

## ANALYSIS OF MOTOR CONDUCTION VELOCITY IN THE HUMAN MEDIAN NERVE BY COMPUTER SIMULATION OF COMPOUND MUSCLE ACTION POTENTIALS<sup>1,2</sup>

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Measurement of motor nerve conduction velocity is an important part of the assessment of patients with peripheral nerve disorders. In routine clinical electrophysiology, this velocity is calculated by determining the latencies to the onset of muscle action potentials after supramaximal stimulation of the nerve at two or more sites. However, the value thus obtained represents the velocity of conduction in the fastest motor fibres only, and provides little information on conduction in other motor fibres within the nerve.

With supramaximal stimulation to a motor nerve the evoked compound muscle action potential represents a summation of all potentials generated by the individual motor units within the muscle. The size and shape of the compound potential is determined by the amplitude and duration of single motor unit action potentials, the number of motor units within the muscle, the range and distribution of conduction velocities in the alpha motor fibres, and the distance the impulse must travel along the nerve. Normally there are only slight differences between compound potentials from hand muscles after stimulation of their nerve at two sites such as the wrist and elbow. However, when conduction is abnormally slow there is a change in the size and shape of the compound potential, which becomes increasingly apparent with longer distances between stimulating and recording electrodes.

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trodes.

The present study is based on the premise that an analysis of differences in the size and shape of the potentials evoked by stimulation of a motor nerve at different sites might provide information on the conduction of a wide range of motor fibres. For this purpose we have developed a computer programme to reconstruct compound muscle action potentials similar to real potentials recorded from the thenar muscles after stimulation of the median nerve at the wrist and elbow. The number of motor units and the conduction velocities in motor fibres needed for accurate reconstruction of compound potentials were correlated with histometric studies of the recurrent branch of the median nerve of three cadavers. In addition, the computer model was used to imitate various patterns of slow conduction and determine the effect of these changes on the main parameters of the compound muscle action potentials.

### METHODS

#### 1. Single motor unit action potential measurements

Voluntary electromyographic (EMG) activity was recorded from the thenar muscles of four normal subjects whose ages ranged from 27 to 37 years<sup>3</sup>. Silver disc surface electrodes, 8 mm in diameter, were taped on the skin over the belly and tendon of the abductor pollicis brevis muscle. The two electrodes were separated

<sup>3</sup> This aspect of the study was carried out at the University of Toronto in accordance with a protocol approved by a Review Committee on Research Involving Human Subjects.

by a distance of approximately 4 cm. Because only a few units can be identified in the surface EMG, single motor units were isolated by a computer averaging technique (Stein *et al.* 1972). EMG activity was recorded simultaneously with a fine bipolar needle electrode, and spike discharges identified as belonging to a single motor unit were used to trigger the computer which averaged the surface EMG signal (Fig. 1). The surface EMG was delayed 5 msec by means of a DISA delay line (Model 14B80) to allow better visualization of the onset of the potentials. By averaging between 100 and 500 discharges of a motor unit, it was possible to record a clearly defined single unit action potential from the surface EMG, even at moderately strong levels of contraction. Action potentials from 20 to 30 different motor units were determined in this manner for each subject. Both low and high threshold units were obtained by having the subject vary the force of muscle contraction. The needle electrode was positioned in different places within the muscle to record close and distant units. In some cases a motor unit which had already been recorded was identified

again when the needle electrode was moved to a new site in the muscle. This situation was recognized by noting the size and configuration of the potential averaged from the surface electrodes, and the surface EMG was examined carefully to ensure that measurements from a single unit were not included more than once.

The single unit potentials were stored on digital tape and then displayed on the computer oscilloscope for study. Potentials from each subject were superimposed to align their onset and initial negative components, they were then averaged to give a representative single motor unit potential for each subject.

## 2 Compound action potentials after median nerve stimulation and maximal motor conduction velocity estimates

The surface electrodes were also used to record compound muscle action potentials after supramaximal stimulation of the median nerve at the wrist and elbow. The distance between stimulating sites was measured, and conduction velocity for the fastest motor fibres was calculated from the latencies to the onset of the

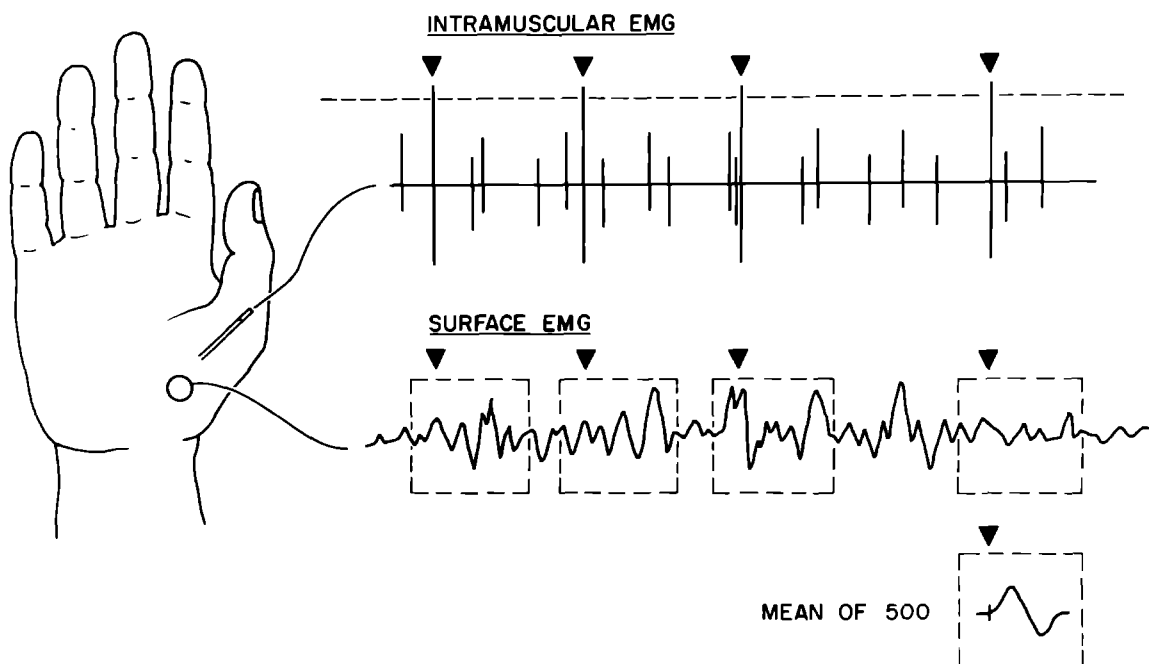


Fig. 1 Method for averaging single motor unit action potentials from the surface EMG. Spike discharges from single units are recorded with an intramuscular bipolar needle electrode and used to trigger an averaging programme which subsequently isolates the contribution of that unit from the surface EMG signal.

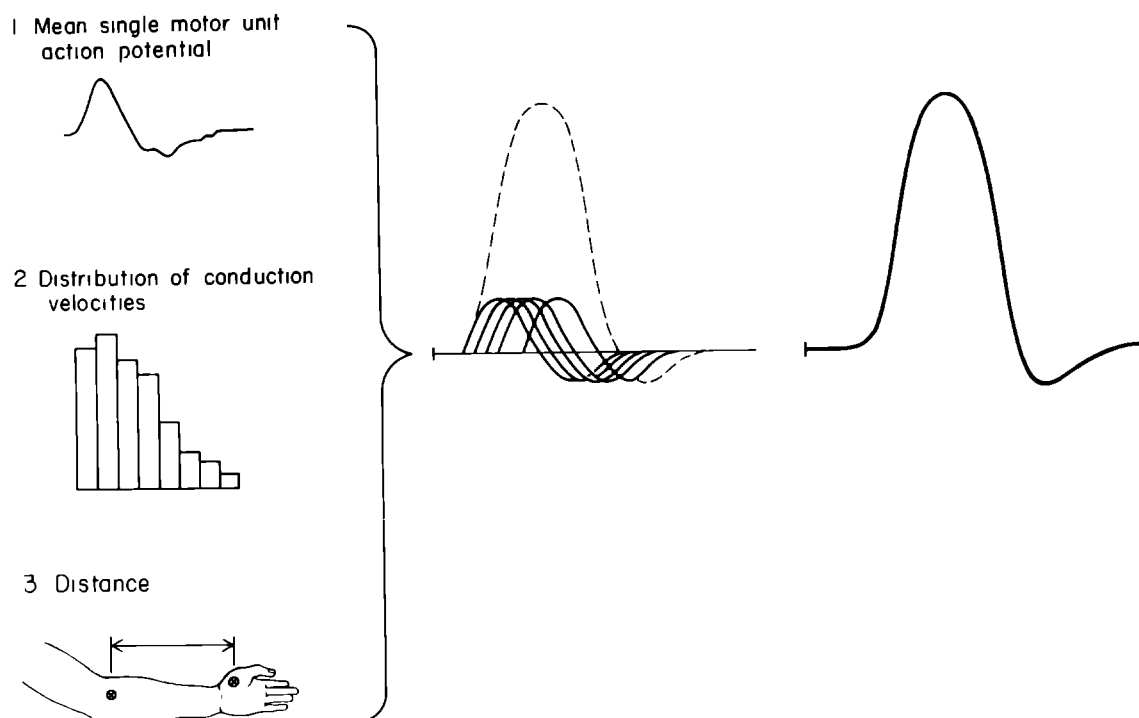


Fig. 2 Schematic diagram to show computer reconstruction of a compound muscle action potential. The computer summates a specified number of single motor unit action potentials, applying a temporal dispersion factor to account for differences in conduction velocity in individual motor fibres and for distance impulses must travel along the nerve.

initial deflections of the compound muscle action potentials

### 3 Computer simulation of compound muscle action potentials

A small laboratory computer (PDP 12A-8K Memory) was used to reconstruct compound muscle action potentials. The programme allowed the following parameters to be varied: (1) the dimensions of a representative single motor unit potential, (2) the number of motor units in the muscle and the range and distribution of conduction velocities in their nerve fibres, and (3) the distance along the nerve from the point of stimulation to the muscle.

The compound potentials were reconstructed by summing individual motor unit action potentials and applying a dispersion factor to account for the difference in conduction velocity in individual motor fibres as well as the time required for neuromuscular transmission (Fig. 2). The reconstruction programme was designed to allow the entry of histograms of nerve conduction velocities ranging from 15 to 75 m/sec, each bin

representing a range of 2.5 msec. For the initial simulation of normal compound potentials we used a range of velocities from 42.5 to 60.0 m/sec. To determine the effect of abnormal patterns of conduction on the compound potentials, histograms were used in which some or all of the fibres in the hypothetical nerves conducted at velocities slower than 42.5 m/sec.

An initial estimate of the number of motor units in the thenar muscle group was obtained by dividing the amplitude of the real compound potential by the amplitude of the mean single unit potential from each subject (McComas *et al* 1971). The total number of units thus obtained was then increased or decreased by trial and error to obtain the best possible match between the simulated potentials and the real compound potentials.

### 4 Histologic methods for estimating the number of motor fibres in the recurrent branch of the median nerve

The recurrent motor branch of the median nerve represents the major innervation of the

thenar eminence in most subjects (Sunderland 1968). This nerve was removed at autopsy from three male cadavers. Medical histories of these patients were reviewed to ensure that there was no clinical evidence of peripheral neurologic disease. All three cases were under age 45 and had died suddenly from myocardial infarction. The median nerve was exposed from the wrist to the origin of the digital branches and examined carefully to ensure that the recurrent motor branch represented the entire median innervation of the thenar muscles. A segment of the recurrent branch 1.0 cm in length was removed and fixed in 3% glutaraldehyde in 0.1 molar phosphate buffer (pH 7.4) at 4°C (Aguayo *et al.* 1971). A segment approximately 2 mm long was sectioned from the most proximal part of the recurrent branch under a dissecting microscope, post-fixed in osmium tetroxide, and embedded in epoxy resin according to standard methods. Phase microscopy sections 1  $\mu$ m thick, cut on an ultramicrotome with glass knives were fixed on glass slides and mounted in glycerin. Whole transverse section montages of all fascicles forming the recurrent motor branch were photographed

with a Zeiss photomicroscope. Photographs were printed at a final magnification of 1000 times and used to determine the number and diameter of myelinated fibres (Dyck *et al.* 1968). Myelinated fibre populations were determined by counting all myelinated fibres for each nerve. Fibre diameters were estimated for at least 80% of fibres; those not included were either too small or obliquely sectioned for accurate measurement to be possible.

## RESULTS

### 1. Single motor unit potential measurements

Single motor unit action potentials from one subject are shown in Fig. 3, and the results for all four subjects are summarized in Table I. There was a wide variation in the size of motor unit action potentials recorded with surface electrodes over the thenar eminence. This variation likely depends on the number of muscle fibres within each unit and the proximity of the unit to the recording electrode. The amplitudes of mean single unit potentials obtained by averaging all the single unit potentials for each of the four

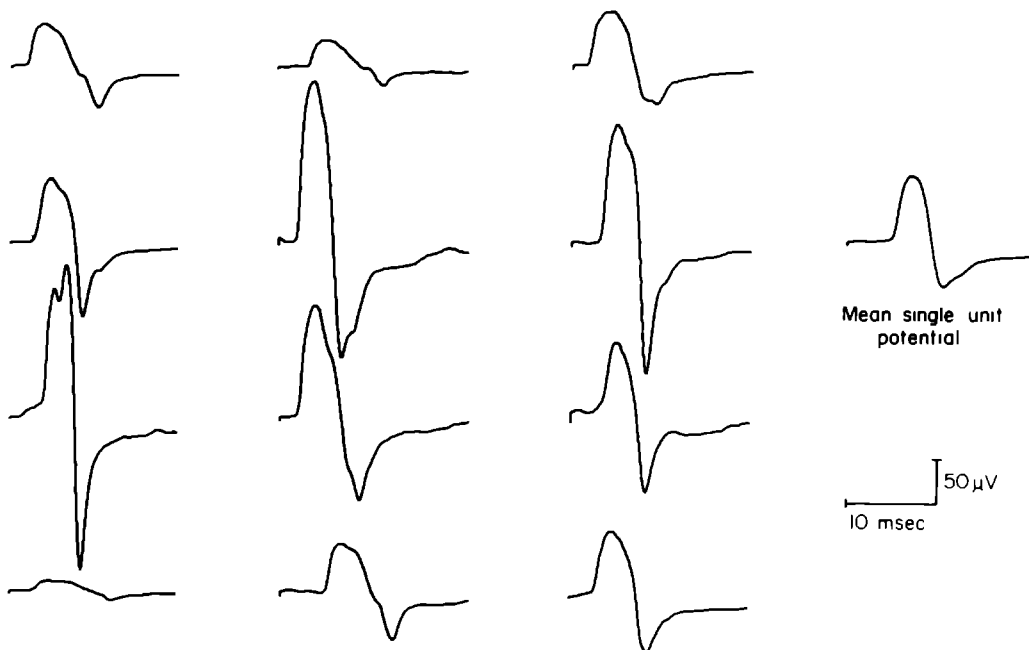


Fig. 3 Single motor unit action potentials from a normal subject (R.L.) recorded with surface electrodes over the thenar eminence. Each potential was obtained by averaging between 100 and 500 discharges of the unit during voluntary contraction. Twelve of the 29 potentials recorded are shown along with the mean potential obtained by averaging all 29. Amplitude of individual motor unit potentials ranged from 9 to 178  $\mu$ V. Amplitude of mean potential is 74  $\mu$ V.

TABLE I

Summary of data for single motor units and compound muscle action potentials recorded with surface electrodes from the thenar eminence of four normal subjects

	R L	D W	P T	R W	Mean values
* Amplitude of representative single motor unit action potential ( $\mu$ V)	74 **(9-178)	124 (23-314)	105 (12-497)	82 (20-397)	96
Number of single units recorded	29	18	20	20	22
* Amplitude of real compound potential (mV)	13.0	18.2	16.5	15.0	15.7
Number of units required to reconstruct compound potential	176	154	152	185	167
Conduction velocity of fastest alpha motor fibres (m/sec)	59.5	60.0	59.0	61.5	60.0

\* Amplitudes were measured from the baseline to the peak of the initial negative component of the potential.

\*\* Figures in parentheses refer to the range of amplitudes for single unit potentials

TABLE II

Histometric analysis of the recurrent motor branch of three human median nerves

	Nerve 1	Nerve 2	Nerve 3
Age of subject	32	43	44
Number of fascicles	2	3	4
Total number of myelinated fibres	1843	1495	1635
Size frequency histogram:			
Location of 1st peak of bimodal distribution ( $\mu$ m)	2.6	3.1	2.6
Location of 2nd peak ( $\mu$ m)	8.9	9.2	8.1
Location of trough ( $\mu$ m)	5.1	5.4	4.3

subjects were 74, 82, 105 and 124  $\mu$ V.

## 2. Compound action potentials after median nerve stimulation and conduction velocity in fastest motor fibres

Compound action potentials from the thenar muscles in the four subjects ranged in amplitude from 13.0 to 18.2 mV (Table I). Subjects with larger compound potentials also had larger mean single unit action potentials.

Values for maximal motor conduction velocity for the four subjects ranged from 59.5 to 61.0 m/sec, and a velocity of 60 m/sec was selected as the upper limit of motor conduction for reconstruction of simulated compound potentials. Direct measurements for slowest conduct-

ing alpha fibres were not made from real potentials. Conduction velocities from these four subjects were quite uniform and the amplitudes of the compound potentials were all near the upper limit of the normal range. The subjects were selected only because of their availability and willingness to participate in the study, but all four had rather bulky thenar muscles which may account for the large compound potentials.

## 3. Simulation of normal compound potentials

Simulated compound potentials for responses after stimulation at the elbow and wrist as well as the mean single unit potentials and histograms of conduction velocities used in the reconstruction are shown in Fig. 4. In all four subjects the initial negative component of the simulated and real potentials was similar. However, there was some difference in the later part of the potential, the simulated potentials having a larger terminal positive phase and a slower return to the baseline. The slight differences in rise time, amplitude, and duration of the negative component which occurred between potentials obtained with stimulation at the elbow and wrist were also seen in the simulated potentials. The number of units required to simulate compound potentials matching the real potentials varied from 152 to 185 (mean 167 units).

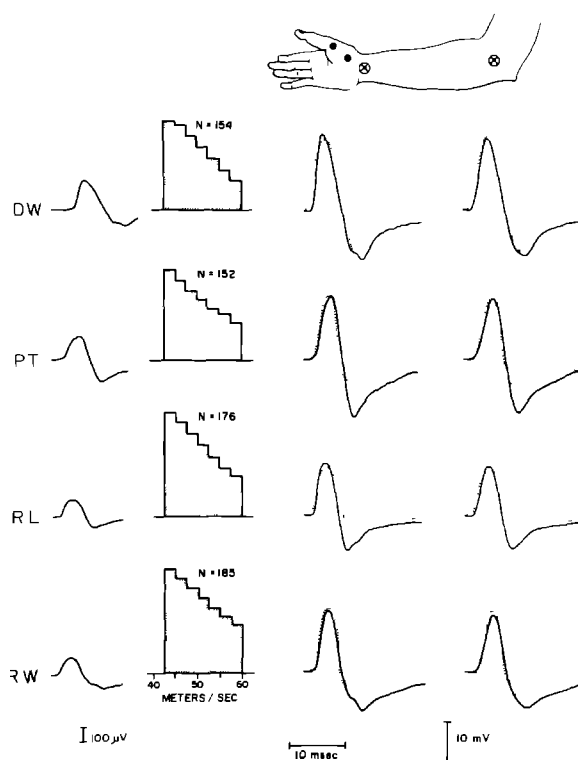


Fig. 4 Computer simulation of compound thenar muscle action potentials for four normal subjects. Potentials represent responses obtained with median nerve stimulation at the wrist and elbow. Real compound potentials for each subject are shown as dotted lines superimposed on the simulated potentials. In the left half of the illustration are the mean single motor unit action potential, distribution of conduction velocities and number of motor units (N) used for each reconstruction.

#### 4. Histologic studies of myelinated fibres in the recurrent motor branch of the median nerve

Myelinated fibres in the proximal segment of the recurrent motor branch were distributed in several fascicles, the number and size of such fascicles being different for all three nerves examined (Table II). There was no evidence of any pathology in these nerves, and myelination appeared normal on cross-section, although teased fibre preparations were not examined for evidence of segmental demyelination. The total myelinated fibre population was similar—1843, 1495 and 1635. These values were within the range observed for recurrent motor branches of median nerves from six other subjects 21–43 years of age (mean  $1562 \pm 163$ ; S.E. 66.5) and for nine nerves from subjects 58–73 years old (mean  $1229 \pm 192$ ; S.E. 65.08) (unpublished

observations). Data from these nerves were not included in the present study either because the ages did not match those of normal subjects used for the physiologic studies or because the distribution of myelinated nerve fibres was not clearly bimodal. For the three subjects in the present study individual size histograms of myelinated nerve fibres were bimodal with peaks at 2.6, 3.1, 2.6  $\mu$  and at 8.9, 9.2, 8.1  $\mu$  respectively. The trough between the two peaks of the histogram was at 5.1, 5.4 and 4.3  $\mu$  respectively (Table II).

#### 5. Anatomophysiologic correlation

Fig. 5 shows a composite size frequency histogram for myelinated fibres in the recurrent branch of the median nerve representing measurements from the three nerves examined. The mean value for the total number of myelinated fibres was 1658. This figure includes gamma fibres and muscle afferents as well as alpha motor fibres (Fig. 5). If it is assumed that the slowest alpha fibres in this age group conduct at 42.5 m/sec (Skorpiol 1965), and a conversion

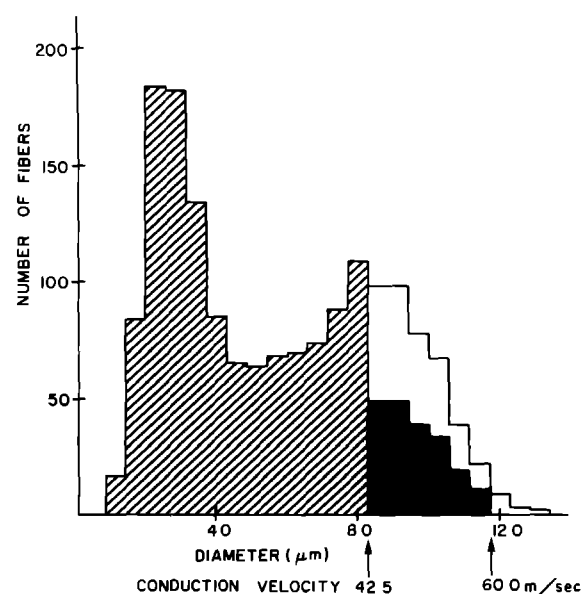


Fig. 5 Distribution of myelinated fibre diameters in the recurrent motor branch of human median nerve. Composite histogram represents measurements from three nerves. Cross-hatched area indicates gamma motor and small afferent fibres, white area the large afferents, and black area alpha motor fibres. Total number of myelinated fibres = 1658. Estimated number of alpha motor fibres = 203. Factor of 5.1 used for conversion from diameter to velocity.

factor of 5.1 is used to relate fibre diameter in microns to conduction velocity in m/sec (McLeod and Wray 1967), the diameter of alpha motor fibres should be greater than  $8.3 \mu$ . Similarly, if 60 m/sec is the upper limit for motor conduction (see foregoing explanation in second section 2 of Results) and it is assumed that 50% of the large fibres are efferent (see Discussion), a portion of the histogram between 8.3 and  $11.8 \mu$  should represent alpha motor fibres. From these calculations we estimated the mean alpha motor fibre population for the recurrent motor branch to be 203.

Because it is not possible to determine histologically how alpha fibres are distributed within this range of 8.3 and  $11.8 \mu$  we have assumed their diameters are distributed as in the total histogram for the range (Fig 5). In effect, changing the shape of the histogram does not significantly alter the compound potentials as long as the range of velocities is kept constant at 42.5–60.0 m/sec. We have reconstructed poten-

tials for several distributions of conduction velocities within this normal range for alpha motor fibres and in each case the compound potentials have been virtually identical.

#### 6. Simulation of abnormal patterns of conduction

The effect on the compound potentials of an identical slowing of conduction in all motor fibres is shown in Fig. 6. Potentials were reconstructed for three different ranges of conduction: 40–60, 30–50 and 20–40 m/sec. For each case the distribution of conduction velocities within the range of 20 m/sec and the total number of motor units were kept constant.

When impulses are conducted over a short distance—wrist to muscle—there is little change in the amplitude and shape of compound potentials even with velocities in the 20–40 m/sec range. However, with conduction over longer segments of the nerve the differences in conduction velocity for individual fibres cause increased separation among the individual motor unit

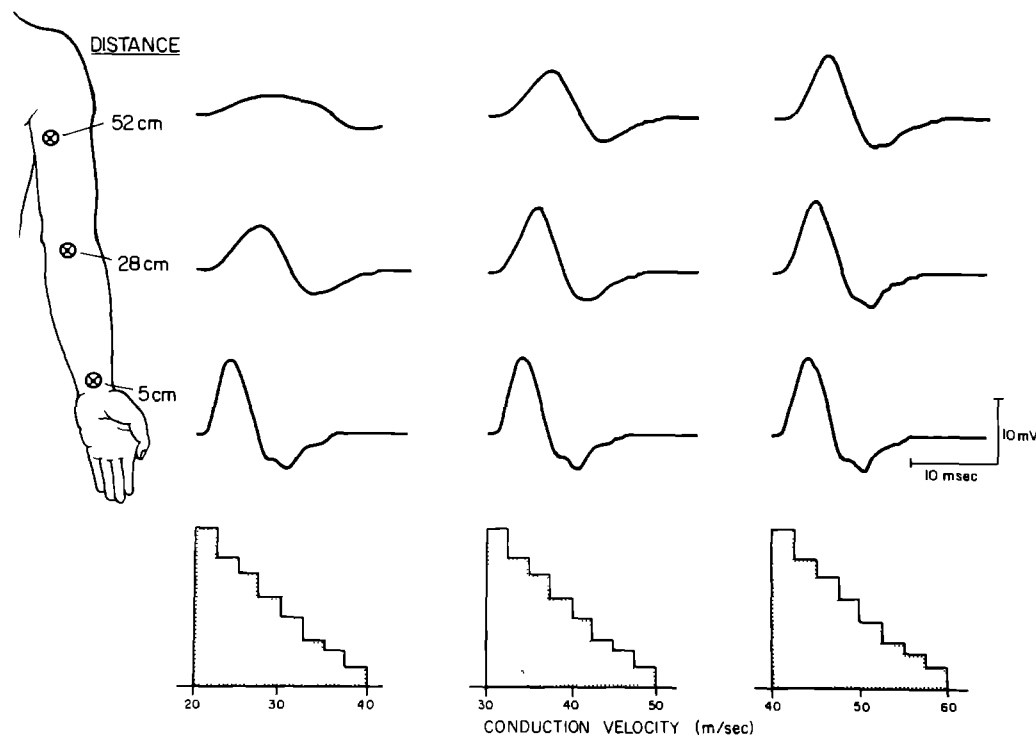


Fig 6 Simulated compound thenar muscle action potentials reconstructed by using three different ranges of conduction velocity. Number of motor units, mean single unit action potential, and conduction distances are the same in each case. The three potentials shown with each histogram simulate responses after stimulation of the median nerve at the axilla, elbow and wrist.

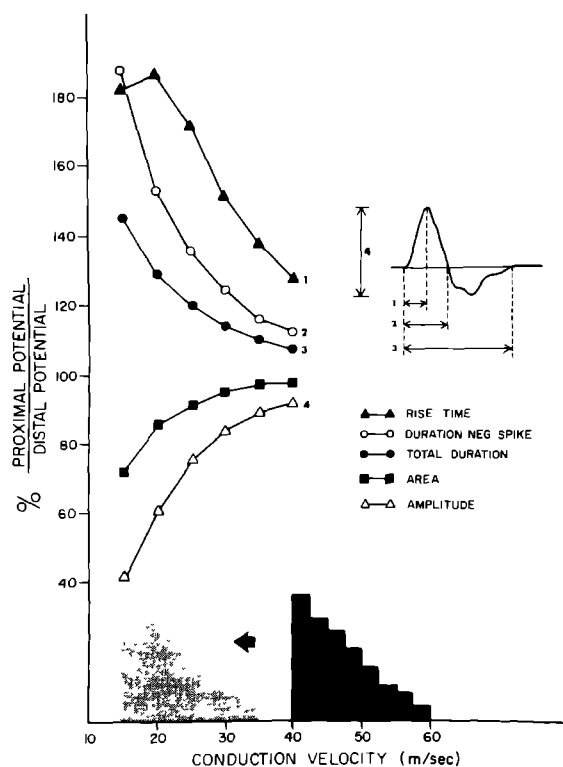


Fig 7 Comparison of pairs of simulated compound potentials after stimulation of the median nerve at wrist and elbow. Points represent measurement for a parameter of the thenar potential after stimulation at the elbow expressed as a percentage of the same parameter for the potential when the nerve is stimulated at the wrist. Six different pairs of potentials were generated by shifting the histogram of conduction velocities to the left in successive steps of 5 m/sec.

potentials that form the compound muscle action potential. As a result the compound potential decreases in amplitude and increases in duration as conduction velocity becomes slower.

To quantitate these changes we compared the main parameters of pairs of potentials obtained by stimulation of a hypothetical nerve at two different sites and plotted the results against changes in conduction velocity (Fig. 7). The rise time to the initial peak appears to be a particularly sensitive indicator of slight slowing of conduction.

Uniform slowing of conduction affecting all motor fibres to the same degree, as shown in Fig. 6, probably does not occur with most peripheral neuropathies. The computer model permits one to simulate many situations where different groups of fibres are affected to varying

degrees. Another pattern of abnormal conduction is illustrated in Fig. 8 where compound action potentials are reconstructed for two hypothetical nerves. In each case there were 200 motor units and the fastest fibres conducted at 50 m/sec. Conventional methods for measuring conduction velocity by using the latencies to the onset of these potentials would give identical values for both nerves. However, in one of the nerves many fibres are conducting at an abnormally slow velocity, and there is a significant dispersion of the potential evoked from a proximal stimulating site.

## DISCUSSION

Previous studies on reconstructed compound potentials have concentrated on nerve action potentials. Gasser and Erlanger (1927) reconstructed the monophasic action potential of an isolated nerve comparing conduction velocities and external fibre diameters. Buchthal and Rosenfalck (1966) did the same for sensory action potentials using single fibre potential measurements and histologic data from human digital nerves.

In the present investigation reconstructed compound muscle action potentials closely resembled real potentials from the thenar muscles obtained with supramaximal stimulation of the median nerve. However, in the four subjects studied there was a difference in the late positive component of these potentials, the simulated potentials showing a larger late positive deflection and a less rapid return to the baseline. One explanation for this finding is that the real compound potential may include a small contribution from the first two lumbrical muscles activated by stimulation of the median nerve. A slightly greater latency of the responses from the lumbrical muscles could result in an alteration of the late phase of the compound potential recorded over the thenar eminence. Simultaneous recording with needle electrodes in both the lumbricals and the thenar muscles might help determine the contribution of the lumbricals to the thenar compound potentials, but this was not included as part of the present study.

Several assumptions have been made for our reconstruction of compound muscle action potentials. Firstly, we have assumed that the mean



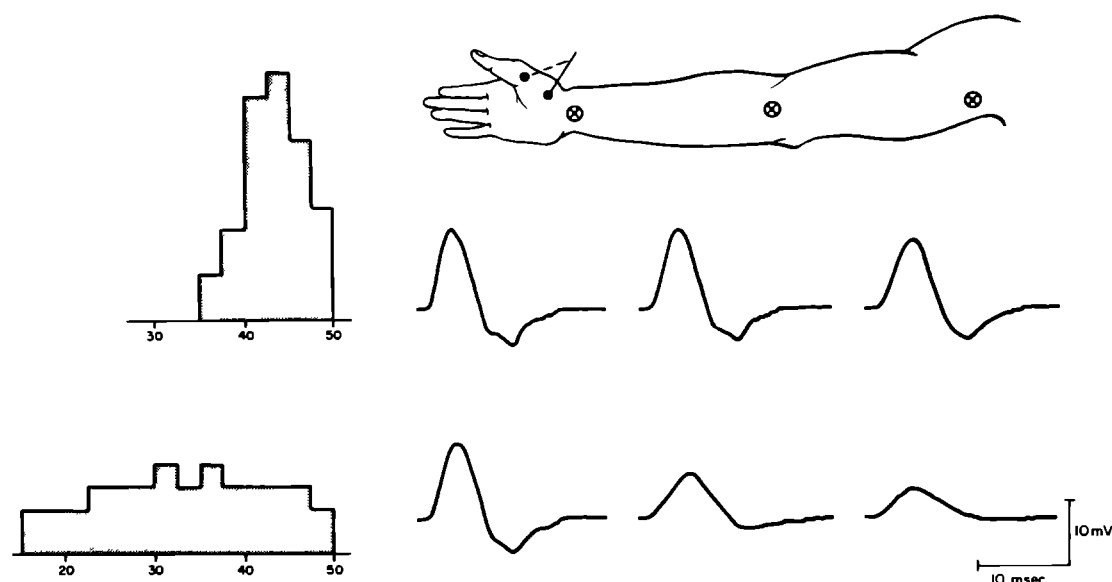


Fig 8 Simulated compound muscle action potentials corresponding to stimulation of the median nerve at the wrist, elbow and axilla reconstructed from two distributions of conduction velocities. In each case the total number of motor units was 200 and the maximal conduction velocity was 50 m/sec. Potentials evoked from proximal sites of stimulation are of lower amplitude and longer duration when the histogram of conduction velocities includes slower conducting fibres.

single motor unit action potentials are representative of the average size and shape of single unit potentials recorded by surface electrodes over the thenar eminence. Although we recorded different motor units by varying the position of the needle electrode and by changing the strength of voluntary contraction of the thenar muscles, there was a wide variation in the size of single unit potentials. In addition, the number of units recorded (20–30 for each subject) may not have been sufficient to provide a representative mean. Secondly, it has been assumed that potentials obtained by the averaging method are potentials generated by single motor units. This should be true if the motor unit whose spike triggers the averaging routine always discharges independently of other motor units. If, however, two or more units discharged synchronously the average potential would represent activity from more than one unit. Milner-Brown *et al* (1973) have studied synchronization during voluntary muscle contraction and developed a method for detecting synchronized firing of motor units. They found that synchronization was rare in normal subjects, but occurred in individuals who were engaged in heavy manual labour (Milner-Brown *et al* 1974). Because none of our subjects

carried out activities that required repeated strong contractions of hand muscles, we assumed that potentials were generated from single motor units.

A third assumption used in developing the model was that conduction in individual axons remained uniform along the entire length of the nerve fibres. Although it is recognized that conduction is slower in the small terminal branches of motor axons (Kaesler 1970), if such slowing is approximately the same for all motor fibres, summation of single units to form the compound potential should not be significantly altered. However, possible slowing in distal portions of the elbow to wrist segment of the median nerve due to a temperature gradient or tapering of axons (Kaesler 1970) was not considered in the computer programme we used to reconstruct potentials. The model also does not consider common factors that alter compound muscle action potentials in peripheral neuropathies—variation in conduction velocity along a nerve fibre due to segmental demyelination (Rasminsky and Sears 1972) and alterations in the size and shape of single motor unit action potentials occurring as a result of denervation and reinnervation (Kugelberg 1947).

In selecting a range of conduction velocities for single alpha motor fibres, the upper limit of 60 m/sec was based on measurements of the fastest conduction velocities for the median nerve in our four subjects. Values for the lower limit of conduction velocity in alpha motor fibres are more difficult to estimate. Thomas *et al.* (1959) indicated that the velocity in the slowest fibres was 30–40% below that of the fastest fibres. Using paired stimulation techniques, Hopf (1963) postulated that the slowest alpha motor fibres, conducted only 4–7 m/sec slower than the fastest motor fibres. Using a similar technique Skorpil (1965) found the velocity of alpha motor fibres to be within a range of  $43.4 \pm 2.9$  m/sec to  $60.1 \pm 2.9$  m/sec for the ulnar nerves of 33 healthy subjects. Lambert (1969) suggested that this range of conduction velocity in the motor fibres could account for the only slight slowing of conduction in patients with amyotrophic lateral sclerosis where the larger, fast conducting fibres are lost early in the disease. In the present reconstruction of normal compound potentials a range of velocities between 42.5 and 60 m/sec provided the best match between simulated and real potentials.

The number of motor units needed for a reconstruction of the compound potential from the thenar muscles for the four subjects studied ranged from 152 to 185 with a mean value of 167. Sica *et al.* (1974), using an electrophysiologic technique (McComas *et al.* 1971) estimated there were  $340 \pm 87$  motor units in the human thenar muscles. With a similar technique Brown (1972) calculated the thenar motor unit population to be  $253 \pm 34$ . These electrophysiologic estimates are higher than the number of motor units which we required to reconstruct the thenar compound potential. This difference may be due to a different estimation of the mean amplitude for single motor unit action potentials. Brown (1972) found this to be 53  $\mu$ V for the 59 subjects he studied, whereas the mean single unit potential size for our subjects was 96  $\mu$ V (range 74–124  $\mu$ V). The electrophysiologic method for recording single unit potentials in our study differed from that used by McComas *et al.* (1971) and Brown (1972) who used weak electrical stimuli to activate the first 5–15 units and determined the mean increment in amplitude due to a single motor unit.

It is not certain whether the first units activated by electrical stimulation are representative of all motor units in the muscle. If smaller motor units were selectively activated because of a lower threshold or the relative proximity of their fibres to the stimulating electrode, the estimate of mean amplitude of a motor unit action potential would be low and consequently the count for total number of motor units would be high. Indeed, Feasby and Brown (1974), studying the size of motor unit increments in the F response obtained with graded stimulation of the median nerve, observed some late units larger than those activated by stimuli just above the threshold of motor fibres.

In our study single motor units were recorded during voluntary activation and special efforts were made to include both low and high threshold units from different locations in the thenar muscles. Providing that there was no significant degree of synchronization between different units and that the averaging technique extracted only potentials generated by single units, this method should provide a reliable estimate of the size and shape of the mean single unit potential.

Histologic examination of the recurrent branch of the median nerve provides another method for estimating the number of alpha motor fibres that innervate the muscles of the thenar eminence. Interpretation of fibre counts by this method also requires several assumptions. First, it has to be assumed that the recurrent branch represents the entire median innervation of the thenar eminence. Although there are anomalies in the innervation of the small hand muscles, visual inspection of the median nerves in the cadavers used for this study did not reveal accessory branches to the thenar muscles. Estimating number of motor units by counting myelinated fibres from cross-sections of a peripheral nerve is valuable only if it can be assumed that there is no significant branching of motor axons. Eccles and Sherrington (1930) demonstrated an increase in the number of fibres in the distal part of a muscle nerve as a result of branching, but felt that such branching would only slightly increase the original number of motor fibres to the muscle. However, a more recent study suggests that a significant amount of branching of motor fibres may occur before the

nerve enters the muscle (Wray 1969). In the nerves we examined it was possible to study serial cross-sections over a distance of approximately 8 mm, and there was a less than 5% variation in the total number of myelinated fibres for this segment, suggesting that branching is uncommon at this level.

Conduction velocity for myelinated fibres is directly proportional to fibre diameter (Gasser and Grundfest 1939, Hursh 1939), but various ratios have been suggested to relate conduction velocity to fibre diameter. This ratio varies for different nerves, species and even for different fibre sizes (Paintal 1973). Suggested values for a conversion factor include 6.0 for the largest fibres in a mixed nerve from the cat (Hursh 1939), 5.7 for large motor fibres in a muscle nerve from the cat (Boyd 1965), 4.4 for the fastest fibres in the rabbit peroneal nerve (Cragg and Thomas 1964) and 5.3 for intact ulnar and median nerves of the baboon (McLeod and Wray 1967). The latter authors found that in deafferented nerves of the baboon the ratio of conduction velocity to fibre diameter was only 4.1, but indicated that this ratio could have been influenced by some unavoidable damage to ventral roots.

Examination of cat deafferented nerves by Rexed and Therman (1948) showed that afferent fibres accounted for 43% of the large fibres to the anterior tibial muscle and 34–37.5% of those to the medial head of the gastrocnemius. Wray (1969) has found that 48% of the large fibres in the nerve to the abductor pollicis brevis in the baboon are efferent. The relative proportion of efferent and afferent fibres in a muscle nerve obviously varies according to the particular muscle and species being studied, and accurate figures for human nerves are not available. In analysing our histologic data we have assumed that 50% of the large myelinated fibres are efferent.

An average of 167 motor units was required to reconstruct compound muscle action potentials matching the real compound potentials in our subjects. Assuming that 50% of the large myelinated fibres identified by histologic methods represent alpha motor fibres which conduct in a range of 42–60 m/sec (see above), and applying a conversion factor of 5.1 to relate diameter of myelinated fibres to conduction

velocities in m/sec, the average number of alpha motor fibres for the nerves examined histologically was 203. These two values (167 and 203) are sufficiently close to justify the use of an electrophysiologic model to study the effects of abnormal conduction on the form of the reconstructed compound potentials.

The main observation which has resulted from the study of abnormal conduction patterns is that slowing of conduction is associated with a greater than normal discrepancy between the main parameters of compound potentials generated from the thenar muscle after distal and proximal stimulation of the median nerve. This difference is apparent even when the fastest alpha motor fibres still conduct at a normal velocity—a situation in which conventional clinical methods for determining conduction velocity would not indicate an abnormality. When amplitude and duration of the entire potential and of its components and the area of the potentials are measured, the best indicator of slight slowing of conduction is a prolongation in the rise time of the initial negative peak of the potential evoked from the proximal stimulating sites.

Theoretically, it should be possible to do the reverse of what is done in reconstructing potentials, that is to extrapolate from the difference between two compound potentials the distribution of conduction velocities for individual fibres of the nerve. However, to do this would require a more complex mathematical analysis of the potentials than what we have so far carried out, and it is possible that more than one distribution of conduction velocities might give rise to identical compound potentials. Nevertheless, we anticipate that further work with this model will lead to methods for analysing compound muscle action potentials which should allow us to do a more complete assessment of abnormal nerve conduction in clinical situations.

#### SUMMARY

A digital computer was used to reconstruct compound muscle action potentials recorded from the human thenar eminence after stimulation of the median nerve. The programme allowed the following parameters to be varied. (1) the dimensions of a representative single motor

unit potential; (2) the number of motor units in the muscle and the range and distribution of conduction velocities in their nerve fibres; and (3) the distance along the nerve from the point of stimulation to the muscle. The reconstructed compound muscle action potentials were similar to real compound potentials recorded from normal subjects. The number of single motor units and the range of conduction velocities required for the reconstruction correlated with quantitative histologic studies of the recurrent branch of the median nerve to the thenar muscles.

By altering the distribution of conduction velocities it was possible to study the effect of abnormal patterns of nerve conduction on the configuration of the simulated compound muscle action potentials. It was found that abnormally slow conduction caused an increased discrepancy between the main parameters of compound potentials corresponding to stimulation of the nerve at proximal and distal sites.

These observations suggest that a careful analysis of the differences between pairs of compound muscle action potentials may provide a method for more detailed assessment of conduction velocity in clinical studies of peripheral nerve disorders.

#### RESUME

#### ANALYSE DE LA CONDUCTION MOTRICE DU NERF MEDIAN CHEZ L'HOMME PAR SIMULATION SUR ORDINATEUR DES POTENTIELS D'ACTION DU MUSCLE

Un ordinateur digital a été utilisé afin de reconstruire les potentiels d'action des muscles de l'éminence thenar humaine après stimulation du nerf médian. Le programme permet de faire varier les paramètres suivants (1) les dimensions du potentiel d'action d'une unité motrice représentative, (2) le nombre d'unités motrices dans le muscle et la distribution des vitesses de conduction dans leurs fibres nerveuses; (3) la distance longitudinale du nerf à partir du point de stimulation jusqu'au muscle. Les potentiels d'action reconstruits sont similaires aux potentiels d'action véritables enregistrés chez des sujets normaux. Le nombre d'unités motrices simples et l'amplitude des vitesses de conduction requises pour la reconstruction correspondent aux études histologiques quantitatives faites sur la branche

"récurrente" du nerf median jusqu'aux muscles thenar.

En modifiant la distribution des vitesses de conduction il a été possible d'étudier l'effet de patrons anormaux de conduction nerveuse sur la configuration des potentiels d'action. On a également trouvé qu'une conduction anormalement lente cause une différence plus grande entre les paramètres principaux des potentiels d'action correspondant à la stimulation du nerf aux sites proximaux et distaux.

Ces observations suggèrent qu'une analyse soignée des différences entre les paires des potentiels d'action peuvent fournir une méthode donnant une estimation détaillée de la vitesse de conduction dans des études cliniques des désordres des nerfs périphériques.

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