

## A CLINICAL AND ELECTRICAL STUDY OF ULNAR NERVE LESIONS IN THE HAND

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Compression of the ulnar nerve at the wrist or in the palm gives rise to a distinctive clinical syndrome of weakness of the small muscles of the hand with or without associated sensory loss (Hunt, 1908; Harris, 1929; Russell and Whitty, 1947; Bakke and Wolff, 1948). Frequently there is a history of trauma which may be occupational and thus often repeated, although in other cases the onset appears to follow a single injury to the wrist or the hand.

In the three-year period ending in November, 1958, nine patients with ulnar nerve lesions in the hand were referred to the Department of Applied Electrophysiology, and we propose to discuss the clinical and electrical findings in this series of patients. A series of normal values for ulnar nerve conduction in the hand has also been obtained and will be described.

The ulnar nerve enters the hand superficial to the flexor retinaculum on the lateral side of the pisiform bone; more distally it overlies the piso-hamate ligament and occupies a groove on the medial aspect of the hook of the hamate. It divides into a superficial cutaneous and a deep muscular branch, the latter passing deeply through the hypothenar muscles, which it supplies, to gain the palm where it innervates the interossei, the medial two lumbrical muscles, and the adductor pollicis.

From clinical and anatomical considerations it is possible to divide ulnar nerve lesions in the hand into three groups according to the clinical signs and the presumed site of the lesion. The first and largest group is characterized by weakness of the hand muscles supplied by the ulnar nerve with the exception of the hypothenar group; there is no sensory loss. This distribution of muscle weakness will arise from a lesion involving the deep palmar branch of the nerve after the branches of the hypothenar muscles have been given off. Russell and Whitty (1947) reported four such cases. A second group of cases has been described in which all the intrinsic hand muscles supplied by the ulnar nerve were

affected, but again without sensory loss. This pattern indicates a more proximal lesion of the deep branch before the hypothenar muscles have been supplied. The original cases of Ramsay Hunt (1908) fall into this category. Finally, there are those cases in which all the intrinsic hand muscles supplied by the ulnar nerve are involved and there is coexistent sensory loss. This represents a lesion of the ulnar nerve trunk or of its two branches shortly after the trunk has divided.

In recent years, writers have tended to emphasize the importance of ganglionic compression of the ulnar nerve in the hand. Such cases afford an opportunity for an anatomical correlation with the clinical findings and, in general, they lend support to the above subdivisions. Seddon (1952) reported four cases of ganglia arising from the palmar surface of the carpus with compression of the deep palmar branch of the nerve. In all cases the hypothenar muscles were spared and sensation was intact. Compression of the main ulnar trunk at the level of the pisiform bone by a ganglion has also been described (Brooks, 1952) with involvement of all the ulnar-innervated hand muscles and accompanied by sensory loss; in the two cases reported by Mallett and Zilkha (1955) sensory loss was not present.

### Case Histories

**Case 1.**—Mr. O. J. (E.M.G. No. 26970), a 66-year-old salesman, first noticed difficulty while playing bowls in September, 1956, when he found that the bowl tended to slip from his finger tips. Thereafter he preferred to hold his pen between the index and middle fingers as the index finger seemed particularly weak, and he commented that “the little finger used to stay out”. No pain or paraesthesiae were recalled.

Examination of the right hand in January, 1957, revealed severe weakness and wasting of the interossei with sparing of the abductor digiti minimi. Sensation was intact.

Radiographs of the wrist showed an old united fracture of the lower end of the radius in good position which was thought to be irrelevant.

Within 11 weeks of his first visit there had been con-

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siderable clinical improvement and when the patient was seen at a follow-up examination in September, 1958, recovery of muscle bulk and power appeared to be complete. On further questioning he then recalled having used a turf cutter without the cross-piece to the handle when gardening before the onset of symptoms in 1956.

**Case 2.**—Miss M. B. (E.M.G. No. 31613), a 51-year-old typist, noticed severe pain in the palm of the left hand lasting three or four days during July, 1958, which was followed by weakness of the hand and later by wasting. By the time she was first seen two months later, some improvement had occurred but she was only just able to adduct the little finger to the side of the ring finger. Paraesthesiae were not recalled. Although no definite history of trauma was obtained, the patient stated that her typewriter carriage was heavier than most and that it was stiff enough to require considerable effort to push it across. She did this with the palm of her left hand.

Examination in September, 1958, revealed moderate wasting of the first dorsal interosseous and flexor pollicis brevis muscles with weakness of all the interossei and adductor pollicis; abduction of the little finger was normal as was the flexor digiti minimi. Sensation was normal on the ulnar fingers.

Gradual improvement ensued and, when last seen, in December, 1958, the patient had no symptoms. Examination then revealed only slight wasting and weakness of the interossei.

**Case 3.**—Miss G. J. (E.M.G. No. 32216), a 62-year-old secretary, suddenly noticed in February, 1958, that she was unable to type or sew properly with her right hand. She was not aware of any further worsening after the onset of the weakness. On being questioned about antecedent trauma, she admitted that she had sawed wood for three-quarters of an hour every Saturday and Sunday during a cold spell before the onset of her symptoms.

Examination in June, 1958, revealed gross weakness and wasting of the interossei; the adductor pollicis was also noted to be severely affected but the abductor digiti minimi was normal and there was no sensory loss.

Steady improvement was observed at subsequent attendances with recovery of power and muscle bulk. When seen in November, 1958, there was only slight wasting in the first interosseous space and weakness was much less marked. By February, 1959, muscle bulk was normal and power full, save for slight relative weakness of the first dorsal interosseous muscle on the right compared with the left.

**Case 4.**—Mr. N. D. (E.M.G. No. 27974), a 37-year-old solicitor, first noticed in 1955 that when using his left hand he developed a sensation of pins and needles in front of the wrist associated with a feeling of tiredness in that hand. Over the next few months weakness gradually developed which mainly affected the grip between the thumb and index finger. Sensory symptoms soon subsided, but when the patient was first seen in May, 1957, motor weakness had been present for 18 months without appreciable change over the previous

six months. At no time had there been any local swelling, and we were unable to elicit any history of trauma to the hand or wrist.

Examination in May, 1957, revealed a relatively mild disability with maximum weakness and wasting of the first dorsal interosseous muscle and lesser involvement of the other interossei and adductor pollicis. The abductor digiti minimi and the other hypothenar muscles were normal, as were the thenar muscles. There was no sensory loss.

Gradual recovery took place and re-examination 17 months later showed complete recovery of function with barely detectable thinning of the first dorsal interosseous muscle.

**Case 5.**—Mr. J. A. (E.M.G. No. 26379), a 58-year-old machine tool fitter, presented in October, 1956, with a history of having suddenly discovered weakness of his left hand two weeks previously. He stated that wasting was evident within four days of the time at which he became aware of weakness. The patient's work involved the use of a special scraping tool and from his description it was clear that he suffered repeated local pressure on the palm of the left hand. He denied pain or numbness.

Examination at that time showed weakness and wasting of the ulnar-supplied intrinsic muscles with the exception of the hypothenar group. Sensation was not disturbed.

In December, 1956, the left hand was explored surgically by Mr. D. M. Brooks at the Royal National Orthopaedic Hospital, and the deep branch of the ulnar nerve exposed, but no abnormality was detected. Post-operative examinations at the Royal National Orthopaedic Hospital showed rapid improvement, clinical recovery being almost complete within two months of operation.

**Case 6.**—Mrs. A. J. (E.M.G. No. 25178), a 32-year-old housewife and part-time typist, first noticed her fingers becoming weak in December, 1955. In particular, fine movements and typing were difficult. There was no pain or sensory disturbance. The patient stated that one month previously she had slipped and fallen onto her left hand.

Examination two months after the onset revealed weakness of the ulnar-innervated intrinsic muscles of the hand with the exception of the hypothenar group. Sensation was intact. These findings were unaltered when the patient was referred for electromyography in March, 1956.

Because of failure to improve over the next seven months, the hand was explored surgically by Mr. D. M. Brooks at the Royal National Orthopaedic Hospital in October, 1956. A synovioma compressing the deep palmar branch was found and was successfully removed. Although considerable post-operative improvement occurred, mild weakness and wasting were still present at the time of the most recent examination in October, 1958.

**Case 7.**—Mr. R. T. (E.M.G. No. 27462), a 42-year-old sheet metal worker, was first seen in February, 1957. About two years previously his foreman had pointed out

to him a depression in the first interosseous space of the right hand. The patient, who used a hammer and a pair of shears regularly in the right hand, had not noticed wasting himself and was not aware of any weakness at that time. About 18 months later, however, he was transferred to a slightly heavier but similar job, and within a day he noticed weakness of the right hand with a dull ache about the base of the thumb and wrist joint. There was also some aching in the biceps and along the lateral aspect of the right forearm.

Examination in February, 1957, revealed moderately severe weakness and wasting of the first and second dorsal interosseous muscles as well as weakness of the adductor pollicis. The remaining interossei and the hypothenar muscles appeared to be spared and no sensory loss could be detected.

As the patient's disability interfered with his work, surgical exploration was advised and was performed by Mr. H. J. Seddon at the Royal National Orthopaedic Hospital in May, 1957. At operation an unusually thickened piso-hamate ligament was noted and the hook of the hamate seemed to overhang the deep branch of the ulnar nerve. The hook of the hamate was removed in order to expose the nerve which was seen to be slightly swollen as it ran under it. When seen again in September, 1958, the patient was pleased with his progress and although some wasting and weakness were still present he was able to manage his job.

In each of the seven cases described above the hypothenar muscles were spared and sensation was unaffected. We therefore presume that in this group the deep palmar branch of the ulnar nerve was affected after the origin of the branches to the hypothenar muscles. From six patients some history of trauma was obtained; in five of them trauma was often repeated and was not a single accident or injury. The onset was sometimes stated to be sudden but in two cases weakness developed over a period of several weeks or months. Four patients were treated conservatively and all of them made a satisfactory clinical recovery. In three patients the hand was explored surgically owing to failure to improve while they were under observation, and in two of them, a local lesion was found.

**Case 8.**—Mr. H. M. (E.M.G. No. 30893), a 40-year-old salesman, was first seen in June, 1958, with an eight-week history of numbness in the tip of the little finger. Three weeks after the onset of symptoms he developed pain in the base of the hypothenar eminence where he noticed a firm swelling. Pressure on the swelling evoked paraesthesiae in the little finger. The numbness gradually spread to involve the tip of the ring finger and the inner side of the hypothenar eminence. Weakness of the index finger then followed, associated with wasting in the first dorsal interosseous space and "flabbiness" of the hypothenar muscles. No occupational hazard could be elicited, but for several years the patient had tended to sit on the floor in a tripod posture taking his weight behind him on the palmar aspects of his wrists.

Examination in June, 1958, revealed weakness and wasting of the ulnar-innervated intrinsic muscles, including the hypothenar group, although the latter were not as severely affected as the interossei.

Discrimination of compass points was impaired over the ulnar one and a half fingers and, although the swelling previously noticed by the patient was no longer palpable, pressure on the radial side of the pisiform bone still evoked paraesthesiae.

Three months later there was still severe weakness of the first dorsal interosseous muscle although wasting was less marked. The other interossei and the adductor pollicis were definitely stronger than before and power in the hypothenar muscles was almost normal. Two-point discrimination was normal but the patient still noticed numbness of the inner side of the ring finger when it was stroked. Palpation of the wrist was without effect. After a further 15 weeks, full muscle bulk had been recovered and there was only slight weakness of the first dorsal interosseous muscle; no subjective or objective sensory disturbance remained.

**Case 9.**—Mrs. N. D. (E.M.G. No. 23975), a 52-year-old housekeeper, had noticed occasional local discomfort in the base of the hypothenar pad for several years, particularly when pushing the handle of her vacuum cleaner, but it was not until April, 1955, that a constant ache developed in the right wrist with weakness of the hand. This weakness made it increasingly difficult for her to manipulate objects with the fingers and she experienced difficulty in picking flowers, using a flint gaslighter, and in applying the brake of her bicycle. Some aching pain was also felt in the upper arm, forearm, and thumb.

Examination in November, 1955, revealed clawing of the ring and little fingers, wasting and severe weakness of the interossei, weakness of the adductor pollicis, and lesser weakness of the hypothenar muscles. The only constant objective sensory finding was reduction of cutaneous sensation over the palmar aspect of the little finger. A small cystic swelling was palpable in front of the wrist.

The right hand was explored by Mr. Kenneth Paine at the National Hospital, Queen Square, in November, 1955, and a ganglion was found lying on the radial side of the main ulnar nerve trunk compressing it against the pisiform bone. The ganglion was excised and clinical improvement was already apparent by the time of the patient's discharge from hospital. Re-examination in September, 1958, revealed no wasting and only slight weakness of the first dorsal interosseous muscle. The adductor pollicis was normal and there was barely detectable weakness of the abductor digiti minimi. No sensory loss was present and the swelling at the wrist had not recurred.

In the two cases described above, there was mild but definite involvement of the hypothenar muscles with slight sensory disturbance. These features suggested a lesion of the main ulnar nerve trunk at the level of the pisiform bone and in both patients a swelling was present in this region. One swelling

was shown at operation to be a ganglion, but the other subsided before the patient was first seen; it was presumed to be a ganglion which had retrogressed spontaneously. In both patients satisfactory clinical recovery followed.

#### Methods Used in Electrical Investigations

The electrical investigations followed the methods described in previous communications from this Department by Simpson (1956), Gilliatt and Sears (1958), and Thomas, Sears, and Gilliatt (1959). After placing coaxial needle electrodes in one or more of the small hand muscles, the ulnar nerve trunk in the arm was stimulated through surface electrodes and the latency of the earliest phase of the muscle response recorded (Fig. 1). The stimulus was a brief condenser discharge (time constant 150  $\mu$ sec.) delivered through an isolating transformer. The stimulating cathode S1 was placed over the ulnar nerve approximately 2 cm. above the distal wrist crease and S2 3 to 6 cm. above the tip of the medial epicondyle. A third stimulus, S3, was applied in the axilla. The surface distance between the stimulating cathode S1 and the recording needle electrode R1 in the abductor digiti minimi varied from 5.5 to 8.5 cm. in different subjects. It was not possible to estimate accurately the length of the deep branch of the ulnar nerve to the interossei by surface measurement. The distance between S1 and S2 varied from 23 to 30 cm. in different patients and the distance between S2 and S3 from 12 to 22 cm. The anode was a plate electrode, 2.5  $\times$  5 cm., placed over the flexor surface of the forearm or over the deltoid insertion; these positions were chosen for convenience and did not affect nerve conduction times.

For recording, a double-beam oscilloscope was used so that when conduction was to be compared in ulnar nerve fibres supplying two different muscles, it was possible to display simultaneously the action potentials of both muscles after a single stimulus to the nerve trunk. Care was taken when positioning the needle within a muscle to ensure that the initial deflection of the muscle action potential was as sharp as possible, thereby aiding the measurement of the time interval. In each case the stimulus used was such that a further increase failed to shorten the latency or to increase the amplitude of the evoked muscle action potential. When a constant appearance of the action potential was obtained in response to nerve stimulation at different levels in the

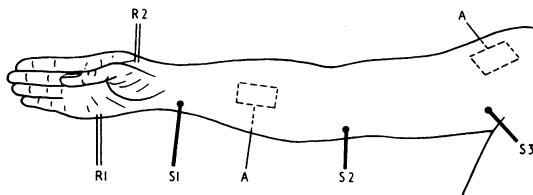


FIG. 1.—Arrangement of stimulating and recording electrodes for measurement of motor nerve conduction time. For explanation, see text.

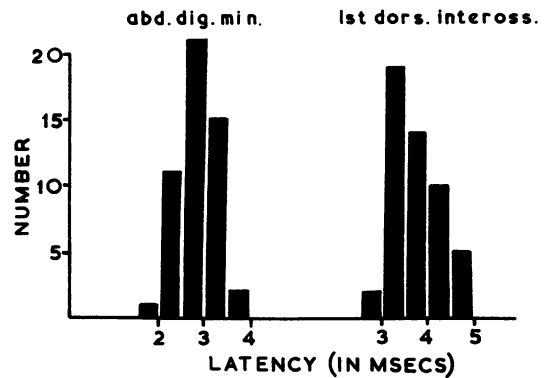


FIG. 2.—Distribution of the latency of the muscle response (in milliseconds) for the abductor digiti minimi and the first dorsal interosseus muscle on stimulation of the ulnar nerve at the wrist in 50 normal subjects.

arm, latencies were measured to the initial deflection of the action potential in each case. By subtraction the conduction time was obtained for the different segments of nerve examined, *i.e.*, axilla-elbow, elbow-wrist, and wrist-muscle. Although several motor units in the vicinity of the recording needle were usually activated by each shock, the conduction times must necessarily refer to the fastest nerve fibres stimulated.

In a few cases, afferent volleys in the digital nerve fibres from the fifth finger were examined, using ring stimulating electrodes around the finger and surface recording electrodes over the ulnar nerve trunk at the wrist as described by Gilliatt and Sears (1958).

#### Results of Electrical Investigations

**Motor Nerve Conduction in the Hand.**—During the course of the investigation 50 control observations were made on healthy nerves to determine the normal latency of the muscle action potentials in the hand after a single supramaximal shock to the ulnar nerve trunk at the wrist. The abductor digiti minimi and the first dorsal interosseus muscle were examined and the results obtained are shown in Fig. 2. For the abductor digiti minimi the values ranged from 2.0 to 3.7 msec. with a mean of  $2.9 \pm 0.39$  msec.; for the first dorsal interosseus the corresponding range was 3.0 to 5.0 msec. with a mean of  $3.8 \pm 0.53$  msec.

Latencies for the abductor digiti minimi and the interossei in the nine patients with ulnar nerve lesions in the hand are shown in Fig. 3 and Table I. In Fig. 3 values for the first dorsal interosseus muscle are shown except for Case 3; in this patient no electrical response to nerve stimulation was obtained from the first dorsal interosseus and latency was measured to the third dorsal interosseus muscle. In Case 8 the first examination was in-

TABLE I  
MOTOR NERVE CONDUCTION TIMES AT INITIAL (a) AND SUBSEQUENT EXAMINATIONS (b, c, and d)

Case	Muscle	Conduction Times (msec.)									Time Interval (in weeks)			
		Wrist-Muscle				Elbow-Wrist			Axilla-Elbow		a-b	b-c	c-d	
		a	b	c	d	a	b	c	a	b				c
1	A.D.M. 1st D.I.	2.5 16.0	2.5 9.0	2.5 5.3		5.3 6.0	5.5 5.5	4.9 5.0		3.0 3.9	2.6 2.5	11	72	
2	A.D.M. 4th D.I.	2.1 16.0	2.0 6.5	3.0 7.0		4.4 6.0	4.2 5.2	4.2	1.7	3.0 3.2		6	6	
3	A.D.M. 3rd D.I.	3.2 29.0	2.5 6.0	2.2 6.0	2.2 5.1	4.0 5.0	4.5 5.6	4.3	3.0 4.0	3.0 3.2		6	8	13
4	1st D.I. A.D.M.	3.0 9.0	15.8 2.7	8.8	6.0	5.5 6.5	5.7 5.7		3.1 4.1	3.3 3.6		68		
5	1st D.I. A.D.M.	2.3 10.0	5.1											
6	A.D.M. 1st D.I.	2.6 17.5	3.0 5.4			4.7 6.5	5.6 5.7					120		
7	A.D.M. 3rd D.I.	2.6 3.8	3.6			5.6 5.3	6.4			3.0		72		
8	1st D.I. A.D.M.	4.2 6.2	5.0 3.5			5.3	6.1 4.6		4.0	3.1 2.2		13	15	
9	4th D.I. 1st D.I. A.D.M. 1st D.I.	4.4 20.5	2.9 4.4	5.5		3.9 6.5	5.3 5.4			2.8 3.2		136		

\* Inexcitable.

complete and the results of the second examination are shown in Fig. 3.

It is clear from Fig. 3 that the latency of the evoked action potentials in the interosseous muscles was markedly increased in nearly every case, values of up to 29 msec. being obtained for the conduction time in the short segment of nerve between the wrist and the affected muscle. These results confirm the findings in the single case described by Simpson (1956).

In the seven patients with sparing of the hypothenar muscles (Cases 1-7) the latencies for the abductor digiti minimi were normal. A representative record from this group is shown in Fig. 4. In this instance the ulnar nerve was stimulated at the wrist and the muscle action potentials were recorded from the abductor digiti minimi and the fourth and first dorsal interossei. The brief latency between the stimulus artefact and the beginning of the action potential in the abductor digiti minimi (2.1 msec.) may be contrasted with the long delay to the fourth and first dorsal interossei (16.0 and 27.5 msec. respectively). Such a result establishes the presence of nerve damage proximal to the branch to the fourth dorsal interosseous but after the origin of the branches to the abductor digiti minimi. Fig. 4 also shows the prolonged polyphasic potentials evoked in the affected muscle which may be compared with the normal contour of the muscle action potential in the abductor digiti minimi. A prolonged polyphasic response of the type shown was commonly seen in the affected muscles of our patients and in one case the potentials lasted for 45 msec.

In only one patient (Case 7) was the latency of the muscle response in the first dorsal interosseous muscle within the normal range. In this patient the initial part of the action potential was of normal form but it was followed by a prolonged polyphasic discharge, similar in appearance to those recorded

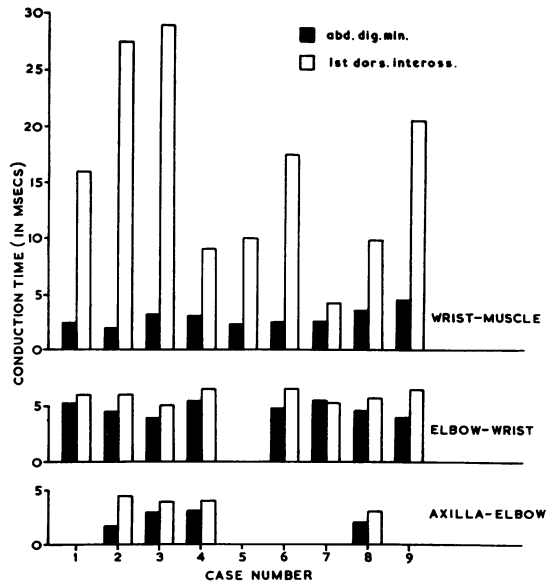


FIG. 3.—Conduction times (in milliseconds) for motor nerve fibres to the abductor digiti minimi and the first dorsal interosseous muscle in patients with ulnar nerve lesions in the hand. (In Case 3, the first dorsal interosseous was inexcitable and conduction times to the third dorsal interossei are shown.)

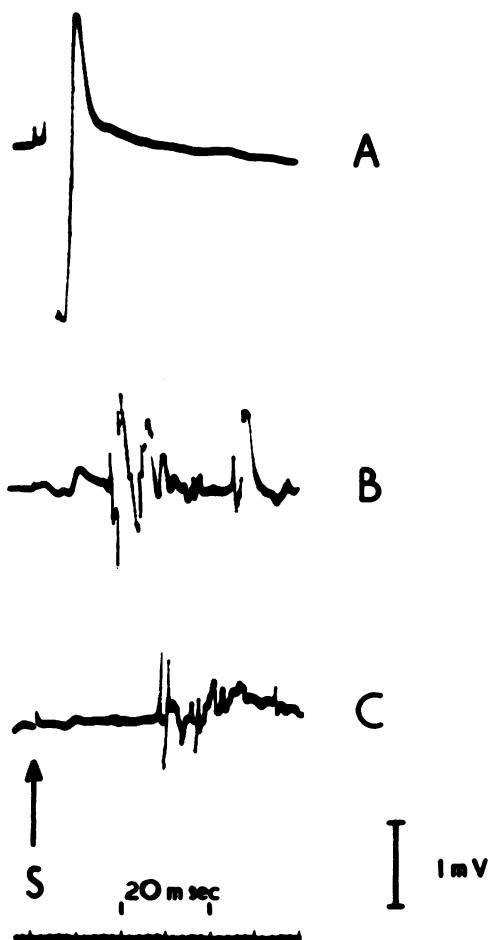


FIG. 4.—Recordings from the abductor digiti minimi (A), the fourth dorsal interosseus (B), and the first dorsal interosseus (C) on stimulation of the ulnar nerve at the wrist in Case 2. Stimulus at S shown by artefact in each trace. The latency of the muscle response is normal for the abductor digiti minimi, but considerably lengthened for the interossei, which also show prolonged polyphasic muscle action potentials.

from the interossei in Fig. 4. This abnormal response was restricted to the interossei and was not present in the abductor digiti minimi or in the abductor pollicis brevis when the median nerve was stimulated; the electrical findings were therefore assumed to support the clinical diagnosis of compression of the deep branch of the ulnar nerve in its distal part.

In one of the two patients in whom the hypothenar muscles were involved (Case 9), the latency of the muscle response in the abductor digiti minimi was slightly increased, whereas in the other patient (Case 8) the latency was at the upper limit of normal.

It is interesting to note that marked increases in latency were not seen in the hypothenar muscles in this group; this may be related to the fact that in both patients the hypothenar muscles were much less severely affected than the interossei.

**Sensory Nerve Conduction in the Hand.**—The afferent volley in sensory fibres from the fifth finger was examined in four of the patients in whom there was sparing of the hypothenar muscles and in one patient in whom the hypothenar muscles were involved. In the first group (Cases 2, 3, 4, and 6), the amplitude of the sensory nerve action potentials (Table II) was within the range of normal described by Gilliatt and Sears (1958).

TABLE II  
SENSORY ACTION POTENTIALS FROM ULNAR NERVE AT WRIST ON STIMULATION OF FIFTH FINGER

Case	Conduction Distance (cm.)	Latency to Peak (msec.)	Amplitude ( $\mu$ V)
2	10.0	2.4	12
3	10.5	2.4	9
4	13.0	2.8	14
6	12.0	3.8	15
8a†	12.5	No response	0
b	12.5	3.2	3
c	11.0	2.8	8
Normal* range		2.2-3.4	8-28

† Intervals a-b and b-c as in Table I

\* From Gilliatt and Sears (1958)

In Case 8, the hypothenar muscles were mildly involved and at the initial examination no sensory nerve action potential could be recorded from the ulnar nerve at the wrist when the fifth finger was stimulated. At that time discrimination of compass points was impaired in the ulnar-supplied fingers and the patient complained of paraesthesiae. Thirteen weeks later some clinical recovery had occurred and a small sensory action potential (amplitude  $3 \mu$ V) could be detected at the wrist with a latency to peak of 3.2 msec. At this stage the patient still complained of mild numbness when the inner side of the tip of the fourth finger was stroked but compass points were appreciated without difficulty. After a further 15 weeks, when no subjective or objective sensory disturbance was present, a potential of  $8 \mu$ V with a latency of 2.8 msec. was recorded.

**Motor Nerve Conduction above the Wrist.**—Conduction times between the elbow and the wrist, and between the axilla and the elbow, for motor nerve fibres supplying both affected and unaffected muscles are shown in Fig. 3 and Table I. It can be seen that in seven of the patients motor nerve fibres to the interossei showed slightly slower conduction above

the wrist than fibres to the abductor digiti minimi. No difference in the rate of conduction exists between the motor fibres to the abductor digiti minimi and the first dorsal interosseous muscle in normal subjects (Thomas *et al.*, 1959) and although the differences in conduction time shown in Fig. 3 are small they seem to show a uniform trend. Errors such as uncontrolled variation in temperature and possible current spread to portions of nerve not immediately beneath the stimulating cathode cannot be relevant here as both affected and unaffected fibres could be stimulated simultaneously by the same shock. In Fig. 5, for example, a recording needle electrode was placed in the third dorsal interosseous muscle and action potentials from this muscle and from the hypothenar muscles appeared on the same trace, enabling a direct comparison of latency to be made. The ulnar nerve trunk was stimulated at the wrist, elbow, and axillary levels, latencies for the onset of the response in the hypothenar muscles being 3.2, 7.2, and 10.2 msec., and 29.0, 34.0, and 38.0 msec, for the affected interosseous muscle. The difference in conduction time in the proximal parts of the two motor nerves is thus in the order of 20%.

TABLE III  
CONDUCTION VELOCITY IN FOREARM

Case	Abductor Digiti Minimi (metres per sec.)	First Dorsal Interosseous (metres per sec.)
1	51	45
2	53	39
3*	62	47
4	50	42
5	—	—
6	50	36
7	50	53
8	58	46
9	59	35
Mean	54.1	42.9
Normal subjects**		
Mean	56.2	55.0
Range	49.0 - 65.6	46.2 - 66.2

\* Third dorsal interosseous examined.

\*\* From Thomas *et al* (1959).

The distance between the electrodes S1 and S2 in Fig. 1 was recorded in each case, and maximal conduction velocities for the fibres concerned have been calculated: they are shown in Table III together with the normal range reported by Thomas *et al.* (1959). From this table it is clear that the values for the abductor digiti minimi are all within the normal range and are grouped around the mean whereas several of the values for the interossei are below the normal range. The difference between the mean values for the interossei for the normal subjects and for the patients is statistically significant when compared by the t test ( $P \approx 0.05$ ).

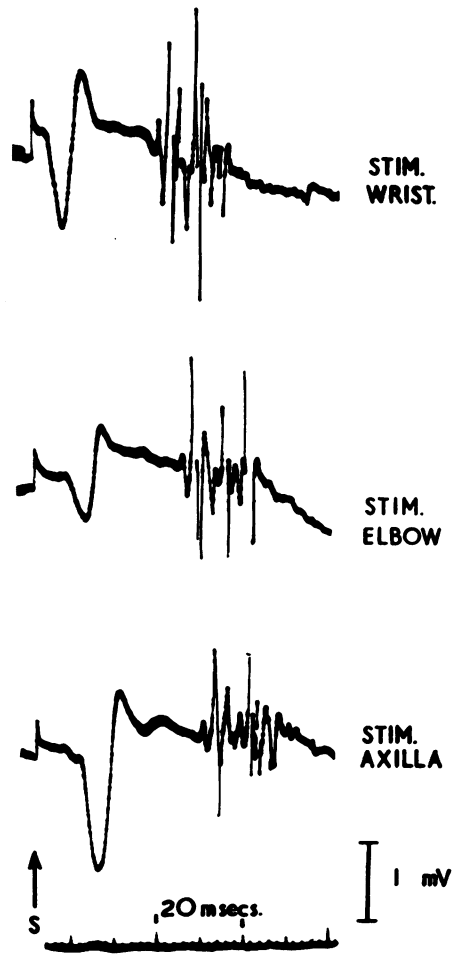


FIG. 5.—Recordings from Case 3. The recording needle was inserted into the third dorsal interosseous muscle, but the earlier response of the hypothenar muscles also appears in each trace. The ulnar nerve was stimulated at the wrist, elbow, and axilla. Stimulus at S shown by artefact in each trace.

**Follow-up Studies.**—All patients were followed up and re-examined by us except for Case 5; this patient was seen post-operatively at the Royal National Orthopaedic Hospital. Serial measurements of motor conduction time below the wrist are shown graphically in Fig. 6 for the five patients who made a spontaneous recovery. Cases 1 and 4 were re-examined electrically after intervals of 18 and 17 months respectively, by which time clinical recovery was virtually complete and latencies below the wrist were only slightly in excess of normal (5.3 and 5.1 msec. respectively). Cases 2 and 3 were re-examined after six weeks and motor nerve conduction was found to be markedly improved

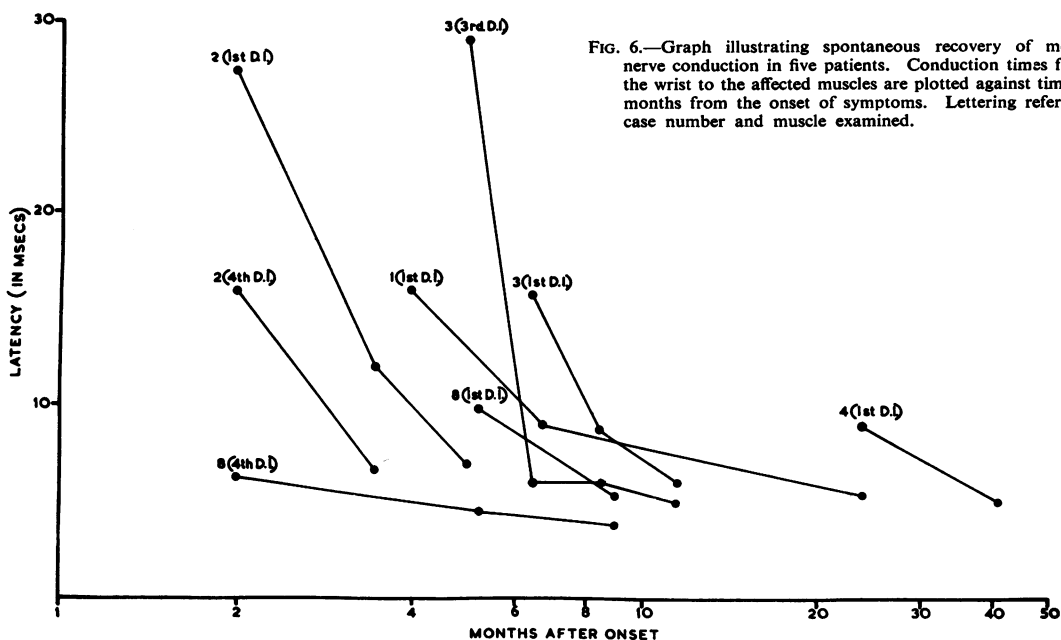


FIG. 6.—Graph illustrating spontaneous recovery of motor nerve conduction in five patients. Conduction times from the wrist to the affected muscles are plotted against time in months from the onset of symptoms. Lettering refers to case number and muscle examined.

although clinical recovery at that stage was slight. This illustrates the value of serial measurements of conduction time in detecting early recovery. In Case 8, the initial abnormality was less marked but clinical improvement was again accompanied by a fall in latency. Values for the whole group, including the operated cases, are contained in Table I, which also shows that, above the wrist, differences in conduction time between affected and unaffected fibres decreased or disappeared during the recovery period.

### Discussion

A history of trauma was obtained in seven of the nine patients, although it was difficult to be certain that this was relevant in every case, *e.g.*, Case 6. In some, however, the story of repeated trauma, either occupational or domestic, seems sufficiently definite to justify describing them as cases of occupational or traumatic neuritis. Most of our cases showed selective damage to the deep branch of the ulnar nerve after the origin of the branches to the hypothenar muscles; even when the main ulnar trunk was affected, weakness of the hypothenar group was mild compared with the other ulnar-supplied muscles. This difference was also reflected in the electrical findings which showed maximal damage to the nerve supply to the interossei. In the two patients in whom the hypothenar muscles were involved, sensation in the fifth finger was mildly

disturbed and we have not met with an example of the syndrome described by Ramsay Hunt (1908) and by Mallett and Zilkha (1955), in which all the ulnar-supplied muscles in the hand were weak but with normal sensation.

Our follow-up examinations were less complete than we would have liked but several interesting facts emerged. Spontaneous improvement without operation occurred in five patients and in three of them final recovery was virtually complete. In the other two steady improvement was occurring at the time the patients were last seen. In one patient improvement was apparent within two months of the onset, but in three others five to seven months elapsed before this was noted. In the fifth patient weakness of the hand had been present for at least 18 months before he was first seen but he also made a satisfactory recovery. In one of the four operated cases no abnormality was found, yet the patient made a full recovery. In each of the other three cases some local abnormality was found at operation. In one a ganglion was present to the radial side of the pisiform bone, in another a synovium was present in the palm, while in the third there was thickening of the pisohamate ligament. It might be thought that the presence of a palpable swelling in front of the wrist should be a clear indication for operation, but in one of our patients, such a swelling subsided spontaneously with subsequent recovery of power in the hand. A ganglion shrinking



spontaneously has been described previously in one patient by Seddon (1952).

Our electrical studies have confirmed Simpson's (1956) finding that a patient with damage to the ulnar nerve in the hand may show profound slowing of nerve conduction over the affected segment. In some of our cases the time taken for a volley to pass from the wrist to the interossei was increased to five or six times the normal figure and some increase in latency was seen in all but one case.

In addition to an increased latency most of our patients showed an abnormal muscle response to nerve stimulation, the muscle action potentials being of low voltage, polyphasic in form, and greatly prolonged in duration. This prolonged response may be due to temporal dispersion of impulses in different nerve fibres but repetitive firing of single motor units could also contribute to it.

Not the least interesting of the findings in this study has been the observation that nerve conduction may be slowed proximal to a local lesion. Two possible explanations suggest themselves. First, it may be that the lesion selectively damages the larger and more rapidly conducting fibres; since recording is made distal to the lesion, conduction will therefore only be measured in the surviving slower fibres which pass through the lesion. Alternatively, it is possible that changes occur proximal to the site of damage in the nerve fibres involved in the lesion (Cragg and Thomas, to be published). This question is being further investigated.\*

### Summary

In a three-year period nine patients with ulnar nerve lesions in the hand were seen. A history of trauma was obtained in seven of them.

There were seven patients with sparing of the hypothenar muscles and of sensation but with

wasting and weakness of the other ulnar-supplied muscles in the hand; in this group, four patients made good recoveries without operation and a fifth patient, in whom exploration was negative, also recovered. In two patients of this group a local compressive lesion was found at operation.

In two of our nine patients the hypothenar group was mildly involved in addition to there being severe weakness of the other ulnar-innervated intrinsic muscles. Some sensory disturbance was present in both. One of these patients recovered spontaneously whereas a ganglion compressing the main ulnar trunk was excised in the other.

Ulnar nerve function was examined electrically in all cases and motor conduction was profoundly slowed in the majority. Gross abnormalities of conduction were confined to the segment of nerve below the wrist but mild changes were also seen in the proximal parts of the affected nerves.

Measurement of motor nerve conduction time provided a quantitative estimate of the severity of nerve damage; serial measurements proved particularly helpful in the detection of early recovery.

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\*Since submitting this paper for publication a report has appeared (Krnjeric and Kiraly, 1959) in which slowing of sleeve conduction proximal to peripheral nerve section in the experimental material was described.