Digital nerve action potentials in healthy subjects, and in carpal tunnel and diabetic patients

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SUMMARY   A technique is described for stimulating and recording from nerves in the finger using surface electrodes. A decrease in amplitude and velocity was found with increasing age. In control subjects the digital potential was approximately one and a half times larger than the potential recorded at the wrist. In patients with carpal tunnel syndrome there was some reduction in amplitude and velocity of the digital potential, but the changes were more marked at the wrist. In diabetic patients more uniform changes were found in the two segments. The technique was particularly useful in enabling conduction velocity to be calculated in the digital nerves when no potential could be recorded at the wrist.

In 1959 Sears described a method of recording volleys of afferent nerve action potentials from human digital nerves evoked by a physiological stimulus of a light tap on the finger nail. McLeod (1966) later used a similar technique to study digital nerve conduction in patients with the carpal tunnel syndrome. More recently Buchthal and Rosenfalk (1971) have recorded from digital nerves through needles inserted near the nerve trunks in the palm after stimulation distally, in patients with the carpal tunnel syndrome.

A problem inherent in all methods for measuring nerve conduction velocity over short distances is the control of stimulus artefact. McLeod (1966) avoided this by using mechanical rather than electrical stimulation, and Buchthal and Rosenfalk (1971) reduced the artefact by modification of the amplifier and by using needle electrodes. In the present study the problem was overcome by improving stimulus isolation and by paying careful attention to preparation of the skin and application of electrodes. By these means it was possible to record potentials through surface electrodes only a few centimetres from the site of electrical stimulation. Full details of the method and results in different conditions have been described by Casey (1971).

McLeod (1966) pointed out that the measurement of conduction velocity over short conduc-tion distances in the finger is of particular value in patients with neuropathies when slowing and dispersion of the volley is of such a degree that an action potential cannot be recorded over the longer conduction distance between the finger and the wrist. A further advantage is that a distal part of the peripheral nervous system can be studied, which may be useful in investigating dying back neuropathies. For example, the technique has proved valuable in demonstrating a distal lesion in alcoholic neuropathy (Casey and Le Quesne, 1972).

METHODS

HEALTHY SUBJECTS

Ninety-four healthy subjects whose ages ranged from 20 to 80 years were examined. None had symptoms or signs of neuromuscular disease. Seventy-five subjects were examined when the digital skin temperature was 35–36° C. This group consisted of 39 females and 36 males. When examining seriously ill patients, it was not always possible to attain or maintain this temperature. A number of results in such patients were obtained when the skin temperature was only 30–32° C. In order to assess these findings a further 19 healthy subjects were examined when the skin temperature of the finger was between 30 and 32° C.

PATIENTS

CARPAL TUNNEL SYNDROME   There were 15 female patients and one male patient in this group, and their ages ranged from 35 to 70 years, the mean age being 55·9 years. All had classical symptoms and 10 had hypalgesia in the fingers of the involved hand supplied by the median nerve. Patients in whom the median nerve sensory action potential recorded at the wrist after digital nerve stimulation was abnormal or at the lower limit of normal were chosen for study. For details of individual patients see Table 2.

DIABETIC PATIENTS   Digital nerve conduction studies were performed on 18 diabetic patients with clinical evidence of peripheral neuropathy. All the patients had reflex changes and distal sensory abnormalities in the lower limbs, consisting of pain and paraesthesiae with sensory loss. In addition, 10 had sensory changes in the upper limbs. For details of individual patients see Tables 3 and 4.

Eleven patients were examined when the skin temperature at the tip of the finger was 35° C throughout the test (group I). As it was not possible to maintain this temperature in all patients, another group of seven patients were examined when the temperature was 30° C (group II).

The age of those in group I ranged from 30 to 75 years (mean 52·4 years), and those in group II ranged

from 37 to 77 years (mean 55·0 years). The mean duration of the diabetes in group I was 13·0 years and in group II was 17·1 years.

ELECTROPHYSIOLOGICAL METHODS

To overcome the problem of stimulus artefact a specially designed stimulator with a high degree of isolation from the recording apparatus was developed. This unit was constructed as a result of preliminary experiments carried out by Mr. P. Fitch, Mr. H. B. Morton, and Dr. R. G. Willison at the Institute of Neurology, London. The isolation unit (Fig. 1) is battery powered and is contained in a metal box which is not grounded and not connected to any part of the circuit. A Devices Mk IV isolated stimulator is used to drive the unit with a pulse of defined length (usually 100 μsec) and variable amplitude. This is triggered by a pulse from the electromyograph. The unit provides a further two stages of isolation by means of transformers. The maximum output is 300 V, a 20 V pulse from the Devices stimulator being required to produce this. The diodes are included in order to reduce the stimulus artefact by making the output pulse as nearly unidirectional as possible. The output transformer (T_2) is a 100 μsec pulse transformer with a ferrite core.

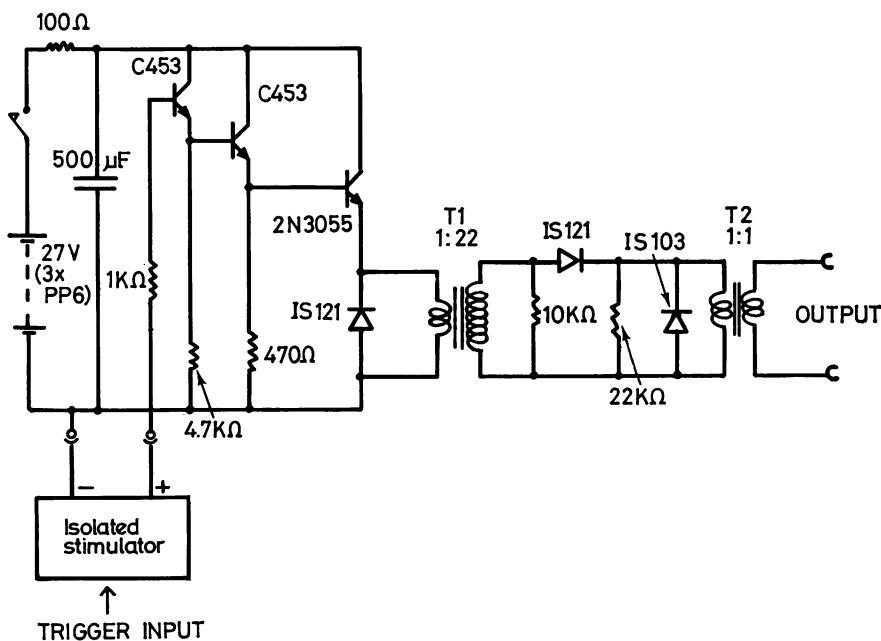Figure 2 shows sensory action potentials from a

FIG. 1.   *Circuit diagram of stimulus isolation unit. T_1: Radio-Spares Hygrade output transformer. T_2: Gardner's Transformers Ltd., GR 34260. Isolated stimulator: Devices Ltd., Mk IV.*
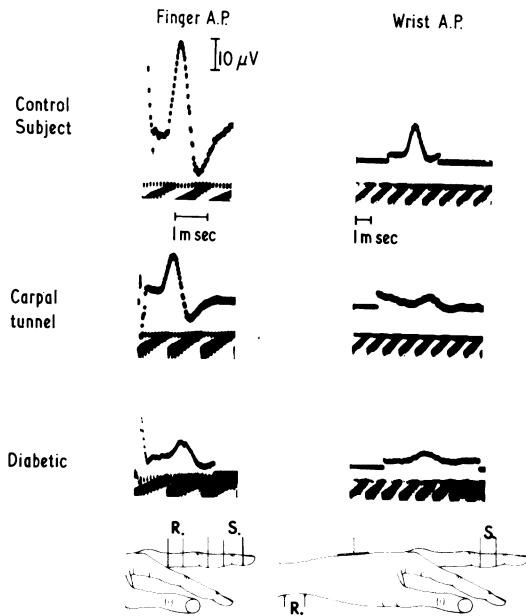
FIG. 2. *Digital nerve sensory action potential from a control subject, patient with the carpal tunnel syndrome, and a patient with diabetic peripheral neuropathy.*

Cambridge electrode jelly was applied beneath the wire by using a syringe with a blunt tipped no. 2 needle. Accurate localization of the jelly under the electrodes was most important in minimizing stimulus artefact.

The recording electrodes were connected to a Medelec MS3R electromyograph and potentials were displayed on one beam of the oscilloscope. Another beam was used to provide a time scale and a third beam provided a display of the response averager (Medelec AVM 3B/1 with a 100 ordinate capacity). At least 30 traces were fed into the averager. Measurements were made from enlarged photographic records.

In all subjects a sensory action potential was recorded from the median nerve at the wrist on stimulation of the middle finger as described by Dawson (1956). The proximal stimulating and distal recording electrodes which were already *in situ* for the digital nerve sensory action potential were used for stimulation.

RESULTS

HEALTHY SUBJECTS

AMPLITUDE   The values of the peak-to-peak

control subject and two patients. The positions of the stimulating (S) and recording (R) electrodes are shown at the lower part of the Figure.

With practice the examination could be carried out in 15 minutes. Careful preparation of the skin was important in reducing stimulus artefact. The subject's hand was immersed in hot water until the skin temperature over the distal phalanx of the finger to be examined was at or above 35° C and the temperature was maintained at this level by radiant heat from a DC lamp. To reduce skin impedance the lateral aspect of the finger was scrubbed with a brush and soap for 30 to 60 seconds. The hand was dried thoroughly and the finger rubbed with gauze soaked in ether and allowed to dry. A hydrophobic plastic sheet covered the finger to avoid contact with the other fingers. Five electrodes of tinned copper fuse wire were applied. The two proximal electrodes were used for recording, the middle was grounded, and the distal electrodes were connected to the stimulator. The distal recording and the proximal stimulating electrodes were placed at least 3·5 cm apart, and the distance between the recording electrodes was constant at 1·5 cm.
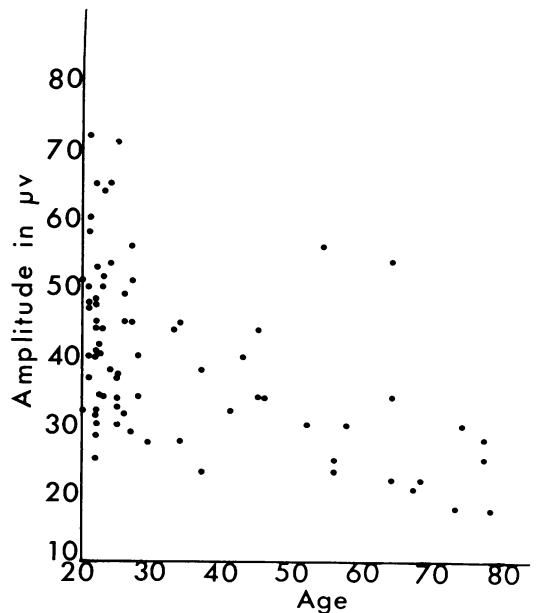

FIG. 3. *Amplitude of digital nerve sensory action potential expressed as a function of age in control subjects.*

TABLE 1

CONDUCTION VELOCITY AND AMPLITUDE OF DIGITAL AND WRIST NERVE ACTION POTENTIALS AT DIFFERENT TEMPERATURES IN CONTROL SUBJECTS

Subjects (no.)	Digital skin temperature (°C)	Mean age (yr)	Amplitude (μV)			Onset velocity (m/sec)			Peak velocity (m/sec)		
			Range	Mean	SD	Range	Mean	SD	Range	Mean	SD
Digital potential											
75	35–36	32·4	18–72	39·8	12·6	41–73	59·2	7·0	34–54	42·2	6·8
19	30–32	34·8	26–77	42·4	15·4	39–63	49·1	6·5	26–40	33·5	3·4
Wrist potential											
75	35–36	32·4	9–49	25·9	8·6				41–60	48·8	4·5
19	30–32	34·8	11–44	25·3	9·3				39–54	47·7	4·5

amplitude of the digital nerve action potentials are shown in relation to age in 75 subjects in Fig. 3. It can be seen that the values varied between 18 and 72 μV. The mean for the whole group was 39·8 μV (SD 12·6) (Table 1). It may also be seen that there was a reduction of amplitude with age; the highest value recorded was 72 μV in a 21 year old female and the lowest value, 18 μV, was obtained in a 78 year old male. Using product moment correlation for age, $r = -0.54$ and $P < 0.01$.

When males and females in the 20 to 30 years age group were considered separately, an interesting difference was seen. There were 21 males and 29 females in this age group and the 13 highest values were in females. The mean amplitude of the sensory action potential in females was 48 μV (SD 12·3) and in males was 37 μV (SD 7·4). The difference is significant using Student's t test (with pooled variances $t = 2.7148$; $N = 48$; $P < 0.01$).

In order to assess the reproducibility of the examination, experiments were repeated on two or more occasions in 11 subjects. In nine the values were within 5 μV of each other, but in two subjects differences of 10 μV and 13 μV (17% and 18%) were found.

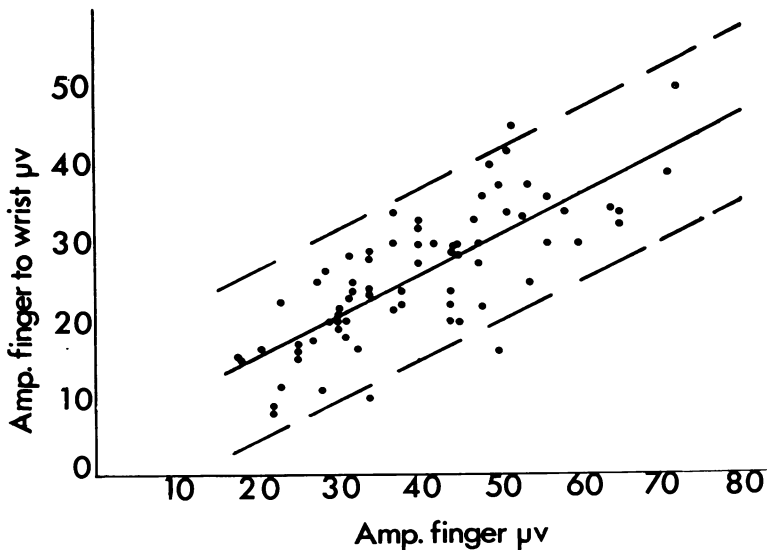Figure 4 shows that there is a linear relation-

FIG. 4. *Amplitude of sensory action potential recorded from the finger plotted against amplitude of the potential recorded from the median nerve at the wrist after stimulation at the base of the finger. The solid line is the calculated regression line and the broken lines are two standard deviations from this line.*

ship between the amplitudes of the digital and wrist potentials. The digital potential was approximately one and a half times greater than the wrist potential over the whole age range. The mean values were 39·8 μV (SD 12·6) for the digital and 25·9 μV (SD 8·6) for the wrist potential.

CONDUCTION VELOCITY   Latency was measured to the onset and to the peak of the negative deflection of the sensory action potential and conduction velocities calculated (Table 1). The velocity to onset ('onset velocity') ranged from 41 to 73 m/sec, with a mean of 59·2 m/sec (SD 7·0) and the velocity to peak ('peak velocity') from 34 to 54 m/sec, with a mean of 42·2 m/sec (SD 6·8). There was a slight decrease in velocity with increase in age, which was more marked when calculated from latency to peak than from latency to onset. The correlation coefficient (r) for onset velocity was $-0·44$, $P < 0·01$ and for peak velocity $r = -0·58$, $P < 0·01$.

For the wrist potential, conduction velocity was calculated only from peak latency, since the onset of the lower amplitude potential was sometimes difficult to define. The mean velocity was 48·8 m/sec (range: 41–60 m/sec; SD 4·5).

TEMPERATURE   Figure 5 shows the action potentials recorded from one subject at different digital skin temperatures. It can be seen that, while the amplitude changed very little, the duration of the digital nerve action potentials became longer, and the latency to onset increased from 0·65 msec at 36° C, to 0·80 msec at 27·5° C, and 0·95 msec at 23° C. This corresponds to a drop of conduction velocity of 1·2 m/sec/° C over the temperature range from 36° C to 27·5° C and of 1·5 m/sec from 27·5° C to 23° C.

The results for 19 control subjects who were examined when the temperature at the tip of the finger was only 30 to 32° C are shown in Table 1 and again the effect of temperature on conduction velocity can be seen. The difference in amplitude of the potentials was not significant (Student's t test; $t = 0·7714$; $N = 92$; $P > 0·20$).

The mean peak conduction velocity for the wrist potential was 47·7 m/sec (SD 4·5), a value which is very close to 48·8 m/sec obtained in the 75 patients examined when the skin temperature of the finger was 35° C. The temperature of this segment of nerve is more likely to be similar in the two groups and less related to the superficial skin temperature at the tip of the finger.

ISCHAEMIA   The effect of ischaemia on the digital nerve action potential was studied in four subjects by inflating a sphygmomanometer cuff to 200 mm Hg around the upper arm for 30 minutes. Since temperature falls particularly rapidly in an ischaemic limb, it was important to ensure that the temperature remained constant during these experiments. This was achieved by shining the DC lamp on the finger continuously throughout the experiment.

Action potentials during ischaemia in one subject (P.M.F.) are shown in Fig. 6, the results in the other subjects being similar. It can be seen that a diminution in amplitude, slowing of conduction velocity, and eventual extinction of the sensory action potential occurred. Four minutes after releasing the cuff the amplitude had returned to 33% of the initial value and by 25 minutes it had almost returned to its preischaemic value.

CARPAL TUNNEL SYNDROME
The results for the 16 patients are shown in Table 2. A control group of comparable age was provided by the 22 healthy subjects aged 30 to 75 years.
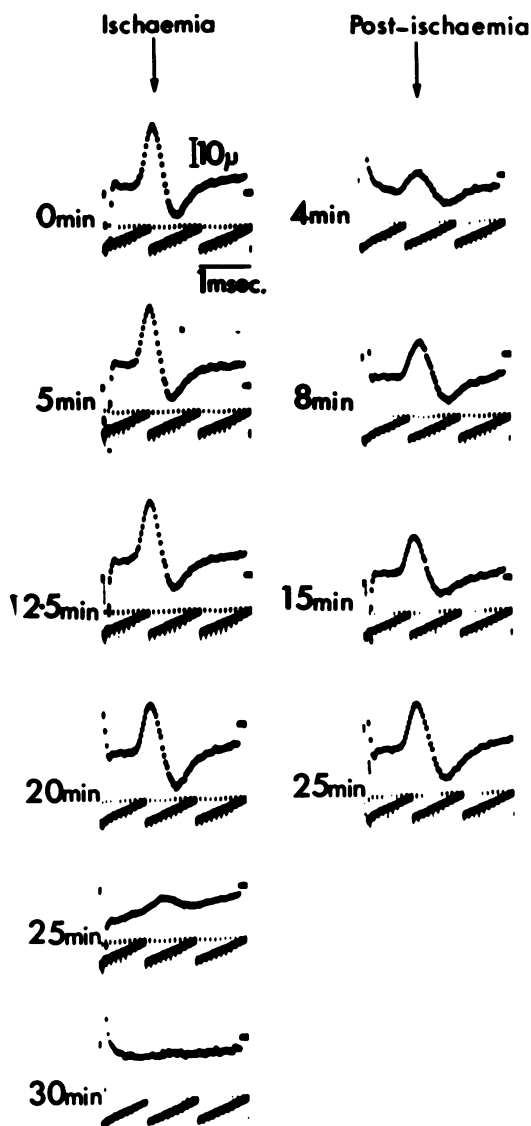

FIG. 5.   *Effect of temperature on digital nerve action potential showing increase in latency with decrease in temperature.*

I 10μv

36°C

27·5°C

23°C

1 msec

FIG. 6. *Effect of 30 minute ischaemia on the digital nerve sensory action potential. The post-ischaemic changes may be seen in the right-hand column.*

AMPLITUDE   In the 14 patients in whom a digital potential could be recorded, amplitude ranged from 1 to 34 μV (mean 14·4 μV; SD 9·8). In five patients the amplitude of the digital potential was within control limits and in nine it was below the control range. No potential could be recorded in two subjects, in both of whom the wrist

potential was also absent. The amplitude of the digital potential was below the control range in four of the six patients in whom there was no clinical abnormality of sensation in the fingers. Illustrative potentials recorded from one subject (L.D.) are shown in Fig. 2. In this patient the amplitude of the digital nerve potential was 20 μV; the wrist potential amplitude was 3 μV.

Figure 7 shows the amplitude of the digital potential in relation to the amplitude of the wrist potential. It may be seen that the linear relationship (compare Fig. 4) between the two potentials is lost, the wrist potential being more abnormal than the digital potential.

VELOCITY   As may be seen from Table 2, the mean onset velocity for the digital potential was 43·8 m/sec and was below the control range in eight patients. The mean peak velocity was 31·4 m/sec, abnormal in seven. Both mean values are below the control range. The peak conduction velocity for the wrist potential was more affected, the mean being 28·2 m/sec compared with a control value of 46·4 m/sec. The values for digital and wrist conduction velocities are shown graphically in Fig. 8.

DIABETIC NEUROPATHY
Tables 3 and 4 and Fig. 9 show the findings in two groups of diabetic patients studied at different digital skin temperatures.

AMPLITUDE   It has been shown that in control subjects of comparable ages there is no significant difference between the amplitude of the digital potential recorded at 30° C and that recorded at 35° C (see Table 1). Both groups will therefore be considered together.

The amplitude of the digital potential ranged from 5–26 μV and was below the control range in 10 of the 18 subjects. The wrist potential ranged in amplitude from 0–17 μV and was abnormal in 11 patients. Cutaneous sensation was impaired in the fingers in 10 patients. In five of these the digital potential was abnormal. The digital potential was also reduced in five of the eight patients in whom finger sensation was normal. It is well recognized that correlation between clinical and electrical findings in diabetic peripheral neuropathy is poor (Gilliatt and

TABLE 2

SENSORY NERVE CONDUCTION IN CARPAL TUNNEL PATIENTS

Name	Sex	Age (yr)	Sensation in fingers	Digital potential			Wrist potential	
				Amplitude (μV)	Onset velocity (m/sec)	Peak velocity (m/sec)	Amplitude (μV)	Peak velocity (m/sec)
B.H.	F	55	Normal	34	44	32	9	25
D.G.	F	61	Normal	16	44	35	8	38
M.B.	F	45	Normal	11	45	36	8	26
P.P.	F	63	Normal	25	44	35	6	31
E.L.	F	52	Reduced	25	46	37	5	31
M.E.	F	35	Reduced	16	57	38	4	20
L.D.	F	49	Reduced	20	44	35	3	27
M.L.	F	48	Reduced	1	—	26	—	—
C.C.	F	68	Reduced	11	39	25	—	—
P.R.	F	50	Reduced	3	—	27	—	—
D.C.	F	70	Normal	7	35	23	—	—
E.C.	F	70	Reduced	4	35	23	—	—
R.M.	F	50	Normal	7	40	30	—	—
C.C.	M	59	Reduced	22	53	38	—	—
M.R.	F	60	Reduced	—	—	—	—	—
M.W.	F	60	Reduced	—	—	—	—	—
Mean		55·9		14·4 (9·8)	43·8 (6·5)	31·4 (5·6)	6·1 (2·3)	28·2 (5·7)
Controls								
Mean (SD)		51·0		33·6 (10·6)	54·8 (7·3)	39·9 (3·3)	19·8 (6·7)	46·4 (3·6)
Range		30–75		18–56	45–68	35–46	9–36	38–53

TABLE 3

SENSORY NERVE CONDUCTION IN DIABETIC PATIENTS: GROUP 1 : TEMPERATURE 35° C AT FINGER TIP AT END OF TEST

Name	Sex	Age (yr)	Duration of diabetes (yr)	Sensation in fingers	Digital potential			Wrist potential	
					Amplitude (μV)	Onset velocity (m/sec)	Peak velocity (m/sec)	Amplitude (μV)	Peak velocity (m/sec)
M.U.	F	58	17	Reduced	19	41	29	9	39
Y.S.	F	42	10	Reduced	26	44	32	16	48
K.B.	M	31	15	Reduced	22	40	31	10	43
F.R.	F	70	12	Reduced	25	39	31	—	—
M.S.	M	48	30	Normal	20	44	27	17	43
P.S.	M	30	15	Normal	24	45	32	11	38
S.O.	M	48	7	Normal	13	39	27	6	44
P.J.	F	75	6	Normal	20	50	32	15	45
M.H.	F	62	10	Reduced	19	37	25	10	40
T.W.	M	53	8	Reduced	5	45	35	2	34
J.B.	M	61	17	Reduced	7	38	27	2	27
Mean (SD)		52·4	13·0		18·1 (7·4)	42·2 (4·1)	29·8 (3·0)	9·8 (5·3)	40·1 (6·1)
Controls at 35°									
Mean (SD)		51·0			33·6 (10·6)	54·8 (7·3)	39·9 (3·3)	19·8 (6·7)	46·4 (3·6)
Range		30–75			18–56	45–68	35–46	9–36	38–53

Willison, 1962; Lamontagne and Buchthal, 1970).

Figure 9 shows the relation of the amplitude of the digital potential to that of the wrist potential in the 18 diabetic patients. The relationship between the two potentials remained linear, and the digital potential was always of higher amplitude than the wrist potential.

VELOCITY It may be seen from Tables 3 and 4 that the mean values for the onset and peak velocities of the digital potential were lower than those obtained in the control groups. In group I the onset velocity was reduced in eight of the 11 patients. In three of seven group II patients onset velocity could not be measured accurately due to stimulus artefact. In one other, onset velocity

TABLE 4

SENSORY NERVE CONDUCTION IN DIABETIC PATIENTS: GROUP II: TEMPERATURE 30° C AT FINGER TIP AT END OF TEST

Name	Sex	Age (yr)	Duration of diabetes (yr)	Sensation in fingers	Digital potential			Wrist potential	
					Amplitude (μV)	Onset velocity (m/sec)	Peak velocity (m/sec)	Amplitude (μV)	Peak velocity (m/sec)
S.K.	M	65	20	Reduced	6	—	22	—	—
D.B.	M	48	10	Reduced	6	—	32	2	35
M.M.	F	77	8	Normal	10	—	25	5	34
M.R.	M	37	20	Normal	13	39	26	5	35
L.L.	M	55	5	Normal	7	39	26	3	35
J.H.	M	42	20	Reduced	9	39	27	5	41
S.W.	M	61	37	Normal	8	30	22	1	28
Mean (SD)		55·0	17·1		8·4 (2·5)	33·0 (4·5)	25·7 (3·4)	3·5 (1·8)	34·6 (4·1)
Controls at 30–32°									
Mean (SD)		34·8*			42·4 (15·4)	49·1 (6·5)	33·5 (3·4)	25·3 (9·3)	47·7 (4·5)
Range					26–77	39–63	26–40	11–44	39–54

* Note younger mean age for controls.

was reduced. The peak velocity was reduced in 10 patients in group I and in three patients in group II. Digital peak velocity was abnormal in two patients (N.S. and P.S.) with unimpaired sensation in the fingers and in whom the amplitude of the finger and the amplitude and velocity of the wrist potentials were within normal limits.

It may be seen from Table 1 that there was no difference between the peak wrist velocity in the control subjects examined at different digital

temperatures and so the results for the two groups may be considered together. In the diabetic patients the peak velocity of the wrist potential ranged from 27 to 48 m/sec and was abnormal in seven. The values for digital and wrist velocity are shown graphically in Fig. 8. (The digital velocities for group II are not shown.)

In two patients (F.R. and S.K.) it was not possible to obtain a value for conduction velocity in the segment of nerve between the base of the finger and the wrist due to absence of a potential
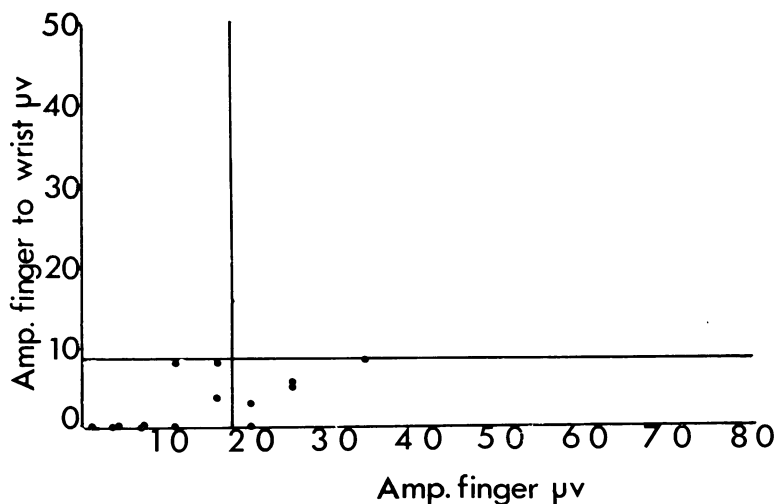

FIG. 7.   *Amplitude of digital potential plotted against amplitude of wrist potential in 14 carpal tunnel patients. Vertical line indicates lower limit of digital and horizontal line limit of wrist potential in control subjects.*
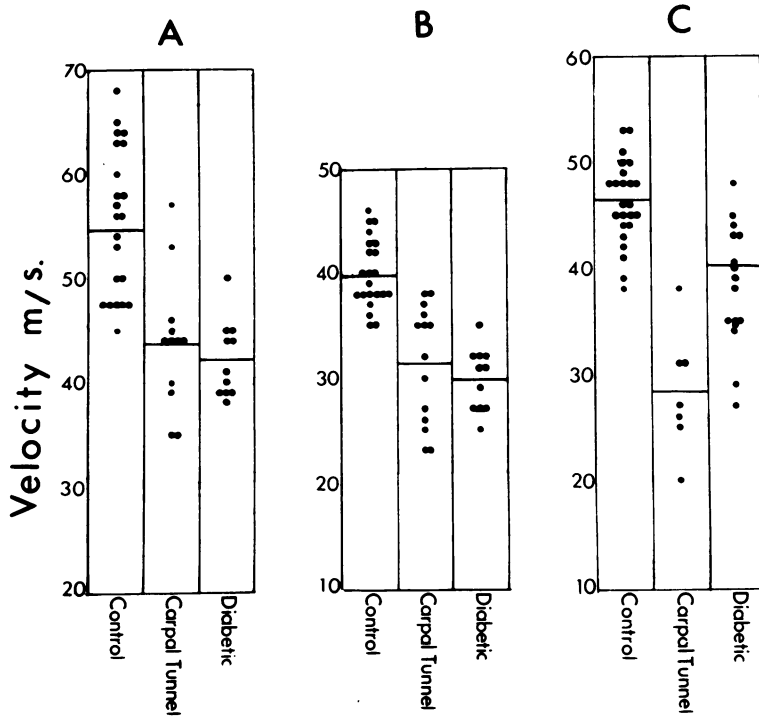
FIG. 8. *Comparison of conduction velocity in the three groups. A = Onset digital velocity. B = Peak digital velocity. C = Peak wrist velocity. The horizontal bar indicates the mean value for each group.*
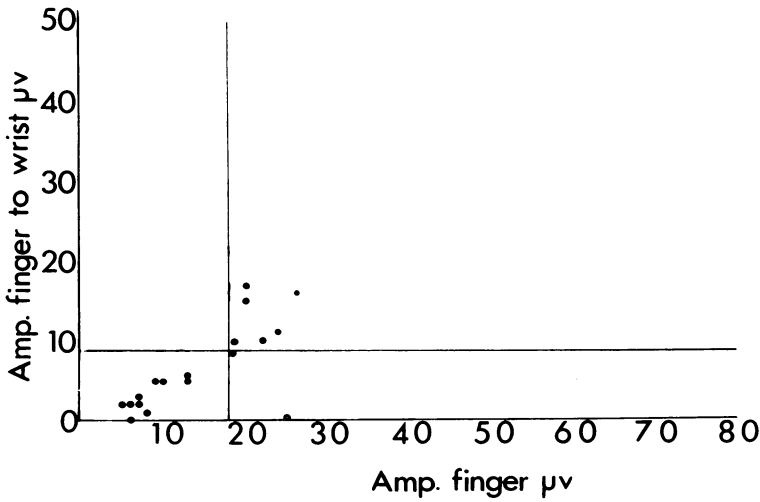

FIG. 9. *Amplitude of digital potential plotted against amplitude of wrist potential in 18 diabetic patients. Vertical and horizontal lines indicate lower limit of digital and wrist potential respectively in control subjects.*

at the wrist. Conduction velocity in the digital nerve was substantially reduced in both patients.

An example of the potentials obtained in a diabetic patient (J.B.) is shown in Fig. 2.

DISCUSSION

It has been shown that it is possible to record peripheral nerve action potentials at a short distance from an electrical stimulus to a nerve. A stimulator with a high degree of isolation is necessary to achieve this, as is attention to details of technique, such as accurate and localized application of electrode jelly beneath the electrodes. The importance of temperature control cannot be too strongly stressed. The finger is situated in one of the most exposed parts of the body and may be almost poikilothermic.

It is interesting that the amplitude of the sensory action potential is greater in females than males in the 20 to 30 years age group but the reasons are unknown. Brown (1968) found a difference in nerve action potential amplitude between males and females and also showed that the size of the sensory action potential obtained from the median nerve at the elbow on stimulation at the wrist could be correlated with arm circumference. Perhaps the expected smaller circumference of the fingers in females may be a factor in the present series. No difference in skin resistance was noted between males and females, and the conduction distances were similar.

Because of the short length of the finger it was not possible to apply more than one pair of recording electrodes and therefore not possible to estimate conduction velocity from the latency difference of two potentials. It has, however, previously been shown (Gilliatt, Melville, Velate, and Willison, 1965) that velocity calculated from the latency to the onset of the negative deflection of a potential recorded from a single site gives a good approximation to conduction velocity in the fastest fibres. Velocity calculated in this way from latency to the peak of the potential is of more doubtful significance. However, peak conduction velocity was used in the present series so that comparisons could be made between different groups of patients. Peak latency is often easier to measure accurately than onset latency

and it is a parameter which has been used by other workers (Downie and Newell, 1961; Fincham and Van Allen, 1964). The conduction velocity measurements calculated from onset latencies were similar to those found by McLeod (1966). Peak velocities cannot be compared, since he estimated velocity from the latency differences of potentials recorded at different sites.

A decrease in conduction velocity of both motor and sensory nerves with increasing age has been found by many workers (Wagman and Lesse, 1952; Norris, Shock, and Wagman, 1953) with sensory affected more than motor fibres (LaFratta and Canestrari, 1966). Lascelles and Thomas (1966) found segmental demyelination of some fibres in sural nerves from healthy subjects over the age of 65 years. O'Sullivan and Swallow (1968) found a tendency for mean fibre density to decrease with age in the sural and radial nerves, mainly due to loss of large diameter fibres. Similar changes in the digital nerves might account for the effect of age in the present study.

In 1966, McLeod measured conduction velocity in digital nerve fibres distal to the lesion in patients with the carpal tunnel syndrome. He found that, although there was no reduction in amplitude compared with control subjects, both onset and peak conduction velocities were reduced, the latter to a greater extent than the former. Conduction was slowed to a greater degree in patients in whom no potential could be recorded at the wrist after electrical stimulation of fingers than in those in whom a potential could be recorded. Buchthal and Rosenfalk (1971) have also measured conduction in sensory fibres distal to the carpal tunnel by recording through a needle electrode in the palm. They found that in most patients conduction was slower across the site of the lesion than distally and suggested that this might be an important diagnostic point, particularly in patients with only minor abnormalities. In the more severely affected patients they also found considerable slowing distally. Similar results have been found in the present study using surface electrodes for both stimulating and recording. The degree of slowing of conduction in relation to the amplitude of the digital potential in all those studies indicates that the changes cannot be explained

merely by failure of conduction in large diameter fibres.

There have been few histological studies of nerves in the carpal tunnel syndrome. Thomas and Fullerton (1963) examined digital nerve fibres histologically in such a patient and found that there was a reduction in fibre diameter, both at the site of the lesion under the flexor retinaculum and distally. Fullerton and Gilliatt (1967) have studied nerves from guinea-pigs that develop a lesion similar to the carpal tunnel syndrome. Segmental demyelination was present at the wrist in mild cases and small diameter myelinated fibres were present distally. Similar histological changes would explain the physiological findings in patients with the carpal tunnel syndrome. However, there is no indication from either of these histological studies as to whether the small diameter digital fibres were regenerating fibres which had not fully matured or whether they were the result of a pathological change in surviving fibres.

Several groups of workers have found that marked slowing of motor conduction may occur in diabetic peripheral neuropathy (Mulder, Lambert, Bastron, and Sprague, 1961; Skillman, Johnson, Hamwi, and Driskill, 1961; Gilliatt and Willison, 1962). As one might expect when conduction is slowed, the predominant lesion in the peripheral nerves of patients with diabetic neuropathy has been shown to be segmental demyelination (Thomas and Lascelles, 1966).

In contrast with the findings in the carpal tunnel patients, the reduction in the amplitude and velocity of the wrist and finger action potentials was of a comparable degree in most diabetic patients. This would suggest that the pathological changes in these patients were of equal severity in both segments of nerve examined.

Previous workers have demonstrated that nerve action potentials might be abnormal in the absence of clinical neurological involvement (Mulder et al., 1961; Gilliatt and Willison, 1962; Lamontagne and Buchthal, 1970). In the present study all the eight patients in whom clinical evidence of disease was lacking in the fingers had abnormalities of either amplitude or velocity of one of the potentials.

Even though the digital potential was not abnormal more frequently than the wrist potential in the diabetic subjects, two patients (S.K. and F.R.) illustrate the particular value of the present technique. Conduction velocity could not be calculated for the segment of nerve between the finger and wrist because no potential was recordable in these two patients—presumably at least partly due to dispersion of the volley over the longer conduction distance. However, over the much shorter distance in the finger a potential was recorded and in each instance marked slowing was found.

Figure 2 shows typical records of the potentials obtained from one subject in each group. In the carpal tunnel patients, the greater abnormality of the wrist than digital potential can be seen and also the considerable increase in latency of the wrist potential compared with the control. In the diabetic patient there was a marked reduction in amplitude and increase in latency of both potentials.

Figure 8 shows the conduction velocities for the digital and wrist potentials in the three groups which were studied. It may be seen that the greatest reduction in digital velocity occurred in the diabetic patients and that the greatest reduction in wrist potential velocity occurred in the patients with carpal tunnel syndrome. This latter finding is not unexpected as examination includes the damaged segment of compressed nerve in these patients.

In the present study it has been found that the technique of recording digital nerve potentials has been particularly useful in the following situations. In diabetic patients in some instances it has been possible to demonstrate slowing of conduction in the digital nerves when it has not been possible to record a wrist potential. In patients with the carpal tunnel syndrome the demonstration that conduction is more abnormal in the wrist segment than in the digital nerves provides additional support for the diagnosis of a local lesion at the wrist.

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