Electrodiagnosis of the carpal tunnel syndrome

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In the carpal tunnel syndrome median nerve motor conduction may be delayed below the wrist, while the motor nerve fibre conduction velocity above the wrist may be relatively unaffected (Carpendale, 1956; Simpson, 1956). Median nerve sensory conduction may also be impaired below the wrist in the carpal tunnel syndrome (Gilliatt and Sears, 1958). No detailed study has been made comparing distal sensory and motor conduction in patients with the carpal tunnel syndrome. Such a comparison is important, since one mode of conduction may be affected before the other and knowledge of this would help to assess the best means of electrodiagnosis for the carpal tunnel syndrome as well as giving further insight into the condition.

INSTRUMENTS, ELECTRODES, AND METHOD OF STIMULATION

The stimulus was provided by an A.E.L. No. 104A laboratory stimulator using a fixed voltage of 250 V and a variable stimulus duration between 0·01 and 0·06 msec, and it was fed through a 1:1 low capacity isolating transformer into the stimulating electrodes. Nerve fibre impulses were amplified by a modified Tektronix 2A61 differential amplifier and displayed upon a Tektronix 564 storage oscilloscope. This was fired by a separate trigger pulse from the stimulator and filters were used to cut out frequencies below 50 c/sec and above 6 kc/sec.

Motor conduction was measured by using a similar technique to that of Hodes, Larrabee, and German (1948). The main median nerve trunk was stimulated at the wrist and the potentials, evoked from motor and sensory fibres (mixed conduction), were recorded at the elbow (Dawson and Scott, 1949). Digital sensory nerve conduction was measured at the wrist and elbow after simultaneously stimulating the thumb and first three fingers with ring electrodes (Kemble and Peiris, 1967). Using these techniques, sensory and mixed conduction times were measured to the peak of the initial positive deflection of the sensory and mixed nerve action potentials and motor conduction times were measured to the onset of the initial negative deflection of the muscle action potentials. All limbs were warmed before testing and surface temperatures were in all instances over 30°C.

A carpal tunnel syndrome (C.T.S.) was defined as a hand (or case) presenting with pain and/or paraesthesiae, which predominantly affected the fingers innervated by the median nerve, and for which there was no other identifiable cause. This broad definition was adopted in order to prevent bias in selecting cases, and in all instances a normal sensory conduction time between the fingers and wrist was obtained in the ipsilateral ulnar nerve (females 1·71 ± 0·20 msec).

RESULTS

Sixty-six female patients with carpal tunnel syndrome were examined in the E.M.G. clinic at Leeds General Infirmary during a six-month period. Males were excluded from the present study, since they were few in number and their normal ranges of median nerve conduction differ from those of females (Kemble, 1967). Electrophysiological measurements were considered to be abnormal if they were more than two standard deviations (2 S.D.) from the normal mean values and significance levels (P) were taken as two tailed estimates.

(1) ELECTROPHYSIOLOGICAL MEASUREMENTS OF MEDIAN NERVE CONDUCTION ON FIRST PRESENTATION OF CARPAL TUNNEL SYNDROME Mean electrophysiological measurements of median nerve conduction, from patients with the carpal tunnel syndrome, were compared with the corresponding normal mean values, from females with the same mean age, by using Student's t test (Table I). The distal sensory and motor latencies (D.S.L. and D.M.L.), the sensory nerve action potential duration at the wrist (S.N.A.P. dur.^{n} wrist), and the ratio of proximal to distal sensory nerve velocities (S_2/S_1) all showed highly significant increases from normal, while the $\log^{10}$ sensory nerve action potential amplitude at the wrist ($\log^{10}$ S.N.A.P. amp. wrist) showed a highly significant reduction from normal (P values <0·001). Other electrophysiological measurements of median nerve conduction showed a less marked but still significant difference or no significant difference from their normal mean values (Table I). Thus, the $\log^{10}$ of the sensory and mixed nerve action potential amplitudes at the elbow ($\log^{10}$ S.N.A.P. amp. elbow, $\log^{10}$ Mx.N.A.P. amp. elbow) and the mixed (Mx), sensory (S_2) and motor (Mo) velocities between the wrist and elbow were

TABLE I

ALL FEMALE CASES OF THE CARPAL TUNNEL SYNDROME, VARIATION FROM NORMAL ON FIRST PRESENTATION*

Electrophysiological Measurements	N	Mean	S.D.	Student's t test Significance (P)
D.S.L.	78	3·14	0·67	<0·001
	63	2·06	0·26	
D.M.L.	78	4·29	0·99	<0·001
	63	3·07	0·46	
$\log^{10}$ S.N.A.P. amp. wrist	78	1·28	0·35	<0·001
	63	1·88	0·14	
S.N.A.P. dur.^{n} wrist	59	1·41	0·39	<0·001
	63	1·21	0·21	
$\log^{10}$ S.N.A.P. amp. elbow	15	0·91	0·25	0·2 ~ 0·1
	63	0·97	0·22	
S.N.A.P. dur.^{n} elbow	10	1·93	0·37	0·005 ~ 0·001
	42	1·51	0·37	
$\log^{10}$ Mx. N.A.P. amp. elbow	62	1·31	0·23	0·1 ~ 0·05
	63	1·39	0·22	
Mx. N.A.P. dur.^{n} elbow	59	1·43	0·30	0·6 ~ 0·5
	30	1·35	0·15	
Mo	78	53·83	6·7	0·05 ~ 0·025
	63	55·71	4·5	
S_2	14	56·86	5·8	0·01 ~ 0·005
	63	61·65	5·1	
Mx	60	65·30	5·4	0·2 ~ 0·1
	30	66·83	3·9	
S_2/S_1	15	1·34	0·17	<0·001
	55	1·07	0·08	

*Normal values in italics, latencies and N.A.P. durations in msec, N.A.P. amplitudes in μV, and velocities in m/sec.

all reduced, while the sensory and mixed nerve action potential durations at the elbow (S.N.A.P. dur.^{n} elbow, Mx.N.A.P. dur.^{n} elbow) were increased compared with normal values.

(2) INCIDENCE OF ABNORMAL ELECTROPHYSIOLOGICAL MEASUREMENTS IN CARPAL TUNNEL SYNDROME The incidence of abnormal electrophysiological measurements was assessed in the hands of 66 female patients with carpal tunnel syndromes (Table II). Absent sensory nerve action potentials at the wrist or increased distal sensory latencies were associated with increased distal motor latencies in 69% of affected hands. Increased distal sensory latencies were the only electrophysiological abnormality in 23% of cases and in one affected hand the only abnormality was a reduced sensory nerve action potential amplitude at the wrist (distal latencies were however increased on the clinically more severely affected contralateral hand).

TABLE II

INCIDENCE OF ABNORMAL DISTAL ELECTROPHYSIOLOGICAL MEASUREMENTS OF MEDIAN NERVE CONDUCTION IN FEMALES AFFECTED BY CARPAL TUNNEL SYNDROME

Electrophysiological Abnormalities of Median Nerve Conduction	N	%
Absent N.A.P. at wrist* or D.S.L. > + 2 S.D. + D.M.L. > + 2 S.D.	83	69·1
D.S.L. alone > + 2 S.D.	28	23·5
S.N.A.P. amp. wrist < − 2 S.D. + normal D.S.L. and D.M.L.	1	0·8
No abnormalities	8	6·6
Totals	120	100·0

*After clinical improvement absent S.N.A.P. at the wrist returned with D.S.L.'s which were more than + 2 S.D. from normal mean values.

(3) COMPARISON OF DISTAL SENSORY AND MOTOR LATENCIES IN CARPAL TUNNEL SYNDROME Distal sensory and motor latencies were compared in patients affected by the carpal tunnel syndrome, since these measurements showed most departures from normal (Table II). There were highly significant positive correlation coefficients measured between distal motor and sensory latencies in patients who first presented with and who were improving from the carpal tunnel syndrome (P values <0·001 Table III). It will also be noted that the same relationship was significant in normal persons. Comparison of the 'z' transformations of these correlation coefficients (Table III) showed that they did not differ significantly from each other (P values >0·2).

The relationship between distal motor and sensory latencies in normal persons and in patients affected by the carpal tunnel syndrome, was plotted by using regression lines. These were virtually identical (Fig. 1). Therefore values were combined from normal females and from female patients affected by the carpal tunnel syndrome in order to obtain a more accurate estimation of the regression coefficient relating distal sensory and motor latencies. The formula for this 'combined' regression line is:

D.M.L. = 3·87 + 1·066 (D.S.L. − 2·75) msec

when the mean distal motor and sensory latencies are 3·87 and 2·75 msec respectively, the regression

TABLE III

CORRELATION COEFFICIENTS CALCULATED BETWEEN DISTAL MOTOR AND SENSORY LATENCIES (MSEC) IN HANDS AFFECTED BY CARPAL TUNNEL SYNDROME (C.T.S.) AND IN NORMAL PERSONS

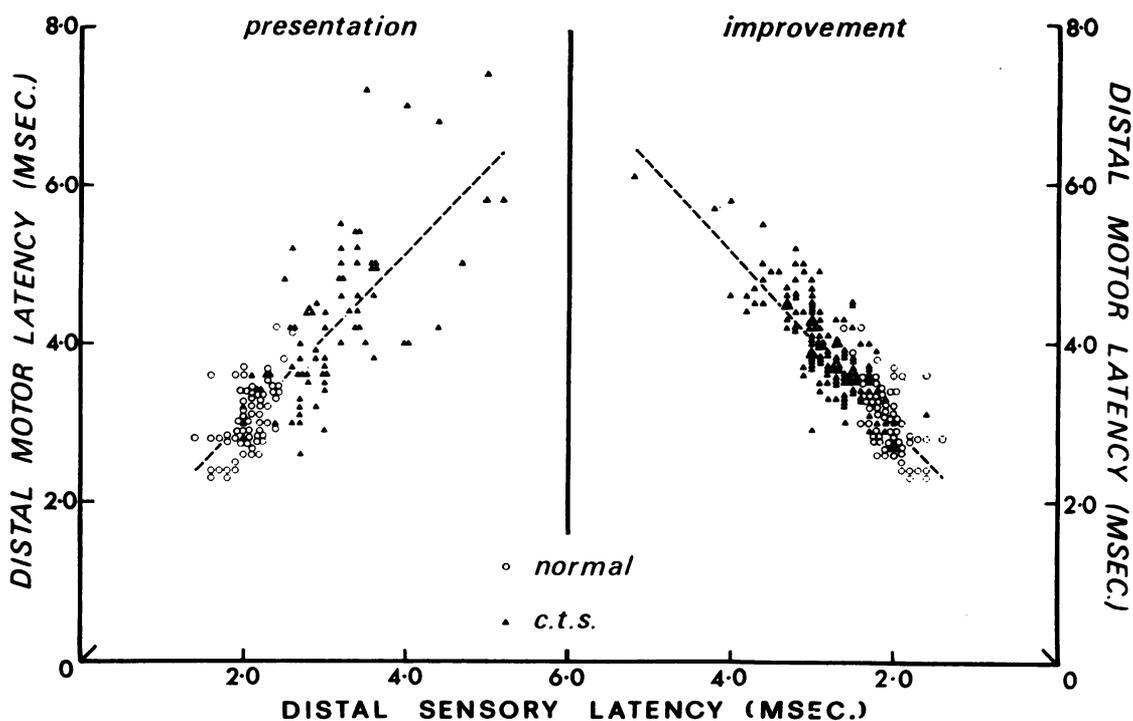| Groups tested	N	D.S.L.		D.M.L.		Correlation Coefficient (r)	Significance (P)
		Mean	S.D.	Mean	S.D.		
C.T.S. on presentation	78	3·14	0·67	4·29	0·99	+0·6900	<0·001
C.T.S. clinically improving	142	2·84	0·44	4·00	0·62	+0·7482	<0·001
Normal persons	63	2·06	0·26	3·07	0·46	+0·5715	<0·001
All cases of C.T.S. + normal persons	283	2·75	0·62	3·87	0·92	+0·7257	<0·001

FIG. 1.   *The relationship between distal sensory and motor latencies in female patients with the carpal tunnel syndrome.*

coefficient is 1·066, and D.S.L. and D.M.L. are the respective estimated values of the distal sensory and motor latencies in individual persons. The simplified version of the above formula is:

$$\text{D.M.L.} = 0.94 + 1.066 \text{ D.S.L. msec}$$

DISCUSSION

Evidence was presented, in cases where sensory nerve action potentials at the wrist could be recorded, to show that distal electrophysiological measurements were predominantly affected in the carpal tunnel syndrome (Table I). Measurements in proximal median nerve segments as far as the elbow also showed significant but less marked differences from normality in most instances when compared by using Student's t test. The most marked differences from normal were the increased distal sensory and motor latencies and sensory nerve action potential durations at the wrist and decreased $\log^{10}$ S.N.A.P. amplitudes at the wrist. The significant increase of S_2/S_1 ratios compared with normal ratios convincingly demonstrated the predominant distal slowing of conduction in the carpal tunnel syndrome. Previous authors described a preponderance of distal abnormalities of motor conduction compared with

measurements in more proximal median nerve segments (Carpendale, 1956; Simpson, 1956; Thomas, 1960). Thomas (1960) also demonstrated a significant but less marked reduction of the mean proximal motor velocity between the wrist and elbow measured in cases of the carpal tunnel syndrome and compared with normal persons.

Assessment of distal electrophysiological measurements of median nerve conduction in the carpal tunnel syndrome showed that by far the largest number of abnormalities (92·6%) were absent sensory nerve action potentials at the wrist or increased distal sensory latencies (Table II). Distal motor latencies were also increased in a large proportion of cases (69·1%), but in all instances they were associated with abnormal distal sensory latencies or unrecordable sensory nerve action potentials at the wrist. Cseuz, Thomas, Lambert, Love, and Lipscomb (1966), and Kaeser (1963) described high incidences of abnormal distal latencies (85%). similar to those described in the present study. However, most previous series have shown smaller percentages of abnormal electrophysiological measurements, which may have been due to too much reliance being placed only on distal motor latencies as a diagnostic index.

The results in Table II indicated that the distal sensory latencies were more accurate than the distal motor latencies as a means of diagnosing carpal tunnel syndromes, and occasionally the reduced S.N.A.P. amplitude at the wrist also indicated that a carpal tunnel syndrome was developing. Most previous series have found more sensory than motor nerve abnormalities. Thomas and Lambert (1962) stated that sensory nerve fibres may be involved selectively in the carpal tunnel syndrome and if motor fibres were affected there was almost always a disturbance of sensory nerve conduction as well. One third of Copp's (1965) cases of the carpal tunnel syndrome had sensory abnormalities only and Cseuz *et al.* (1966) described sensory abnormalities in 85% and motor abnormalities in 69% of a large series of patients with carpal tunnel syndromes. On the other hand, Goodman and Gilliatt (1961) found that either the D.S.L. or the D.M.L. could be abnormal, while the other was within the normal range. None of the previous series gave any statistical evidence as to why there were more sensory than motor nerve abnormalities.

There were two possible reasons for the fact that distal sensory latencies were abnormal more often than distal motor latencies. Firstly, the standard deviation of the normal D.S.L.'s estimated as a percentage of the normal female mean value was less than the same measurement applied to the normal D.M.L.'s (Table III). This finding provided some evidence that D.S.L.'s were more accurately measured compared with D.M.L.'s in this study and would explain why minor variations from normal would therefore be detected by measuring D.S.L.'s. Secondly, distal sensory latencies could have been increased to a greater extent than distal motor latencies. This possibility was assessed by calculating correlation and regression coefficients relating distal sensory and distal motor latencies. These relationships were statistically significant in normal persons and in all groups of patients whether they were presenting with, or improving from, the carpal tunnel syndrome, irrespective of aetiology. Furthermore, there was no significant difference between these relationships in different groups of patients affected by the carpal tunnel syndrome or between normal persons. These findings therefore indicated that the distal motor and sensory latencies retained a constant relationship to each other (Fig. 1), and could be expressed as a single regression line for normal persons and patients with the carpal tunnel syndrome. (D.M.L. = 0·94 + 1·066 . D.S.L.). Observation of this formula shows that an increase of the distal sensory latency from 2 to 4 sec (100%) would be associated with an approximate increase of the distal motor latency from 3 to 5 sec (66%).

Therefore distal sensory and motor latencies varied together but the D.S.L. showed a more marked variation than the D.M.L., both in normal persons and in patients with the carpal tunnel syndrome. This meant that the distal sensory latencies measured in the carpal tunnel syndrome would show proportionately greater increases from normal when compared with distal motor latencies. A possible interpretation is that the larger sensory fibres were reduced in diameter to a greater extent than the slightly smaller motor nerve fibres in patients with the carpal tunnel syndrome, since nerve fibre diameter is inversely proportional to nerve fibre conduction time (Tasaki, Ishii, and Ito, 1944). This explanation would apply if the relative proportions of the distal motor latency due to the residual latency and the motor nerve fibre conduction time remained constant. There is some evidence that larger nerve fibres are reduced in diameter more than smaller nerve fibres under the flexor retinaculum in patients with the carpal tunnel syndrome (Thomas and Fullerton, 1963). The histological lesion is in all probability localized segmental demyelination, since this has been shown to occur in the pressure neuropathy of the hind foot of the guinea-pig (Fullerton and Gilliatt, 1967).

SUMMARY

Most electrophysiological measurements of median nerve conduction in the carpal tunnel syndrome showed differences from normal values, although measurements were more markedly abnormal in distal compared with proximal nerve segments.

The distal sensory latencies afforded a better means of electrodiagnosis for the carpal tunnel syndrome than distal motor latencies, partly because they were more accurately measured but also because they were affected to a greater extent than distal motor latencies. It is suggested that the latter finding is due to the large digital sensory nerve fibres being affected more than slightly smaller motor nerve fibres, possibly because of involvement by localized segmental demyelination of nerve fibres in the carpal tunnel.

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