An Electrophysiological Quantitation of the Cubital Tunnel Syndrome

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SUMMARY: *Four hundred and ninety-two ulnar nerves were studied in 237 patients with cubital tunnel syndrome and 233 subjects without clinically apparent ulnar neuropathy. Terminal motor and sensory latencies, motor and sensory conduction velocities, sensory action potential amplitudes and dispersions, and needle electromyography were analysed by a 0 to 10 rating — the EMG score. The patients were clinically graded from 1 (least severe) to 4 (most severe). The median EMG scores of these were 0.9/10 (N=70); 1.6/10 (N=81); 4.7/10 (N=46); and 7.1/10 (N=56), respectively. The median EMG score for the controls was 0.6/10 (N=239) and 14.7% had asymptomatic ulnar entrapments. This incidence reached 30% in controls who were 60 years or older. In 25 other controls, the above sulcus sensory action potential was analysed. Its mean dispersion was 4.9 ± 1.0 s-3. This measurement was significantly prolonged in 84.6% of 13 studies in which the EMG score was 0/10. These patients had either sensory symptoms only or an additional mild sensory deficit. Comparison of the EMG score obtained with sequential studies would allow one to objectively evaluate improvement or deterioration of the clinical state, giving a rational means of determining the need for surgery.*

RÉSUMÉ: *Nous avons étudié 492 nerfs cubitaux chez 237 patients avec syndrome du tunnel carpien et 233 sujets sans neuropathie cubitale cliniquement apparente. Nous avons établi un "score EMG" analysé de 0 à 10 pour les latences terminales motrices et sensitives, les vitesses de conduction motrices et sensitives, l'amplitude et la dispersion des potentiels d'action sensitifs et l'électromyographie à l'aiguille. Les patients furent classés cliniquement du grade 1 (le moins sévère) au grade 4 (le plus sévère). Les scores EMG médians furent 0.9/10 (n=70); 1.6/10 (n=81); 4.7/10 (n=46); et 7.1/10 (n=56) respectivement. Le score EMG médian pour les témoins était 0.6/10 (n=239) dont 14.7% avait des engorgements asymptomatiques. Ce chiffre atteint 30% chez les témoins âgés de plus de 60 ans. Chez 25 autres sujets témoins, le potential d'action sus-sulcus fut analysé. Sa dispersion moyenne était de 4.9 ± 1.0 s-3. Cette mesure était significativement prolongée dans 84.6% de 13 études dont le score EMG était de 0/10. Ces derniers patients n'avaient que des symptômes sensitifs ou un léger déficit sensitif additionnel. La comparaison séquentielle du score EMG permettrait une évaluation objective de l'amélioration ou de la détérioration de l'état clinique, et du moment de la décision chirurgicale.*

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Supported by the Muscular Dystrophy Association of Canada.

INTRODUCTION

A host of etiological factors have been incriminated as causing the cubital tunnel syndrome (Gay and Love, 1947; McGowan, 1950; Kopell and Thompson, 1963; Sunderland, 1968; Staal, 1970). In the majority of cases, however, this compression neuropathy of the ulnar nerve at the elbow arises without associated diseases or major trauma (Payan, 1969; Eisen, 1974). Spontaneous recovery of mild and occasional severe cubital tunnel syndrome occurs (Gay and Love, 1947; McGowan, 1950; Payan, 1970; Eisen and Danon, 1974) and therefore judicial selection of patients for surgical intervention is required.

Electrophysiological studies have contributed considerably to the diagnosis and localization of the syndrome (Kaeser, 1970; Nakano, 1978). Although a number of different electrophysiological characteristics have been shown to be abnormal in various combinations in the disease (Gilliatt and Thomas, 1960; Carpendale, 1966; Payan, 1969; Eisen, 1974), no single feature is abnormal in all patients (Payan, 1969; Eisen, 1974). Therefore, electromyographic improvement or deterioration of the disease during its course, one important indication for or against surgery, requires statistical analysis of each characteristic measured.

This study describes an electrophysiological quantitation system which correlates well with the clinical severity of the disease. It provides a single parameter for assessing changes in the neuropathy which can be used as a guide to management. A modification of the high-gain sensory conduction studies described by Payan (1969) is included. This was useful in localizing

https://doi.org/10.1017/S0317167100023799 Published online by Cambridge University Press

the lesion in patients with minimal or no neurological deficit.

METHODS

Subjects and patients

Two hundred and fifty-three ulnar nerves were studied in 237 patients with symptomatic cubital tunnel syndromes. The majority of these patients (87%) developed their disease spontaneously, although most admitted to frequently leaning upon their elbows. Thirty patients (11.9%) developed symptoms after recovery from general anaesthesia (28 cases) or coma (2 cases). Two other patients had sustained elbow injuries 3 and 35 years prior to the onset of symptoms. In another case, symptoms developed in association with the application of a plaster cast. Studies were also performed on 239 ulnar nerves in 230 subjects without clinically apparent ulnar neuropathy (the control group). One hundred and twenty of these had carpal tunnel syndromes, 5 had median nerve injuries, 2 radial nerve injuries, 1 a peroneal palsy, 5 lumbar disc disease, 1 Parkinson's disease, and the remaining 99 had no neurological abnormality. Subjects or patients were excluded from the study if they had clinical or electrophysiological evidence of 1. an ulnar nerve lesion at the wrist (Aguayo, 1975); 2. a lesion of the brachial plexus; 3. a thoracic outlet syndrome (Gilliatt et al, 1978); 4. disease of the cervical roots; 5. anterior horn cell disease; 6. a generalized polyneuropathy; 7. familial multiple entrapment neuropathy (Behse et al, 1972); and 8. exposure to neurotoxins.

The ulnar nerves studies were classified according to clinical severity as follows:

GRADE 0: asymptomatic (N = 239)

GRADE I: sensory symptoms (often intermittent) but no objective motor or sensory deficit (N = 70)

GRADE II: objective sensory deficit but no motor weakness (N = 81)

GRADE III: objective sensory deficit and mild motor weakness but no muscle wasting (N = 46)

GRADE IV: sensory deficit, motor weakness and muscle wasting (N = 56)

Electrophysiology

Motor and sensory conduction studies and needle electromyography were performed as previously described (Eisen, 1974). Below sulcus stimulation during motor conduction studies was, however, eliminated. Unless the nerve is superficial, percutaneous stimulation in this region results in significant error of both the calculated motor conduction velocity and amplitude of the evoked response (Schubert, 1964; Checkles et al, 1971). Electrophysiological results were quantified according to a rating system (Table 1), which was based upon previously published data in normal subjects (Gilliatt and Thomas, 1960; Payan, 1969; Eisen, 1974; Bhala, 1976; Eisen, Schomer and Melmed, 1977). Each electrophysiological abnormality was scored and the sum of these gave the EMG score for the patient. The maximum score possible is 10 because some abnormalities are mutually exclusive. The normal score is zero. Pre- and post-operative scores were compared in 10 patients who underwent surgical decompression of their ulnar nerves.

In an additional 25 normal subjects near nerve recording techniques (Buchthal et al, 1975; Rosenfalck, 1978) were used to study the above sulcus sensory potential evoked by stimulating the fifth digit. Amplitude, latency, and dispersion of the response were measured. To facilitate recognition of the slowest conducting myelinated sensory filbers, a high gain sensory amplifier (amplification capability of 0.5 uV/div) was used. The responses were electronically averaged and consecutively averaged traces following 32, 64, 128 and sometimes 256 sweeps respectively were compared. In this way it was possible to recognize which of the smallest peaks had "grown" and were therefore representatives of a true nerve potential. In contrast, those that got sequentially smaller were taken to reflect electronic noise. The same technique was used to record the above sulcus sensory action potential in 23 nerves from 13 patients. Thirteen of the limbs were symptomatic (Grade I or II above) but had shown no electrophysiological abnormality by the more usual studies. The other 10 nerves were from the contralateral asymptomatic limbs of the same patients.

Chi-square and students' t-tests were used in the statistical analysis. Correlation between clinical severity

TABLE 1

EMG Rating Used to Score Electrophysiological Severity of Cubital Tunnel Syndrome

Electrophysiological Abnormality		Score
Distal (wrist) motor latency	> 4.0 s^{-3}	1
Proximal (above sulcus) motor latency	> 8.9 s^{-3}	1
Motor conduction velocity from above sulcus to wrist	< 46 m^{-1}	1
Unobtainable motor response with:		
stimulation above sulcus		2
stimulation at wrist		3
Distal (wrist) sensory latency	> 2.4 s^{-3}	1
SNAP amplitude at wrist	< 8.0 μV	1
SNAP unrecordable at wrist		3
Needle electromyography:		
denervation		2
reduced recruitment		1
neurogenic motor units		1

Some abnormalities are mutually exclusive so that the maximum score possible is 10. The values given are the mean ± 2.5 SD of normal values (Eisen, 1974). SNAP = sensory nerve action potential. Needle electromyography refers to abnormalities found in the first dorsal interosseus and/or abductor digiti minimi muscles. Denervation = fibrillation and/or positive sharp waves. Neurogenic motor units = units having an increased duration, amplitude or number of phases compared to normal.

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TABLE 2

Clinical Grading and Values of Ulnar Nerve Conduction Studies. Results given as Mean ± SD. Ranges in Parenthesis.

Clinical severity	Mean age (yrs)	Mean duration of symptoms	DML (s^{-3})	PML (s^{-3})	MCV (m^{-1})	DSL (s^{-3})	Amplitude of SNAP (μV)
Asymptomatic (N = 239)	48.8 (17 - 88)	—	2.8 ± 0.4	7.5 ± 1.0	57.9 ± 11.0	1.9 ± 0.3	17.9 ± 10.2
GRADE I (N = 70)	45.6 (22 - 77)	16.4 months (2 days - 10 yrs)	3.0 ± 0.4**	8.0 ± 1.1***	55.1 ± 7.7	1.9 ± 0.4	17.5 ± 12.6
GRADE II (N = 81)	49.2 (16 - 70)	11.2 months (10 days - 9 yrs)	3.0 ± 0.5*	8.4 ± 1.2***	52.6 ± 8.0***	1.9 ± 0.3	10.4 ± 6.9***
GRADE III (N = 46)	49.8 (30 - 78)	9.6 months (6 days - 9 yrs)	3.3 ± 0.6***	9.4 ± 1.4***	47.4 ± 9.5***	2.3 ± 0.9***	6.6 ± 4.1***
GRADE IV (N = 56)	56.1*** (21 - 83)	34.4 months (4 wks - 53 yrs)	3.6 ± 1.1***	11.2 ± 3.6***	41.8 ± 10.8***	2.4 ± 0.6***	5.3 ± 5.3***

DML = distal (wrist) motor latency. PML = proximal (above-sulcus) motor latency. MCV = motor conduction velocity from above sulcus to wrist. DSL = distal (wrist) sensory latency. SNAP = sensory nerve action potential (wrist). *, **, *** = $p < 0.05, < 0.01$ and < 0.001, respectively when compared to controls.

and electrophysiological results were computed with Spearman's Rank Correlation test (r_s). A contingency coefficient test was used to detect significant association between the clinical severity and the EMG score.

RESULTS

The mean age of the symptomatic Grade IV patients was 56.1 years, which differed significantly from the asymptomatic controls and other patients (Table 2). The Grade IV patients also had the longest mean duration of their symptoms. There were significant correlations between clinical severity and the mean values of proximal motor latency ($r_s = +1.000$, $p < 0.05$); motor conduction velocity ($r_s = -1.000$, $p < 0.05$) amplitude of the sensory nerve action potential ($r_s = -1.000$, $p < 0.05$). There were positive, but non-significant, correlations between clinical severity and distal motor latency ($r_s = +0.975$) and distal sensory latency ($r_s = +0.894$).

Table 3 shows the frequency with which electrophysiological abnormalities occured in the symptomatic and asymptomatic nerves. There were positive correlations between the clinical severity and the incidence of abnormalities of each characteristic

TABLE 3

Percentage Frequency of Electrophysiological Abnormalities in 492 Ulnar Nerve Studies

	MOTOR			SENSORY			NEEDLE EMG		
Clinical Groups	DML > 4.0 s^{-3}	PML > 8.9 s^{-3}	MCV < 46 m^{-1}	DSL > 2.4 s^{-3}	SNAP < 8.0 μV	SNAP absent	Denervation	Abnormal recruitment	Neurogenic units
Asymptomatic (N = 239)	1.3	8.4	4.2	2.5	7.5	0.0	0.4	0.8	4.6
GRADE I (N = 70)	0.0	24.3	12.9	4.3	20.0	0.0	0.0	4.3	22.9
GRADE II (N = 81)	0.0	29.6	16.0	2.5	39.5	7.4	0.0	3.7	35.8
GRADE III (N = 46)	8.7	58.7	43.5	13.0	37.0	37.0	6.5	37.0	89.1
GRADE IV (N = 56)	19.6	71.4	53.6	16.1	37.5	51.8	53.6	89.3	96.4

DML = distal motor latency. PML = proximal motor latency. MCV = motor conduction velocity from above sulcus to wrist. DSL = distal sensory latency. SNAP = sensory nerve action potential. Denervation = fibrillation and/or positive sharp waves. Neurogenic units = motor unit potentials having an increased duration, amplitude and number of phases. In 8.9% of studies in the Grade IV group motor responses were absent.

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measured. The correlations were significant (r_s = +1.000, $p < 0.05$) for prolonged motor latency, slowed motor conduction velocity, low amplitude or absent sensory nerve action potential, and neurogenic motor units recorded on EMG. A single criterion was neither abnormal in all the symptomatic patients, nor in all of the Grade IV group who, however, showed the greatest number of abnormalities.

The percentage frequency distributions of the EMG scores in each group are shown in Fig. 1. All the patients with Grades III or IV lesions had scores above the normal zero. The median scores for each grade from 0 to IV were 0.6, 0.9, 1.6, 4.7, and 7.1, respectively, and there was a significant correlation between the clinical severity and EMG score (r_s = +1.000, $p < 0.05$). There was also a significant correlation between the clinical severity in any given individual and their respective EMG scores (contingency coefficient c = 0.68, $p < 0.001$). Table 4 shows that 35 (14.6%) of the control group had abnormal scores (i.e. > zero) indicating the presence of an occult cubital tunnel syndrome. In most of these (69%), the score was 2 or less. Thirty percent of the asymptomatic subjects with abnormal EMG scores were 60 years or older compared to an incidence of 10.6% in those subjects younger than 60 years (X^2 = 11.927, $p < 0.001$). The incidence of abnormal

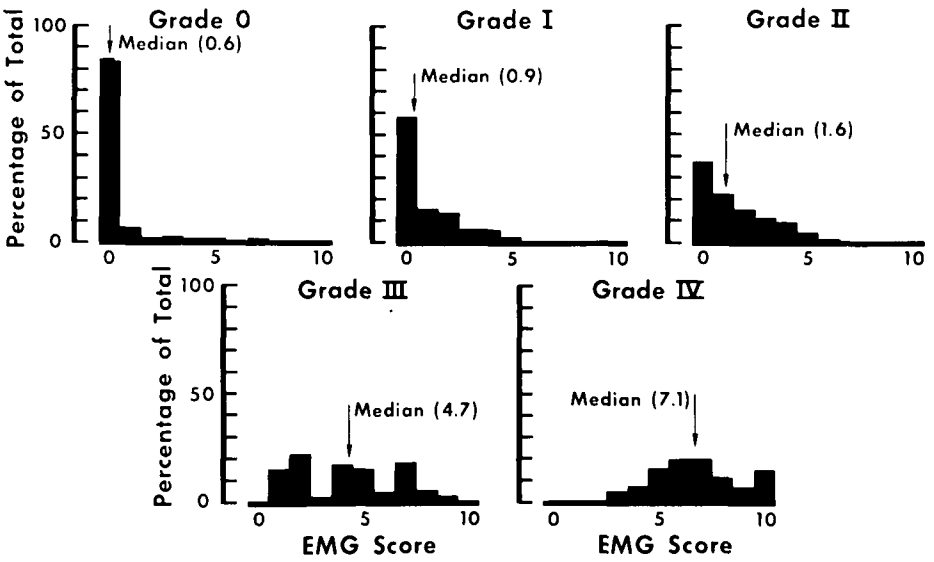

Figure 1 — Frequency distribution histograms of the EMG scores in controls (Grade 0) and patients with cubital tunnel syndromes (clinical Grades I to IV). The median score for each grade is indicated by the arrow.

scores was, however, not significantly higher in those of the control group with carpal tunnel syndromes (16.7%) than occurred in others (12.6%).

EMG scores were rated in 10 patients before and after surgery (Table 5). Pre- and post-operative median scores were 5.4 and 4.2, respectively. Five patients had improved scores following surgery; in 3 the score did not change and in 2 the score increased.

The EMG scores in 72 (28.5%) of the symptomatic studies were normal (0). These all belonged to clinical Grades I and II (Table 4). In these patients, the clinical impression of a cubital tunnel syndrome could not therefore be confirmed. The diagnostic value of electrophysiology might be improved in this type of patient by study of the above sulcus sensory action potential. This was done in 13 cases of mild cubital tunnel syndrome including 10 contralateral asymptomatic limbs of the same patients, and the results were

TABLE 4

Frequency Distribution of EMG Scores in the Asymptomatic and Symptomatic Ulnar Nerve Studies

	EMG SCORES*						
Clinical Groups	**0**	**1 - 2**	**3 - 5**	**6 - 8**	**9 - 10**	**Percentage With Abnormal EMG Score**	**Median EMG Scores****
Asymptomatic (N = 239)	204	24	10	1	0	14.6	0.6 (0 - 7)
GRADE I (N = 70)	41	20	9	0	0	41.4	0.9 (0 - 5)
GRADE II (N = 81)	31	29	20	1	0	61.7	1.6 (0 - 6)
GRADE III (N = 46)	0	17	16	12	1	100.0	4.7 (1 - 9)
GRADE IV (N = 56)	0	0	16	28	12	100.0	7.1 (3 - 10)

* scores grouped to simplify tabulation and facilitate analysis and ** range in parenthesis.

https://doi.org/10.1017/S0317167100023799 Published online by Cambridge University Press

TABLE 5

EMG Score Before and After Surgery

Patient	Duration of symptoms	EMG score before surgery	EMG score after surgery	Interval between surgery and second study
AY	2 yrs	5 – 3*	0	5 yrs
BO	2 yrs	3	5	6 mths
ES	?	6	4	9 mths
GN	2 mths	5	4	4 mths
GR	3 wks	2	1	10 mths
IN	3 yrs	8	7	3 yrs
LA	7 mths	8	7	3 mths
TE	4 mths	6	6	4 mths
PR	1 yr	7	3	2 yrs
TO	9 yrs	4	5	9 yrs

* patient had two electrophysiological studies, six months apart.

compared to 25 controls (Table 6). Patients and controls all had EMG scores of zero. The most significant abnormality occurred in the above sulcus sensory action potential dispersion (see Fig. 2). This was prolonged (> 6.9 msec, normal mean +2 SD) in 84.6% of symptomatic limbs and 50% of asymptomatic limbs. The means of the distal sensory action potential amplitude and dispersion and the proximal sensory action potential amplitude also differed significantly from the control means (Table VI). However, the individual incidence of abnormalities of these measurements were much lower than occurred in the proximal sensory action potential dispersion.

DISCUSSION

The diagnosis of a cubital tunnel syndrome could be confirmed in the majority of our patients by evaluation of; 1. the motor responses evoked by stimulation above the elbow and at the wrist, 2. the sensory action potential recorded at the wrist, and 3. needle electromyography of the ulnar supplied intrinsic musculature of the hand. The 28.5 percent of nerves in which this could not be done were from patients who either had no abnormal physical signs (clinical grade I) or a mild sensory deficit only (clinical grade II). In this type of case, study of the above sulcus sensory nerve action potential substantially added to the diagnostic yield (see Table 6). The dispersion or extent of desynchronization of the above sulcus sensory action potential proved to be the most useful characteristic to measure. This was significantly prolonged (> 6.9 s^{-3}) in 85 percent of the patients with clinical grades I or II in whom it was measured. Increased dispersion is a physiological reflection of demyelination which is the morphological hallmark of chronic entrapment neuropathy (Ochoa et al, 1972; Ochoa and Marotte, 1973; Neary et al, 1975).

There was an excellent correlation between the clinical severity of the ulnar neuropathy and the degree of electrophysiological abnormality, which was reflected in the EMG score (see Table 4 and Fig. 1). The EMG score

TABLE 6

Mean Values (± SD) of Ulnar Nerve Sensory Conduction Studies

	Distal sensory latency s^{-3}	Distal sensory dispersion s^{-3}	Distal sensory amplitude μV	Proximal sensory latency s^{-3}	Proximal sensory dispersion s^{-3}	Proximal sensory amplitude μV
Control (N = 25)	2.1 (± 0.3)	1.6 (± 0.3)	21.0 (± 7.6)	6.3 (± 0.9)	4.9 (± 1.0)	10.1 (± 5.0)
Contralateral Asymptomatic (N = 10)	2.3 (± 0.2)	1.7 (± 0.4)	14.5* (± 4.9)	7.3** (± 0.7)	7.2*** (± 1.5)	6.2* (± 2.7)
Mild "Cubital Tunnel Syndrome" (N = 13)	2.3 (± 0.3)	1.9** (± 0.3)	14.5* (± 5.5)	6.9 (± 0.6)	8.5*** (± 2.4)	6.2* (± 4.0)

*, **, *** = $p < 0.05, < 0.01, < 0.001$ respectively, when compared with values in the control group.

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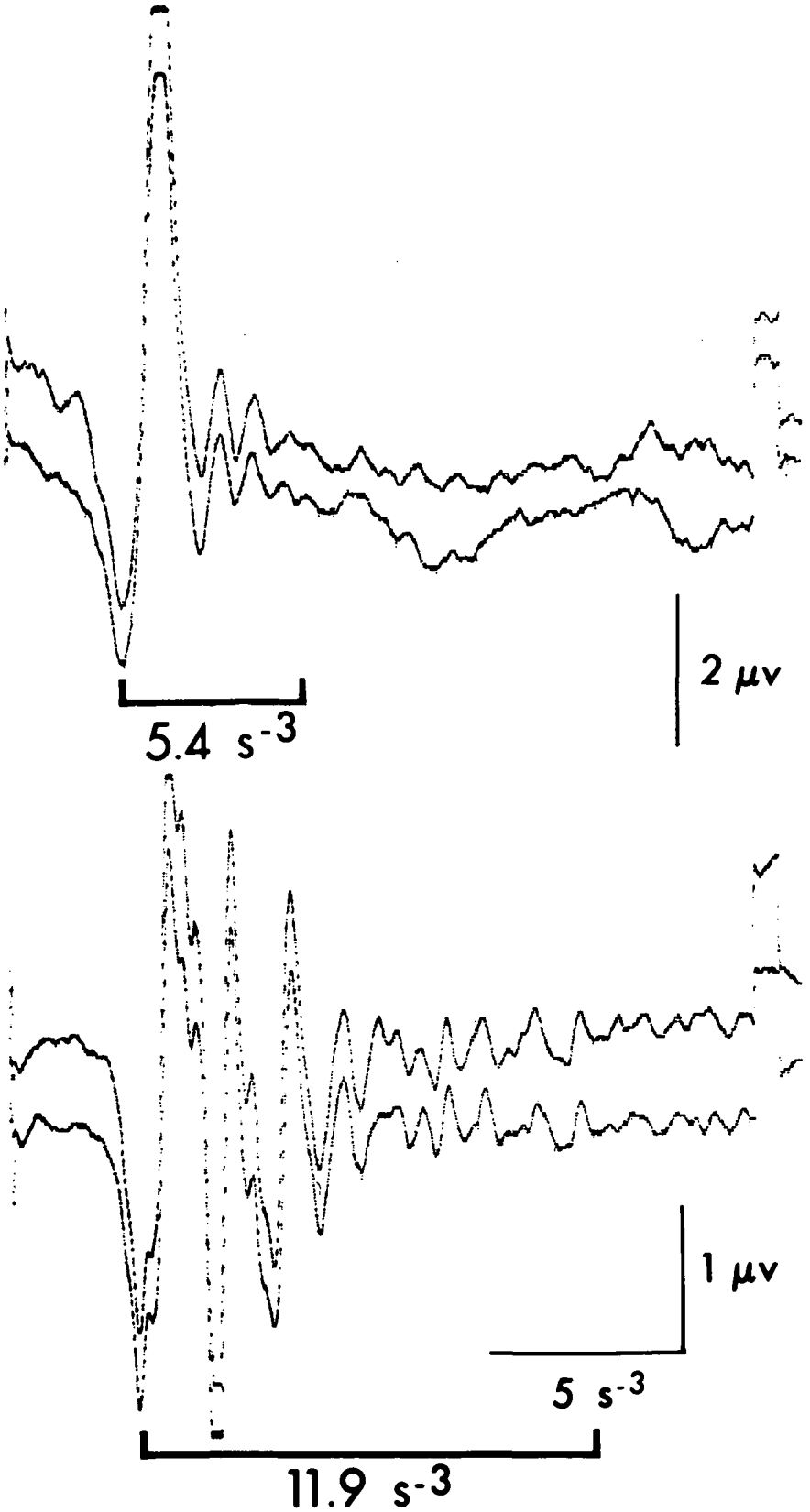

Figure 2 — Sensory action potentials recorded above the cubital sulcus in a normal subject (top pair) and patient (bottom pair). The labelled bars mark the total dispersion of the responses which are an average of 128 sweeps. The procedure was carried out twice in each case. Normal dispersion $< 6.9\ s^{-3}$. The patient had a mild sensory deficit and other physiological studies were normal.

can therefore be potentially useful in following the progress of the disease and its response to surgical or conservative management. It could also be of particular value in determining the necessity for surgery. Further work, however, is required before an optimum value, indicating surgery, can be determined. The mean EMG score of our small group of patients studied pre- and postoperatively did not change very much (5.4 before and 4.2 following surgery). This might suggest that surgery should be contemplated when scores are lower than 4. Certainly, if sequential studies show an increasing EMG score, this would favor surgical therapy. In contrast, a decreasing score would warrant conservative management. Changes in the EMG score, although not as sensitive as the more complex statistical analysis of individual characteristics (Payan, 1970; Harrison and Nurick, 1970; Eisen and Danon, 1974), are more easily appreciated by both the electromyographer and physician.

About 15 percent of our controls had abnormal EMG scores indicating a subclinical lesion. Asymptomatic disease of the ulnar nerve has been previously documented electrophysiologically (Payan, 1969; Eisen, 1974) and subclinical carpal tunnel syndrome is well recognized amongst electromyographers. Morphological confirmation of asymptomatic mononeuropathies has recently been described by Neary et al (1975). They studied 12 ulnar nerves obtained at autopsy from patients without ante mortem evidence of disease of the peripheral nervous system. Five (42%) had changes typical of those found in chronic experimental entrapment or pressure neuropathy (Ochoa and Marotte, 1973). The earliest abnormality is one of paranodal demyelination. This would be unlikely to cause any detectable electrophysiological abnormality. It is therefore not surprising that the incidence of asymptomatic disease revealed by morphological studies is higher than that disclosed electrophysiologically.

Asymptomatic entrapment neuropathies are increasingly frequent with age, and we found a 30 percent incidence in subjects over the age of 60 years. Occult cubital tunnel syndromes

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also occur in association with symptomatic carpal tunnel syndromes (Sedal et al, 1973). We found an incidence of 16.7 percent of this combination, about half that reported by Sedal et al (1973) and no different from the incidence of asymptomatic cubital tunnel syndromes found in our controls without carpal tunnel syndromes. Appreciation that both entrapments can occur together is important because it is common electromyographic practice to use the results of the non-symptomatic nerves as a control for the symptomatic nerve. This could result in a misleading or overemphasized interpretation (Gilliatt, 1978).

Almost 90% of our patients developed their cubital tunnel syndrome spontaneously. We believe that there are two interacting sites and mechanisms responsible for this form of the disease. One, probably the more important, is a compressive entrapment neuropathy of the nerve within the cubital canal. Both surgical and autopsy studies have shown macroscopic abnormalities at this site (McGowan, 1950; Feindel and Stratford, 1958a and b; Chang et al, 1963; Neary et al, 1975). Microscopically, the changes which typically are those of a chronic entrapment neuropathy (Ochoa and Marotte, 1973) are maximum under the tendinous arch joining the ulnar and humeral origins of the flexor carpi ulnaris muscle (Neary et al, 1975). When the arm is flexed, the canal is narrowed and the nerve's intraneural pressure increases (Strain and Olson, 1975; Pechan and Julis, 1975; MacGregor et al, 1975). This produces a swelling of the nerve proximal to the site of entrapment which is forcibly squeezed into the canal each time the arm is subsequently flexed. The relationship of the nerve to the canal is thus further compromised and a vicious cycle is set up.

The other mechanism of injury is probably one of repeated minor percussion to the nerve at a site somewhat proximal to the cubital canal. Recent experimental studies have shown that percussive injury produces a mixed pathology of segmental demyelination and axonal degeneration (Richardson and Thomas, 1978). This mechanism is probably responsible for those forms of cubital tunnel syndrome which remit spontaneously, an unlikely event in true entrapment neuropathy. Both mechanisms, i.e. entrapment within the cubital canal and percussion proximal to it, invariably occur together when leaning upon the elbow. Since this posture is repeated throughout life, it is not surprising that the incidence of asymptomatic disease and severity of symptomatic disease both increase with age.

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https://doi.org/10.1017/S0317167100023799 Published online by Cambridge University Press