Percutaneous Localization of Conduction Abnormalities in Human Entrapment Neuropathies

W.F. BROWN and S.K. YATES

SUMMARY: *In human entrapment neuropathies the characteristic abnormalities in conduction are frequently limited to a short segment of the nerve. Recognition and precise localization of these discrete conduction abnormalities may require measurement of conduction over shorter lengths of the nerves than those lengths commonly employed in the clinical laboratory. Techniques are described for the more precise location of the primary conduction abnormalities in median, ulnar and peroneal nerve entrapments. Distinctive or atypical locations of the major conduction abnormalities may point towards different mechanisms in the pathogenesis of these localized neuropathies.*

RÉSUMÉ: *Dans les neuropathies compressives humaines les anomalies caractéristiques de conduction sont souvent limitées à un court segment du nerf. Pour reconnaître et localiser convenablement ces anomalies discrètes de conduction il faut modifier les méthodes courantes et mesurer la conduction sur de plus courtes distances. Nous décrivons des techniques de localisation pls précises pour les neuropathies compressives des nerfs médian, radiaux et péronnés. Certaines localisations distinctes ou atypiques de ces anomalies indiquent peut-être des mécanismes pathogéniques différents pour ces neuropathies localisées.*

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INTRODUCTION

Entrapment neuropathies are common. Possible physiological abnormalities in these 'entrapped' nerves include conduction delay, conduction block and possibly wallerian degeneration. To establish the level of the injury to the nerve, it is necessary to demonstrate abnormalities in conduction at the level of the entrapment which: (1) are not present proximal or distal to the level of the injury, or (2) exceed those abnormalities in conduction which may be present proximal or distal to the level of the entrapment.

Conventional electrophysiological techniques for the assessment of peroneal and ulnar nerve entrapments measure conduction over relatively long segments of these nerves (100-120 mm) (Payan, 1969; Singh, et al, 1974; Eisen, 1974; Eisen and Danon, 1974) and even in the carpal tunnel entrapment, over 100 mm in sensory fibers and 60-80 mm in motor fibers (Simpson, 1956; Thomas, 1960; Thomas, et al, 1967; Buchthal, et al, 1974).
1974).

In previous intra-operative investigations, it was learned that when the nerves were stimulated at 5-10 mm intervals, the most abnormal and sometimes only abnormal segment could be as short as 5-10 mm in length (Brown, et al, 1976). Over this short length, conduction was much more abnormal than over the much longer 100-120 mm lengths employed to measure conduction across the levels of the entrapments by conventional techniques (Payan, 1969; Singh, et al, 1974; Eisen, 1974 and Eisen and Danon, 1974). Indeed, with these techniques, conduction velocities could be in the normal range, when the abnormal segments are very short in relation to the overall length of nerve over which their velocities were measured.

Recognition and localization of discrete conduction abnormalities requires 'a priori' that measurements are taken over the shortest segments of nerve practical. Stimulation at short distance intervals has been advocated before in the diagnosis of human entrapment neuropathies (Kimura, 1978; Kimura, 1979; Miller, 1979; Brown and Yates, 1981). These techniques have advantages in the electrophysiological diagnosis of entrapment neuropathies but have important technical limitations.

It is our intention to present here our observations with these techniques in median, ulnar and peroneal nerve entrapments. Observations, based on our now extensive experience with intra-operative localization of the various entrapment neuropathies are included to illustrate, where relevant, certain points but will be the subject of a more extensive report now in preparation.

METHODS

(1) *Techniques for percutaneous stimulation:*

For these investigations DISA 13K62 percutaneous bifocal electrodes were chosen. Other bifocal electrodes with different interelectrode distances and monopolar stimulation offered no advantages in terms of better localization or ease of testing. Stimulus pulses were brief (0.05 - 0.1 ms) and adjusted to be just supramaximal to evoke the related maximum 'M' response or antidromic digit sensory nerve action potential. For the median nerve the technique described by Kimura (1978, 1979) was adopted (Figures 1 and 2). The nerve was stimulated at 20 mm intervals proximal (-) and distal (+) to the main wrist flexor crease. Provided stimulus artifact was not excessive, the nerve could be stimulated as close as 50 mm from the proximal digital ring electrode (DISA 13L69).

The ulnar nerve was stimulated at 20 mm intervals proximal (-) and distal (+) to the tip of the medial epicondyle

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(0) to include the retro-epicondylar and trans-cubital tunnel segments.

Likewise the peroneal nerve was stimulated at 20 mm intervals both proximal (-) and distal (+) to the lateral tip of the head of the fibula (0). It was, however, hard to stimulate the peroneal nerve much more than 60 mm distal to '0' because the common peroneal nerve divides into its major superficial and deep divisions at this level, the deep peroneal branch which innervates EDB turning deep to the extensor digitorum longus muscle. The situation is akin to the ulnar nerve, where just beyond the entrance to the cubital tunnel the ulnar nerve penetrates between the two heads of the flexor carpi ulnaris to lie beneath that muscle and on the surface of the flexor digitorum profundus. The result in both instances was unavoidable uncertainty, especially with percutaneous stimulation techniques, about the exact level at which the respective nerves were excited in relation to the overlying cathode. For this reason, attempts to stimulate with percutaneous electrodes more than 20 mm distal to the entrance of the cubital tunnel or 40 mm beyond the fibular head were abandoned.

(2) *Measurement of the trans-retinaculum conduction velocity in the median nerve:*

The techniques were similar to those reported by Wiederholt (1970), Buchthal and Rosenfalck (1971) and Daube (1977). The 2nd or 3rd digits were stimulated through DISA 13L69 electrodes (cathode proximal) and the maximum orthodromic sensory nerve action potential recorded just distal and proximal to the flexor retinaculum and 50-100 mm proximal to the flexor retinaculum. Bi-polar near nerve recording with DISA 13L64 electrodes was employed (inter-electrode distance 30 mm).

(3) *Parameters measured:*

The thenar (T), hypothenar (HT), and extensor digitorum brevis (EDB) maximum 'M' responses were recorded by surface electrodes in all instances. The location of the 'stigmatic' electrode was adjusted to obtain the maximum peak-to-peak voltage (p-pV), least time to negative peak and an initial negative deflection. Measurements of p-p voltage (p-pV) and p-p duration (p-pD) were taken of the maximum 'M' response to look for evidence of abnormal temporal dispersion.

Less than a 5% reduction in p-pV of the 'M' response was observed in control subjects over conduction distances of 100-200 mm in the ulnar and peroneal nerves. There were parallel

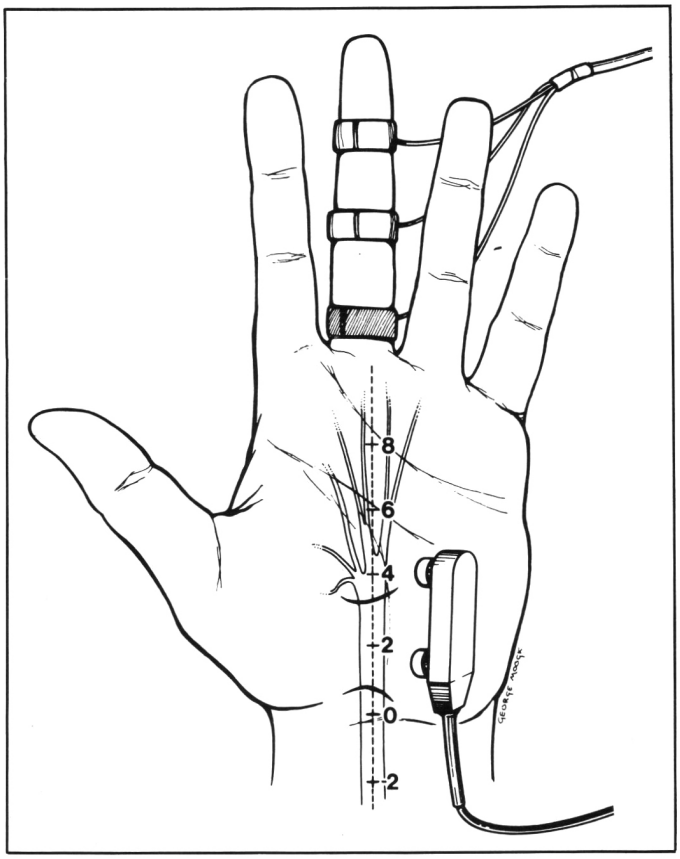

Figure 1 — Illustration of the percutaneous method to measure the maximum conduction velocities of sensory fibers at 20 mm intervals proximal to, through and distal to the flexor retinaculum. The maximum antidromic sensory nerve action potential was recorded by bi-polar electrodes about the interphalangeal joints (stigmatic electrode proximal). The earth electrode was located at the base of the digits.

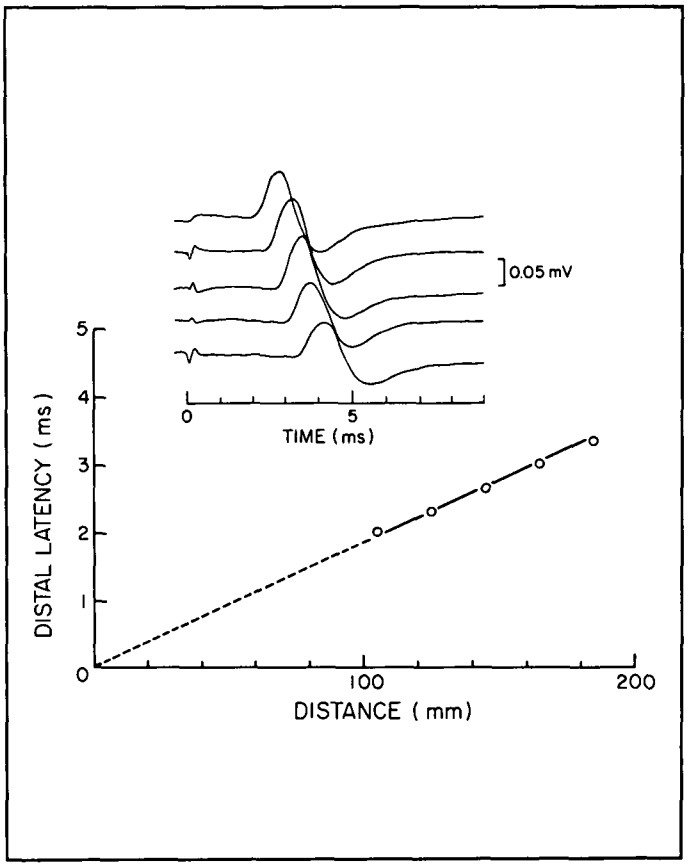

Figure 2 — *Top:* Illustration of a series of second digit antidromic sensory nerve action potentials evoked by stimulation at 20 mm intervals proximal to, across and just distal to the flexor retinaculum.
Bottom: Plot of the distal latencies to onset of the above potentials at varying distances from the stigmatic recording electrodes.

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equivalent increases in the p-pD of the 'M' response. It did not prove necessary in the majority of nerves to measure the 'M' response areas. Conduction block was judged to be present if a reduction of over 20% in the p-pV over a 120 mm or shorter length of nerve was observed provided no reciprocal and proportionate increase in 'M' response duration or other evidence of temporal dispersion was observed.

(4) *Other electrophysiological techniques:*

Concentric needle examination of the flexor carpi ulnaris, hypothenar and first dorsal interosseus muscles in the ulnar nerve distribution and tibialis anterior, extensor digitorum longus, peroneus longus and EDB muscles in the peroneal nerve distribution was included to look for denervation to suggest the presence of wallerian degeneration in the respective motor nerve fibers. Less direct evidence of wallerian degeneration in sensory fibers was looked for by recording the amplitudes of the orthodromic sensory nerve action potential recorded at the wrist in response to stimulation of the 5th digit for the ulnar nerve and the superficial peroneal sensory nerve action potential recorded proximal to the ankle in response to stimulation of the distal branches on the dorsum of the foot. All the electrophysiological investigations were carried out at least three weeks following the onset of the neuropathy.

(5) *Subjects*

In the median nerve, two patient groups were examined. The percutaneous stimulation technique was limited to patients who had abnormal prolongations of the motor terminal latencies to their thenar muscles and in the latencies of their orthodromic digital sensory nerve action potentials (latencies exceeded the mean values of controls + 2 s.d.). Conventional electrophysiological techniques were employed for both tests (Simpson, 1956; Thomas et al, 1967; Buchthal and Rosenfalck, 1971). A larger group of thirty patients with complaints typical of carpal tunnel entrapments but who had normal sensory and motor terminal latencies were examined by the orthodromic trans-retinaculum technique. No patient in the above groups had evidence of a more general neuropathy or systemic disease. In the ulnar and peroneal neuropathies, the possibly important mechanisms and systemic disorders are described in the results.

RESULTS

(1) *Peroneal Nerve:*

Twelve peroneal neuropathies were included. Though the majority had no obvious systemic disorder, there were two alcoholic patients, one patient with polyarteritis nodosa and one diabetic patient, who had bilateral peroneal neuropathies which developed spontaneously within ten weeks of one another. In control subjects the maximum motor conduction velocities and the EDB maximum 'M' response p-pV and p-pD are included in Table 1. It is important to note that less than a one percent reduction in p-pV and no significant change in negative peak area or p-pD were observed across the 100-200 mm segment of the common peroneal nerve between the popliteal fossa and fibular head.

In all twelve peroneal neuropathies the maximum motor velocities between the fibular head and popliteal fossa were below the two standard deviation lower limit in control nerves. Over this same segment, reductions in the EDB 'M' response p-pV of over 40% (mean 78%, range 47% - 94%) were observed in ten of the neuropathies. Conduction block was therefore present in nearly one half of the total remaining EDB motor fiber population in these ten neuropathies.

The locations of the most abnormal increases in conduction time measured over 20 mm segments in the peroneal neuropathies, are illustrated in Table 2. The most common location of the worst conduction delay was just beyond the fibular head (five); less frequent locations being across the level of the fibular head (two) or proximal to the latter (Figures 3,4 and 5). In two peroneal neuropathies the most abnormal segment was more than 40 mm proximal to the fibular head. One of the latter two patients had polyarteritis nodosa.

In the ten peroneal neuropathies which had obvious conduction block, the location of the predominant block was either identical to the segment with

TABLE 1: *Tabulation of maximum motor conduction velocities (MCV) in normal and abnormal peroneal nerves between the ankle and the fibular head and between the fibular head and popliteal fossa. Shown also are the maximum p-pV in mV and p-pD in ms of the EDB 'M' responses evoked by stimulation of the peroneal nerve at the levels of the ankle, fibular head and popliteal fossa.*

A Ankle
FH Fibular head
PF Popliteal fossa

Peroneal nerve:
Control Values:

		A	FH	PF
EDB	MCV (M/s)	50.0		57.2
	± 1 S.D.	5.0		6.5
	p-pV (mV)	14.1	12.3	12.2
	± 1 S.D.	3.1	3.1	3.1
	p-pD (ms)	4.4	4.4	4.5
	± 1 S.D.	0.7	0.7	0.7
Peroneal Neuropathies:				
MCV (M/s)		42.8		26.1
range		(35.3 - 53)		(14.2 - 37.7)

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TABLE 2: *Locations of the maximum reductions in conduction velocity or EDB 'M' response p-pV (or both) in the peroneal nerve with reference to the fibular head.*

Peroneal nerve:

Location (mm) with reference to the F.H.	Number of nerves
+40 to 0	1
+40 to +20	1
+20 to 0	3
+10 to -10	2
0 to -40	1
-40 to -60	1
-60 to -80	1

\+ distal
\- proximal

the most abnormal increase in conduction time (Figure 3 lower, Figure 4 and Figure 5) or the two segments were adjacent (Figure 3 top). The mean reduction in p-pV over a 20 mm segment in the peroneal neuropathy group was 72% (range 53% - 95%).

In ten of the twelve neuropathies there were fibrillation potentials and positive sharp waves in one or more of the muscles innervated by the peroneal nerve. The superficial peroneal nerve was examined in ten subjects and the sensory nerve action potential was found to be absent in four and the amplitude abnormally low in one of the remaining six nerves.

(2) *Ulnar nerve:*

Seventeen ulnar neuropathies were included in this investigation. Etiological factors of possible importance are listed in Table 5. In healthy subjects the HT 'M' response, p-pV, negative peak area and p-pD are almost constant when the nerve is stimulated between levels 40 mm distal and 60 mm proximal to the tip of the medial epicondyle. However, in ten abnormal ulnar nerves where there was no obvious increase in the p-p duration of the HT 'M' response to suggest abnormal temporal dispersion, a substantial reduction in p-pV (20 - 96%) was observed across the elbow, a length which included the trans-cubital tunnel and retroepicondylar segments. Similar evidence of probable conduction block was present in four other ulnar neuropathies but evidence of temporal dispersion in the 'M' response precluded quantitation of the degree of conduction block. In healthy subjects the maximum motor conduction velocity measured across the elbow segment (80-100 mm) was practically identical to that across the forearm (Table 3). However, abnormally low maximum motor conduction velocities (less than 2 S.D. lower limit of controls) were present across the complete elbow segment in all the ulnar neuropathies.

The locations of the 20 mm segment(s) which had the maximum conduction block and delay relative to the tip of the medial epicondyle are illustrated in Table 4. It was obvious that the locations of the major conduction abnormalities were primarily concentrated distal or proximal to the tip of the medial epicondyle. There was exact correspondence between the locations of the segments with the maximum conduction delay and block in all but one ulnar neuropathy. In two ulnar

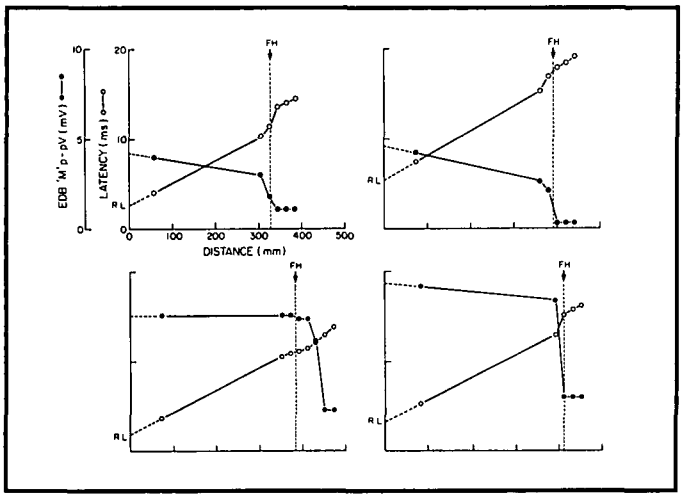

Figure 3 — Plots of the p-pV and least latencies to the EDB 'M' responses evoked by stimulation of the peroneal nerve at 20 mm intervals proximal and distal to the fibular head and at the ankle in 4 illustrative patients.
Note: In the example in the lower right the 20 mm segments which had the maximum reduction in p-pV and largest conduction delay corresponded whereas in the example on the top left these segments did not correspond.
In the bottom left example the major conduction block was in the segment 40 to 60 mm proximal to the head of the fibula unlike the other three more typical examples where the primary abnormalities were adjacent to the fibular head.
F.H. location of the tip of the fibular head
R.L. residual latency

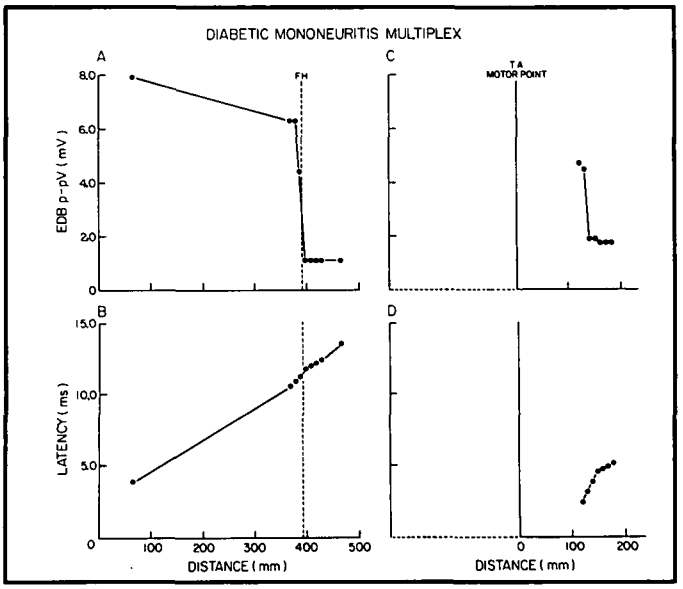

Figure 4 — Plots of the EDB (A and B) and tibialis anterior (C and D) maximum 'M' response p-pV and latencies at 20 mm intervals proximal and distal to the fibular head and in the case of EDB, at the level of the ankle. The locations of the maximum reductions in EDB and T.A. p-pV were identical, namely the 20 mm segment bridging the lateral tip of the fibular head. The location of the T.A. motor point is illustrated by the solid vertical line and the distances are shown with reference to this motor point.
F.H. fibular head
T.A. tibialis anterior

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neuropathies the most obvious conduction abnormalities were distributed over a longer length of the nerve (30-40 mm). In some of the ulnar neuropathies the maximum conduction velocities over the worst 20 mm segment were very low (4.3, 5.9, 6.3 and 8.0 meters per second). The maximum p-pV reductions in the HT 'M' responses over the worst 20 mm segments in the ulnar neuropathies were between 14.8 and 90 percent (mean forty-four percent). Hence, the maximum conduction velocity may be very abnormal and substantial conduction block present in a relatively short segment of the nerve (Figures 6 and 7).

The locations of the major conduction abnormalities in the ulnar nerve were distal to the medial epicondyle in those neuropathies which followed a fall and a blow to the elbow respectively (Table 5). Both post-operative ulnar neuropathies were located proximal to the medial epicondyle. There was no obvious relationship between the locations of the primary conduction abnormalities and the clinical histories or examinations in the remaining patients. No fractures, dislocations, major deformities or arthritis involving the elbow were included in the present study.

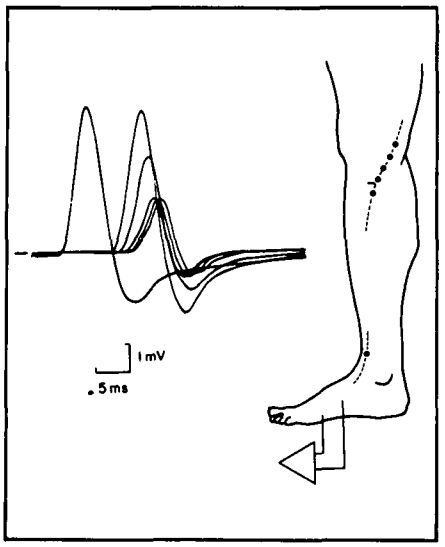

Figure 5 — Illustration of the changes in EDB maximum 'M' response (left) and the locations (right) of the stimulation points proximal and distal to the lateral tip of the fibular head and just proximal to the ankle. Note, the major conduction delays and reduction in EDB p-pV occurred across the 20 mm segments just proximal and distal to the fibular head.

TABLE 3: *Tabulation of the maximum motor conduction velocities (MCV) measured across the forearm and elbow segments in control and ulnar neuropathies.*
** distance measured with the elbow in 90° flexion and over a total distance of 100-120 mm which included the entrance to the cubital tunnel and retro-epicondylar regions.*

Ulnar nerve:

		Maximum MCV (M/s) ± 1 S.D.	2 S.D. lower limit
Control	Forearm segment	62.2 ± 6.8	48.6
	Elbow segment*	60.7 ± 4.3	52.1
		Maximum MCV (M/s)	**Range**
Ulnar Neuropathies	Forearm segment	54.5 ± 7.3	44.9 - 64.0
	Elbow segment*	33.1 ± 10.4	13.5 - 47.6

TABLE 4: *Locations of the maximum reductions in HT p-pV and maximum conduction delays for a 20 mm segment in the ulnar neuropathies. In 2 ulnar neuropathies the abnormal segments were longer than 20 mm (see text).*

Ulnar nerve:

Location proximal (-) and distal (+) to the F.H.	Number of ulnar nerves	
	Maximum conduction block	**Maximum conduction delay**
20 mm segment or less		
+30 to +20	1	
+20 to 0	5	5
+10 to -10	2	2
0 to - 20	5	6
greater than 20 mm length		
+20 to -20		1
-10 to -40		1

TABLE 5: *Location of the segment(s) with the most abnormal conduction in the ulnar nerve with reference to the medial epicondyle in relation to factors of possible etiological significance.*

Ulnar nerves:

Location of primary conduction abnormalities in relation to the medial epicondyle tip.

Etiology	Proximal	Across	Distal	Total
Unknown	4	2	4	10
Post-operative	2	0	0	2
Post-traumatic	0	0	2	2
Diabetes mellitus	1	0	0	1
Rheumatoid arthritis	0	1	0	1

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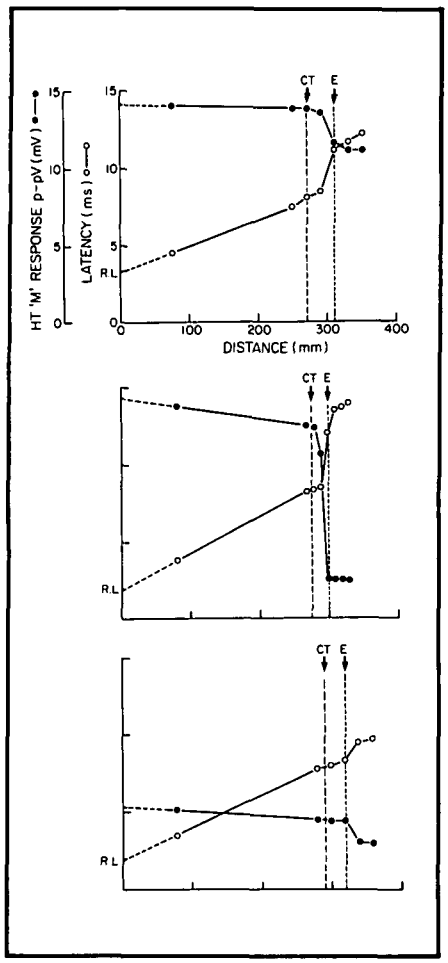

Figure 6 — Plots of the maximum p-pV and least latencies of the HT 'M' responses evoked by stimulation of the ulnar nerve at 20 mm intervals both proximal and distal to the tip of the medial epicondyle and at the level of the wrist in 3 illustrative patients. The relative positions of the tip of the medial epicondyle (E) and the entrance to the cubital tunnel (CT) are indicated by the interrupted vertical lines.
In the top and middle examples the major reductions in p-pV and increases in conduction time were distal to the epicondyle, whereas in the bottom illustration the major conduction abnormalities were proximal to the epicondyle (see Figure 7 for a similar example).

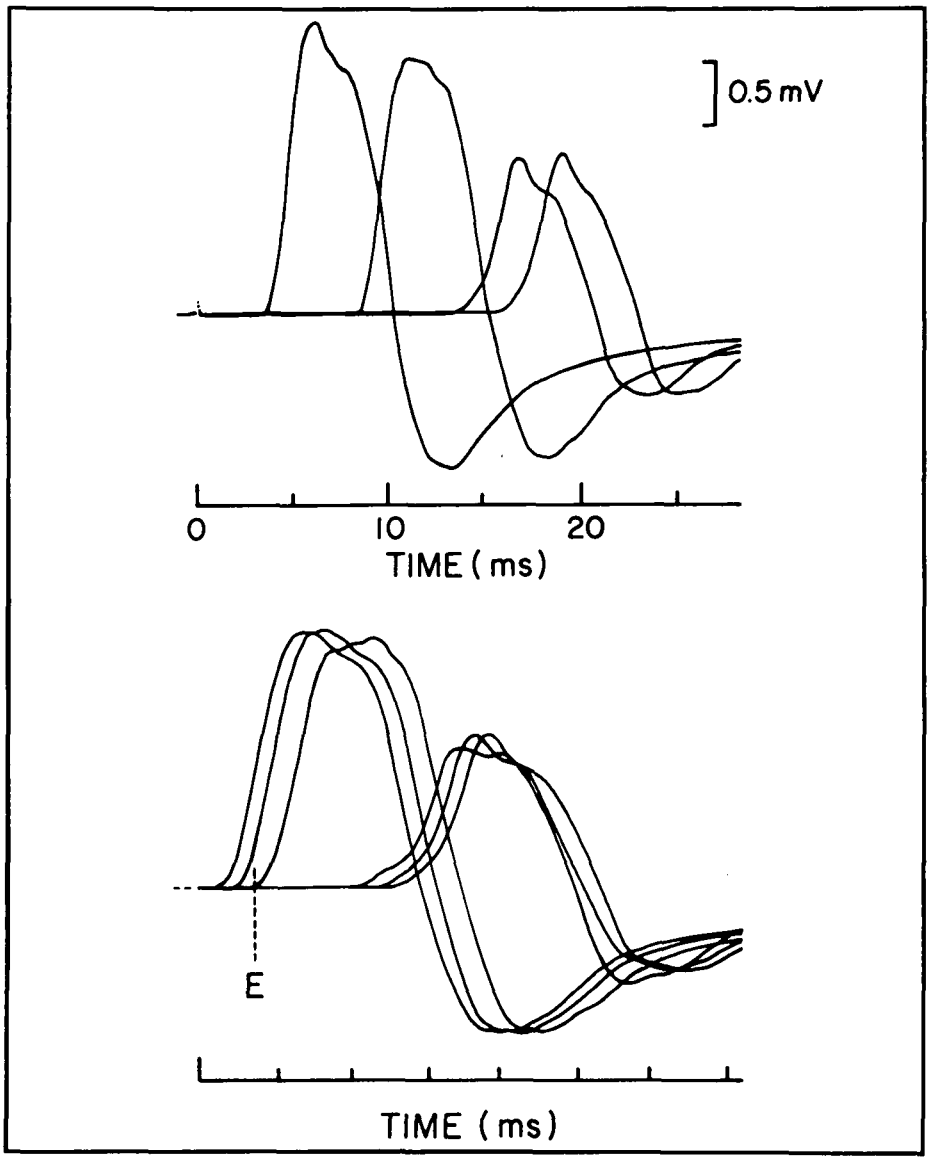

Figure 7 — Top: Illustration of the change in HT maximum 'M' response to stimulation of the ulnar nerve at the levels of the proximal and upper arm, proximal and distal to the elbow (120 mm apart) and wrist. The maximum MCV across the elbow segment was 24 m/s.
Bottom: Changes in the HT maximum 'M' response to stimulation at 20 mm intervals proximal and distal to the tip of the medial epicondyle (E). Here, reduction in conduction velocity (13.3 m/s) was primarily limited to the 20 mm segment just proximal to the tip of the medial epicondyle. Across the latter segment there was a 41% reduction in p-pV, a 3% increase in p-pD and 44% reduction in negative peak area: all pointing to conduction block in close to 45% of the hypothenar motor axon population across the abnormal segment.

Positive sharp waves were observed in muscles innervated by the ulnar nerve in eleven of fourteen ulnar neuropathies. The fifth digit sensory nerve action potential was absent in five and the amplitude lower than normal in one of the total of thirteen ulnar neuropathies where this was checked.

(3) *Median nerve:*

The conventional measurements of sensory and motor conduction were in the normal range in the thirty patients included in the trans-retinaculum measurements. The diagnosis was established by a typical history together with comparisons of the latencies of orthodromic sensory potentials evoked by stimulation of the fourth and first digits to recording electrodes at equivalent distances located over the respective ulnar, median and radial nerves at the level of the wrist. The orthodromic sensory latencies recorded over the median nerve in the patients

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were one millisecond or more longer than the latencies to the radial and ulnar electrodes. Therefore even though the absolute latencies of the median sensory potentials were within our normal ranges the excess in latency of median sensory fibers relative to radial or ulnar sensory fibers measured over equivalent distances was taken to indicate a significant delay in conduction in median sensory fibers. This conclusion has been substantiated in our laboratory by demonstration in a few such subjects of a localized delay at operation in the median nerve beneath the flexor retinaculum. It was further supported by the demonstration of slowing in the conduction velocities of sensory fibers beneath the flexor retinaculum.

Table 6 illustrates the conduction velocities measured proximal to, across and beyond the flexor retinaculum. In every patient the maximum sensory conduction velocity across the flexor retinaculum was ten meters per second less than the velocity proximal to the retinaculum. By contrast, in healthy subjects the velocity across the retinaculum was never more than ten meters per second less than the velocity proximal to the retinaculum. In twenty-three patients, the maximum velocity beyond the retinaculum increased relative to the velocity across the retinaculum but in only two was the pre-retinaculum velocity attained. In the other seven patients, the maximum velocity beyond the retinaculum was the same or lower than the velocity measured across the retinaculum.

TABLE 6: *Tabulation of the maximum orthodromic sensory conduction velocities measured between the proximal stimulating electrode (cathode) on the digit and the distal border of the flexor retinaculum (distal), between the distal and proximal borders of the retinaculum (trans-retinaculum) and between the latter and a point 60-80 mm proximal (proximal).*

Median nerve:

Maximum (orthodromic) sensory CV with reference to flexor retinaculum in 30 carpal tunnel patients

	MCV (M/s) ± 1 S.D.		Range
Proximal	63.6	9.8	50.3 - 70.0
Trans-Retinaculum	44.6	5.9	30.5 - 53.8
Distal	52.0	6.2	43.8 ± 63.8

From the investigation employing percutaneous stimulation at 20 mm intervals, it was learned that the 20 mm segment with the lowest maximum conduction velocity was most commonly located 20-40 mm distal to the main wrist flexor crease (five of six median nerves). In the only other acceptable record obtained by this technique the location was 40-60 mm beyond the crease.

DISCUSSION

Our intra-operative experience which stands as the background for the present investigation, established that the primary abnormalities in conduction in entrapment neuropathies were, except in severe lesions or in some metabolic disorders (Brown, et al, 1976) concentrated in short lengths of the nerve and at least in the median and ulnar nerves, there was good correspondence between the abnormal appearing regions and the major conduction abnormalities in the nerve. The consequence of the discrete nature of these lesions is that when conduction

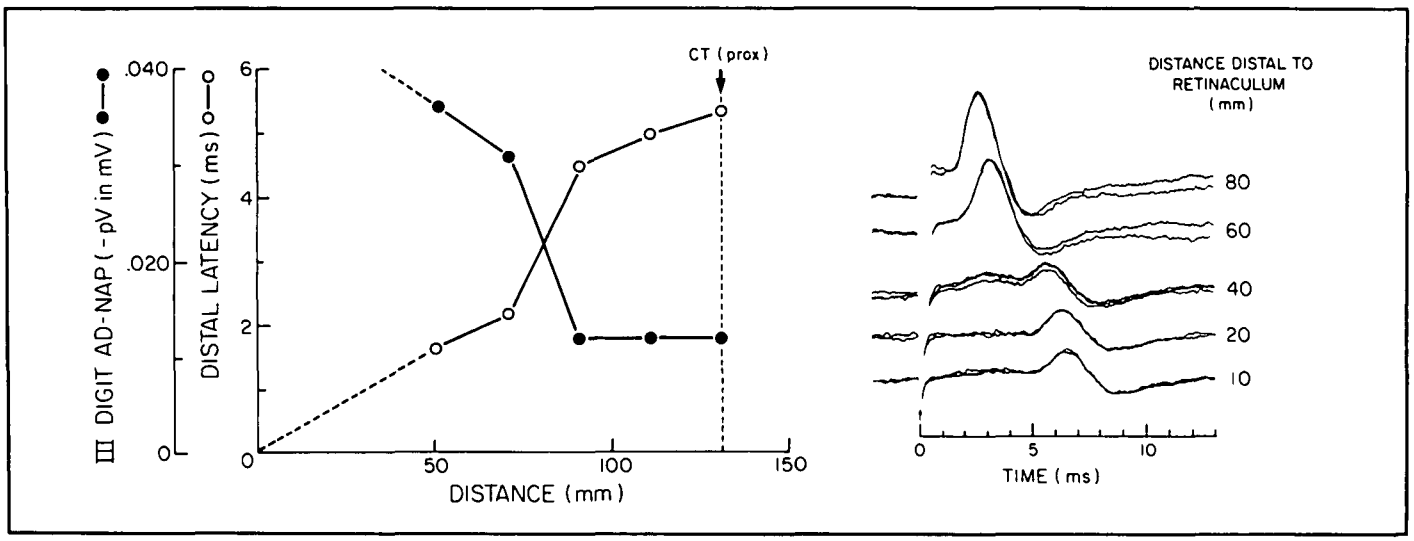

Figure 8 — Plot (left) of changes in the third digit antidromic sensory nerve action potential (III Digit AD-NAP) negative peak voltage (-pV) and distal latency in response to percutaneous stimulation at 20 mm intervals beginning at the main flexor crease at the wrist. Note the major increase in conduction time and reduction in -pV were observed 40-60 mm distal to the flexor crease. The original records are illustrated on the right.

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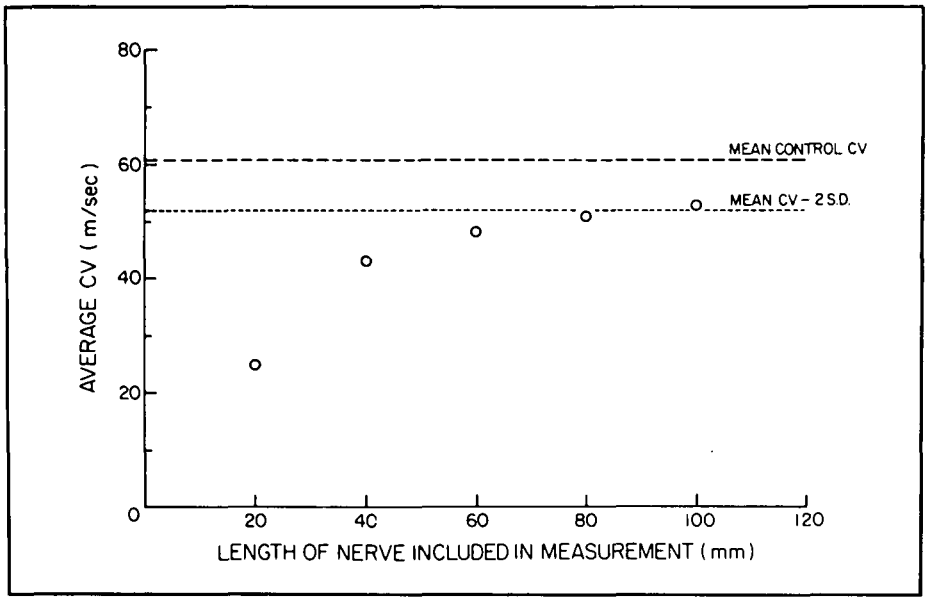

Figure 9 — Illustrative plot of the changes in the calculated MCV of a nerve produced by the addition of successive normal 20 mm lengths of nerve (normal MCV just over 60 m/s) to a 20 mm segment with abnormal conduction — in this example 25 m/s. In this example the addition of only 60 mm of normal nerve to the overall test segment brought the calculated MCV to within 2 S.D. of the normal mean.

velocities are measured over long segments (80-100 mm), conduction could look much less abnormal because of the inclusion of longer normal or near normal segments in the calculation. This problem is illustrated by Figure 9. The capacity to detect abnormally slow conduction is governed by the degree of the slowing across the most abnormal segment, the length of this segment, and the length of the remaining normal or relatively normal nerve included in the measurement. The problem is compounded in the ulnar nerve where the maximum motor conduction velocities measured across the elbow segment are often lower relative to the velocities across the upper arm and forearm even in healthy subjects, especially when the investigations are carried out with the arm in extension (Payan, 1969; Checkles, et al, 1971; Eisen, 1974).

Percutaneous stimulation at short intervals along the length of a nerve is attractive because it shortens the segment over which the conduction is measured to lengths which approximate better the distribution of the most abnormal regions in the nerve. The abnormal regions could bracket two or three adjacent test segments because the stimulation points on the surface, fixed by reference to a surface or bony landmark, may not bridge in a single short segment the main lesion in the nerve. This explanation could account for the examples when the apparent conduction abnormalities seemed more extensive. However, the lesions and physiological abnormalities could be more extensive.

Thresholds for excitation of nerve fibers are a function of their diameters (Gasser and Grundfest, 1939), their position within the nerve relative to the overlying cathode, and especially in neuropathies, the possible presence of abnormalities in the myelin sheath or axonal membrane properties which alter the relative excitabilities of the nerve fibers (Waxman, 1977; Bostock and Sears, 1978; Waxman and Foster, 1980). These factors help to determine just where the nerve fibers are excited in respect to the overlying cathode. Thus the stimulus could excite the nerve at more distant normal and lower threshold sites than at abnormal regions which, though nearer to the cathode, have much higher thresholds for the initiation of nerve impulses. Moreover, thresholds of the various nerve fibers could be so different that stimulus intensities supramaximal for the 'M' response or sensory nerve action potential, may activate the lowest threshold fibers well ahead of the cathode. This effect is just an exaggeration of the same phenomenon in normal nerves (Gasser, 1960) and various human neuropathies (Simpson, 1964; Hausmanowa-Petrusewicz and Kopec, 1967; Wiederholt, 1970).

These problems are compounded by the use of the relatively large diameter (7 mm) surface stimulating electrodes employed here, their short interelectrode distances (center to center, 22 mm) and the changes in depth of the nerves relative to the stimulating electrodes at various sites of stimulation. However, despite uncertainties about the exact level at which impulses are initiated in the nerves relative to cathode, the percutaneous techniques provide approximate indications of the location of the predominant conduction abnormalities in the entrapments. More precise localization of the stimulus current may be obtained by near nerve electrodes. This technique however, is much less convenient and when comparisons were made with surface stimulation, the locations of the primary conduction abnormalities were similar. The present techniques are a compromise between attempts to detect and locate with more precision, local conduction abnormalities and the inherent technical limitations in surface stimulation at short (20 mm) intervals.

In the nerves examined by the percutaneous stimulation, the major conduction abnormalities were distributed over distances which were less than one-half and frequently less than one-fifth of the total distance over which conduction is routinely measured in the clinic. In the peroneal nerve, the major abnormalities were at or adjacent to the fibular head. However, in two peroneal nerves the abnormalities were much more proximal to the fibular head. One of these patients had polyarteritis nodosa. It is tempting here to suggest that the much more proximal location of the conduction abnormalities in this case was a reflection of probable infarction of the nerve and not the usual mechanical entrapment. In the other more proximal peroneal neuropathies evidence of vasculitis was absent. The locations of the major conduction abnormalities in the diabetic patient with

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bilateral peroneal palsies were near the fibular head, perhaps implying that the primary causative factor was compression not ischemia.

In the ulnar nerve the major conduction abnormalities were concentrated just proximal or distal to the medial epicondyle; those distal to the epicondyle probably originating at the cubital tunnel. Intriguing, though hard to explain, was the observation that both post-operative ulnar neuropathies were located in the retro-epicondylar segment or proximal to the epicondyle whereas the two post-traumatic neuropathies were located distal to the epicondyle, possibly at the level of the cubital tunnel. In the remainder, no obvious correlation was evident between the clinical histories, abnormalities on examination or pattern of the EMG abnormalities and the location of the most abnormal segments of the nerve. In the diabetic patient, the major conduction abnormalities were proximal to the medial epicondyle but in other diabetics the location has varied.

In the median nerve, percutaneous stimulation proved less satisfactory than in the ulnar and peroneal nerves because stimulus artifact presented more of a problem. Also in some of the more abnormal median neuropathies the low amplitude of the antidromic sensory nerve action potentials made it difficult to measure the latencies properly. The short recovery amplifier described by Kimura (1978) was not available to us at the time of this investigation. There was also more uncertainty about just where the nerve was excited in relation to the surface electrode because the median nerve is much nearer the surface proximal and distal to the flexor retinaculum than beneath the retinaculum. Indeed, inspection of Kimura's records shows that the stimulus at some of the points on the surface was sub-maximal (see his Figures 4,5,6,7 and 8, 1976). It was possible, with care, to locate the predominant conduction delays in five median nerves by these percutaneous stimulation. The worst conduction delays were towards the distal end of the flexor retinaculum in agreement with Kimura's own evidence and with observations in some of our direct intra-operative examinations.

In intra-operative investigations where the median nerve could be directly stimulated, the worst delays were in the 20 mm segment just beyond the proximal border of the flexor retinaculum. This level corresponded to the segment with the most abnormal appearance. The apparent discrepancies between the locations of the most abnormal segment determined by direct nerve and percutaneous stimulation could reflect uncertainties about the relation of the proximal border of the flexor retinaculum to the major wrist creases.

Measurement of the maximum conduction velocity across the flexor retinaculum is a simple technique and though it does not provide as discrete localization as percutaneous stimulation at shorter intervals, the technique can demonstrate conduction delays beneath the retinaculum in early median neuropathies, as was true in the thirty examples of this investigated here. There seems therefore, little need for the percutaneous stimulation technique in the majority of median neuropathies.

Overall, there was excellent correspondance between the locations of the most abnormal conduction delays and conduction block. When conduction block is present, the conduction velocities may be less than normal across the same or more proximal segments either because the velocities in the remaining unblocked nerve fibers are truly reduced or because conduction in the fastest fibers has been blocked, the low velocities in this case simply reflecting the velocities in normally slower conducting, but in this case, unblocked nerve fibers. The fastest and largest nerve fibers are blocked earliest by mechanical compression (Gelfan and Tarlov, 1955). Thus, only when the velocities of remaining unblocked fibers can be demonstrated to be higher over more proximal segments is it justifiable to conclude that a velocity in a more distal segment is truly abnormally low. This is an important point to remember because the range of conduction velocities of human motor fibers is about fifty percent (Thomas, et al, 1959; Gilliatt, et al, 1976).

In the majority of the neuropathies characterized by substantial conduction blocks and delays there was evidence of wallerian degeneration in motor and sensory fibers. This conclusion was based on the presence of fibrillation and positive sharp wave activity in muscles innervated by the respective nerves and absent or abnormally low amplitude sensory nerve action potentials distal to the level of the entrapment. Therefore in the median and ulnar nerves tested in this investigation structural abnormalities typical of chronic nerve compression (Gilliatt, 1975; Neary, et al, 1975; Neary and Eames, 1975; Gilliatt, 1980) and wallerian degeneration were likely present.

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