS are useful to locate the lesion, which is often located above the medial epicondyle. We suggest that short-segment NCS are performed in every patient with UNE, and not just those with CB on routine NCS.

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