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An improved technique for radial nerve conduction studies

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A technique for recording evoked sensory potentials from the radial nerve has already been reported by us (Downie and Scott, 1964). This technique, although reliable, is time consuming and occasionally difficult and the amplitude of potentials may be as low as 1 to 2 microvolts. The purpose of this communication is to describe a simpler technique by which potentials of greater amplitude can be obtained. In addition, the segment of nerve tested is one which can be readily identified and biopsied if so desired without causing unpleasant or disturbing sensory loss to the subject.

The location of a nerve which is to be tested is usually determined by stimulating its motor fibres and finding the stimulus site from which maximal muscle contraction is obtained. In the case of pure sensory nerves, electrodes of rather wide extent are used, such as ring electrodes for the digital nerves, or the broad electrode around the radial aspect of the wrist as described in the previous report (Downie and Scott, 1964).

In studying the anatomy of the superficial terminal branches of the radial nerve it can be seen that one or more branches pass superficial to the tendon of extensor pollicis longus. In most people these can be palpated by running an examining finger along the tendon. Active extension of the thumb makes such examination easier. The largest branch crosses the tendon about 1 cm. distal to the extensor retinaculum. This site is sufficiently constant to allow accurate placement of an active recording electrode even when the nerve cannot be palpated. Proximally the superficial branches of the radial nerve merge and run along the radial border of the forearm on its flexor surface in close apposition to the cephalic vein to a point just distal to the mid forearm where the nerve passes more deeply.

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MATERIALS AND METHOD

The apparatus used was a TECA two-channel electromyograph. The recording electrodes consisted of a pair of chlorided silver discs 1 cm. in diameter, mounted 2·5 cm. apart on a plastic base. The active recording electrode was placed over the largest palpable branch of the radial nerve as it crossed the tendon of the extensor pollicis longus. The distal recording electrode was placed over the first dorsal interosseous muscle but not necessarily over the nerve, of which the position in this area cannot be precisely determined (Fig. 1). An experiment made to assess the importance of the position of this electrode showed no significant difference in latency to peak when it was placed in turn on three points along a line between the tendons of the extensor pollicis longus and extensor indicis provided the interelectrode distance was maintained constant. The amplitude of the potential was least when the electrode was nearest the tendon of the extensor indicis when the recording was more nearly of monopolar than bipolar type. Even here, however, the smallest potential was only 25% less than the largest. The stimulating electrodes consisted of a pair of rods 1·75 cm. apart whose tips were covered with gauze soaked in saline. These were placed over the nerve near its mid forearm position. The cephalic vein

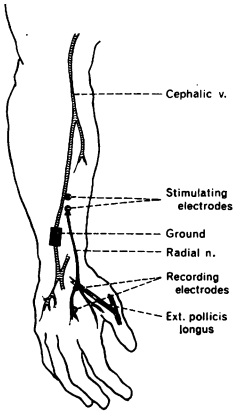

FIG 1. *Diagram to show electrode placement.*

was used as a landmark but even when it could not be seen stimulating electrodes were placed in this approximate position and then moved medially or laterally until an evoked sensory potential was obtained by antidromic conduction. Orthodromic conduction was performed on occasion by reversing the position of the stimulating and recording electrodes.

To establish a normal range for adults the technique was tested in 50 normal subjects aged between 11 and 64 years. Individuals with various types of neuropathy were also tested. A stimulus of 0·1 msec. and of amplitude up to 250 volts sufficient to produce a maximal evoked potential was used. The latency from the onset of the stimulus artefact to the peak of evoked potential was correlated with the measured distance between the stimulating cathode and the midpoint between the two recording electrodes. From this, the conduction rate was estimated.

Latency to peak was used rather than latency to 'take off', as in pathological states where the amplitude is low this may be the only point which can be accurately measured. As this point in time occurs later than the time of arrival of the impulse at the nearer recording electrode, latency to peak has been correlated with distance from the stimulating cathode to the midpoint between recording electrodes, rather than to the nearer recording electrode. Reasons for such a compromise have been discussed before (Downie, 1964).

RESULTS

Using this method, very satisfactory evoked potentials were obtained in all control subjects. Antidromic conduction more consistently, but not always, produced a higher amplitude response than did the orthodromic method (Fig. 2 A and B). The results in control subjects are shown in Table I.

TABLE I

RADIAL NERVE CONDUCTION IN 50 CONTROL SUBJECTS

	Range	*Mean*
Distance (cm.)	8·4—16·6	12·8
Latency (msec.)	1·8—3·2	2·4
Amplitude (μV)	5—20	11·4
Rate (m./sec.)	47—64	53·7[1]

[1]Standard deviation ± 3·8 metres per second.

The mean conduction rate for the 50 control subjects was 53·7 metres per second with a range of 47 to 64 metres per second. The mean conduction rate in the 16 patients over the age of 40 years was 52·4 metres per second as compared with 54·2 metres per second for the 34 patients under the age of 40.

The technique was also tested in several patients

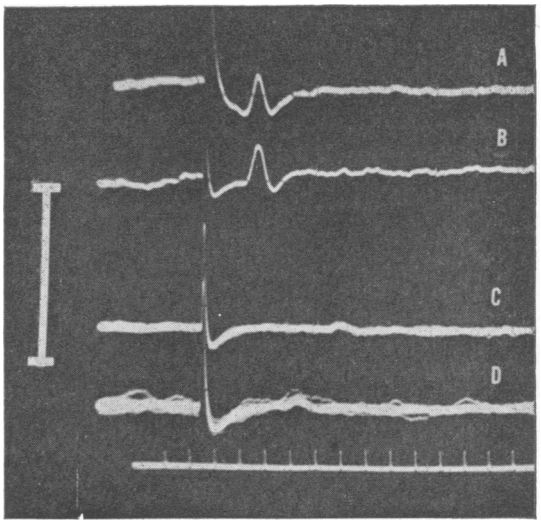

FIG. 2.A *Single sweep, orthodromic conduction.*

FIG. 2.B *Single sweep, antidromic conduction.*

FIG. 2.C *Multiple superimposed sweeps of conduction over radial nerve from wrist to spiral groove by previous technique. Note the small potential with 5·5 millisecond latency.*

FIG. 2D. *Multiple superimposed sweeps yielding a small potential with 3·8 millisecond latency recorded from spiral groove. Stimulating electrodes over radial nerve in midforearm.*

The amplitude marker represents 50 microvolts and time marker is at one millisecond intervals.

in whom abnormal findings might be expected (Table II). In two patients with diabetes mellitus but without clinical evidence of neuropathy conduction rate was within the normal range. In five other patients with clinical evidence of diabetic neuropathy affecting the lower limbs the conduction rate varied between 38 and 45 metres per second in four (more than 2 standard deviations lower than the mean control rate) and in the fifth no evoked potential could be elicited in the radial nerve. This last patient had clinical evidence of sensory neuropathy in the upper limbs as well as the lower. One alcoholic patient with evidence of mild peripheral neuropathy also showed a slower rate of 44 metres per second in the radial nerve. In three additional patients with polyneuropathy, one due to arsenic and the other two of undetermined cause, no potential could be obtained.

Table III indicates the findings in patients who had received trauma to the radial nerve. Patients 1 to 5 developed the wrist drop during sleep and

TABLE II

RADIAL NERVE CONDUCTION RATES IN PATIENTS WITH DIABETES MELLITUS AND POLYNEUROPATHY

No.	*Age and Sex*	*Diagnosis*	*Involvement with Neuropathy*	*Conduction Rate (metres per sec.)*
1	38 F	Diabetes mellitus	None	65
2	43 M	Diabetes mellitus	None	51
3	32 M	Diabetes mellitus	Lower extremities	38
4	55 M	Diabetes mellitus	Lower extremities	40
5	54 M	Diabetes mellitus	Lower extremities	45
6	80 F	Diabetes mellitus	Lower extremities	45
7	45 M	Diabetes mellitus	All extremities	N.P.[1]
8	46 M	Alcoholism	Lower extremities	44
9	17 M	Arsenic ingestion	All extremities	N.P.
10	48 F	Polyneuropathy of undetermined cause	All extremities	N.P.
11	16 F	Recurrent polyneuropathy of undetermined cause	All extremities	N.P.

[1]N.P.—No potential could be elicited.

TABLE III

RADIAL NERVE CONDUCTION RATES IN PATIENTS WITH TRAUMA TO RADIAL NERVE

No.	*Age and Sex*	*Aetiology*	*Initial Disability*		*Course*	*Duration at Time of Study*	*Conduction Rate (metres per sec.)*
			Weakness	*Sensory Loss*			
1	27 M	Compression during sleep	Severe	Yes	Improved rapidly; recovered in 2 months	1 month	62
2	38 M	Compression during sleep	Severe	Yes	Sensation recovered rapidly; well in 4 months	1 month 2 months	44 50
3	57 M	Compression during sleep	Severe	Mild	Sensation recovered in 1 month; well in 3 months	3 weeks	50
4	37 M	Compression during sleep	Moderate	Yes	Improving at time of examination	1 week	58
5	52 M	Compression during sleep	Severe	Yes	Improving in 2 weeks; recovered in 3 months	1 week 2 weeks 6 months	44 38 47
6	62 F	Compression during sleep Diabetes mellitus	Severe	Yes	Weakness became more severe and did not improve	4 days 7 days 1 month	52 N.P.[1] N.P.
7	21 M	Compression Alcoholism	Severe	Yes	Sensation improved rapidly; weakness improved slightly	7 weeks	56
8	21 M	Brachial neuritis	Severe	Yes	No change	1 month	N.P.
9	42 M	Penicillin injection in arm	Severe	Yes	Weakness improved only slightly. Still had sensory loss	5 months	N.P.
10	59 F	Nerve involved by lipoma in arm; nerve divided and reanastomosed at surgery	Severe	Yes	No improvement	1 year	N.P.

[1]N.P.—No potential could be elicited.

were thought to have no other contributing causes. Each of these was improving at the time of the examination. In the four instances where follow-up data are available, complete recovery occurred in two to four months. A potential was obtained in each of these but in two instances the rate of conduction was slow early in the course and later was normal. Two additional patients had developed a wrist drop during sleep, one with diabetes mellitus, the other with alcoholism, but neither had evidence of a polyneuropathy. The alcoholic patient had a normal conduction rate and improved much the same as cases 1 to 5. The diabetic patient had a normal conduction rate but low amplitude potential in four days and no potential by the seventh and thirtieth day. She did not improve clinically during this time. Three patients, cases 8, 9, and 10, developed a radial nerve palsy secondary to brachial neuritis, penicillin injection, and lipoma respectively. No potential could be obtained in any of these and they did not improve clinically. Sensory loss in the distribution of the radial nerve was present initially in every case either by history or on neurological examination. In all of the cases due to compression, sensation recovered first and more rapidly than did motor function.

DISCUSSION

A mean conduction velocity of 53 metres per second in this present study is similar to the conduction rate in the wrist-to-elbow segment of the median and ulnar nerves and faster than the conduction rate in the finger-to-wrist segments as determined in this laboratory. The mean amplitude of the potential (11 microvolts) evoked in this way is about three times greater than by the previous technique (Downie and Scott, 1964). This is partly due to the closer apposition of the recording electrodes to the nerve and also to the reduction of temporal spread of individual fibre potentials associated with shortening of the length of the nerve tested. The mild slowing in conduction rate in older control subjects and the findings in patients with neuropathies are similar to what has been observed by previous techniques in other nerves (Mulder, Lambert, Bastron, and Sprague, 1961; Norris, Shock, and Wagman, 1953).

It is interesting that all patients with uncomplicated compression lesions of the radial nerve had normal conduction rates at some time during the period of follow-up, and showed improvement or recovery clinically during this time. In two of these the conduction rate was slowed to 44 metres per second but later the rate returned to normal. The site of involvement of the radial nerve was proximal to the division into the posterior interosseous branch and the superficial sensory branch because in each case there was weakness of the brachioradialis muscle with a normal triceps contraction. Possible reasons for the preservation of the evoked sensory potential distally could be either that the nerve injury produced a segmental conduction block (neurapraxia) or that the sensory fibres were selectively spared because of a less vulnerable position within the nerve. The more rapid recovery of sensation in these cases as compared with strength might suggest a milder degree of injury to the sensory fibres. In two patients (cases 9 and 10) with severe direct injury to the radial nerve absence of potential correlated with absence of clinical improvement. Since the group of patients is small definite conclusions cannot be reached but a preserved potential may indicate a milder injury and therefore more favourable prognosis for recovery, provided examination is carried out after an interval of at least five days from the time of injury. Such a time is necessary to allow degenerative changes caused by a more proximal lesion to affect conduction distally. Case 6 (Table III) emphasizes the importance of this delay.

As abnormal findings on conduction rate testing may antedate clinical abnormality in the more chronic neuropathies, this test may offer a useful guide in the selection of suitable subjects who are being considered for nerve biopsy. Nerve biopsies in the past have been more commonly taken from the lower limbs, particularly the sural nerve. Evoked sensory potential examination has also been carried out in this (Deaton and Downie, 1967). In 78 control subjects an evoked potential could be obtained from the sural nerve in 58 (73%) and showed a mean conduction rate of 41 metres per second. As these potentials could not be regularly obtained even in normal subjects, their absence in a patient would not be adequate evidence on which to select the sural nerve for biopsy study. Such is not the case with the radial nerve as potentials were obtained in all control subjects.

The authors have had the opportunity to observe four patients in whom the superficial sensory branch of the radial nerve was biopsied. The specimen was taken from the largest branch crossing the tendon of the extensor pollicis longus. The ability to locate this branch by palpation through the skin made the procedure very simple. Two patients had diabetes mellitus with mild polyneuropathy. One patient had a recurrent, predominantly motor, polyneuropathy, and one a chronic mixed polyneuropathy of unknown cause. Follow-up of the first three after one year showed no clear-cut sensory loss in radial nerve distribution and no unpleasant dysaesthesiae had been experienced. In the fourth, no increase in sensory loss present before biopsy could be noted.

It is possible that conduction studies could be carried out similarly on many more cutaneous nerves provided only that they are sufficiently superficial and constant enough in position so that stimulating and recording electrodes can be accurately sited. This, however, is a major qualification. In cooperative control subjects superficial sensory nerves can be located by the subjective radiating (shock) sensation which passes along the distribution of the nerve when a stimulating electrode is close to it. By this means recording has been made by us from the medial cutaneous nerve of the forearm and also from the dorsal branch of the ulnar nerve. Unfortunately in patients with involvement of these nerves the subjective sensations may be absent and thus make it impossible to determine their position.

SUMMARY

A rapid and simple method for recording conduction rate in the distal portion of the sensory division of the radial nerve is described. The findings in control subjects and in patients with neuropathies

are presented. Since those patients with a radial nerve palsy who had normal conduction rates recovered clinically, this test may prove to be of prognostic value.

Abnormal findings may help to select a suitable nerve in patients in whom nerve biopsy is contemplated and may allow interesting correlations between histological abnormalities and abnormalities on the electrical tests. The application of these techniques to other cutaneous nerves is discussed.

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