# Section of Physical Medicine

*President* A T Richardson MRCP

*Meeting December 13 1961*

## Short Papers

### The Carpal Tunnel Syndrome: Investigation and Assessment of Treatment

by E D R Campbell MA BM MRCP
(*Royal Free Hospital, London*)

Pain and paræsthesiæ in the fingers may arise from several causes and, as many of these can be helped by specific treatment, the importance of precise diagnosis is evident. The commoner causes of acroparæsthesiæ are nerve compression syndromes (cervical spondylosis, thoracic inlet syndrome, carpal tunnel syndrome) and degenerative or inflammatory lesions (polyneuritis, subacute combined degeneration and, occasionally, disseminated sclerosis). In the latter group there are usually associated symptoms and signs elsewhere and therefore only the nerve compression syndromes will be considered further.

Where the symptoms affect only the fourth and fifth fingers, clinical differentiation between a lesion of the ulnar nerve and one of the eighth cervical nerve root is not difficult: for the sensory supply of the ulnar nerve is limited strictly to the hand below the wrist creases, whereas an irritative lesion affecting nerve root C.8 will cause symptoms in the same hand distribution as the ulnar nerve, but also extending some way up the forearm. The same type of differentiation, however, cannot be made between irritative lesions of the median nerve and of nerve roots C.6 and 7, especially as many patients have difficulty in localizing paræsthesiæ with accuracy, tending to incriminate the whole hand when more than two fingers are involved.

Obviously a clear history of cervical spondylosis need not be confused with a typical carpal tunnel syndrome, but early or minimal signs of either may be difficult to distinguish. As acroparæsthesiæ is most common in the middle and older age groups, the presence of radiological signs of cervical spondylosis cannot be taken to be a decisive proof of the cause, for the occurrence of symptomless cervical spondylosis is well known in this age group. Furthermore, as Lishman & Russell (1961) have shown, the two conditions may co-exist.

It has been well established that measurements of conduction time in both motor (Simpson 1956, Thomas 1960) and sensory (Dawson & Scott 1949, Gilliatt & Sears 1958) elements of the median nerve as it passes through the carpal tunnel show slowing of transmission in carpal tunnel compression. The final proof of this was provided by Goodman & Gilliatt (1961) who showed the return to normal conduction times after operative decompression of the carpal tunnel. In all these papers, the tests were done on patients in whom the clinical diagnosis of carpal tunnel syndrome appeared clear, though Thomas (1960) does state that some of his series with normal findings may represent misdiagnoses: the present study shows that in a wide group of patients with acroparæsthesiæ, those whose symptoms are due to carpal tunnel syndrome are accurately differentiated.

*Method*

Motor and sensory nerve conduction times of the ulnar and median nerves of both hands were measured in all patients on their first attendance, thereafter only those nerves giving abnormal readings were re-tested. The patients waited and were tested in a warm room and did not uncover their arms until immediately before testing.

For measurement of motor conduction time the nerve was stimulated 1–2 cm above the distal wrist crease, the evoked muscle action potentials being collected by twin surface electrodes over the thenar or hypothenar muscle groups. The distance from the cathode of the stimulator to the nearest collecting electrode varied from 4 to 6 cm, and the inter-electrode distance of the surface electrodes was 2·5 cm.

Sensory conduction was measured by stimulating the nerve at the same site and collecting the

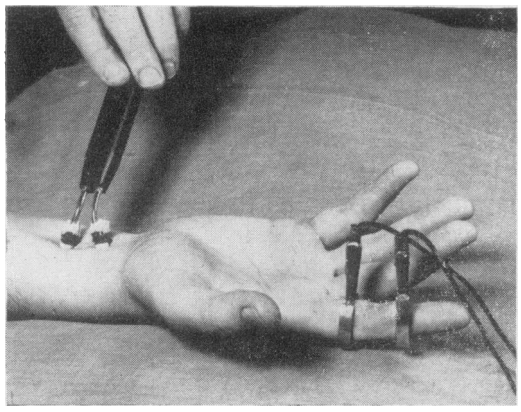

Fig 1 *Arrangement of stimulating and sensory pick-up electrodes*

antidromic impulses in the digital nerves of one finger by means of a pair of silver ring-electrodes (Fig 1). The distance from the cathode to the first collecting electrode was 9–11 cm for ulnar nerve, and 11–14 cm for median nerve studies. The inter-electrode distance of the collecting electrodes varied from 2·5 to 3 cm. A large earthing pad was placed under the forearm.

The skin under the collecting electrodes was prepared by a brisk rub with spirit followed by the application of Cambridge electrode paste. The inter-electrode resistance was 5,000–7,000 ohms. The stimulus used for both sets of investigations was a rectangular pulse of 0·1 msec duration delivered from an earth-free source. The usual rate of stimulation was 2 per second, though rates of up to 50 per second have also been tried without affecting latency measurements.

This method for sensory measurements is a departure from those usually described for diagnostic purposes though it has been used by Sears (1959) in a physiological investigation. It has the advantages of speed and increased patient comfort; similarly, the use of surface instead of needle electrodes for motor measurements makes the entire investigation one of no great discomfort, giving a degree of accuracy commensurate with the needs of a clinical test.

Initially the method of Gilliatt & Sears (1958) for sensory measurements was also used on the same subjects (patients showing abnormal readings, and normal controls) as the method just described, and latency times were entirely similar in all cases. Small variations up to 5 microvolts (μV) in the amplitude of the evoked potential occurred occasionally, the present method giving the larger amplitude.

Muscle action potentials conducted through the tissues to the sensory collecting electrodes were never a source of confusion. Dawson & Scott (1949) showed that sensory fibres are more excitable than motor fibres (*see* their Fig 5) so that sensory potentials may be obtained before the threshold for motor excitability has been reached. Even when such conducted potentials from muscle do appear, however, there has always been sufficient time separation to make distinction quite clear: the much greater amplitude and time duration of the conducted muscle action potentials further diminish the possibility of confusion (Fig 2).

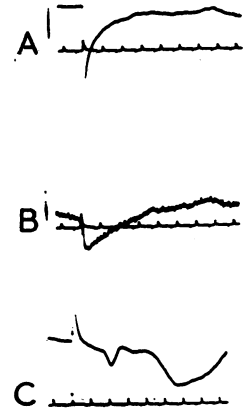


Fig 2 A, *delayed (6 msec) and diminished sensory potential.* B, *same as above without high frequency filter.* C, *normal sensory potential followed by muscle action potential conducted through tissues. Calibration as sensory series in Fig 4*

The collecting electrodes were connected to a Medelec electromyograph with special additions for latency measurements. The electrical characteristics of this apparatus are a flat frequency response from 3 to 5,000 cycles per second with high input impedance and high gain (max. 20 μV per cm deflection). A high frequency filter (3 dB down at 1 kc/s) can be switched into the circuit to exclude unwanted background noise (Fig 2). The lower beam of the oscilloscope carries a time scale marked at 1 msec intervals. The stimulus is applied at a constant marked point on the sweep. A variable electronic marker can be moved along the lower trace until its peak corresponds exactly with the initial deflection of the evoked potential. The latency can then be read off directly on decatron counters, saving the necessity of photographic records for accurate measurement (Fig 3). In a straightforward case sensory and motor measurements on four nerves can be made in six or eight minutes with the result immediately available.

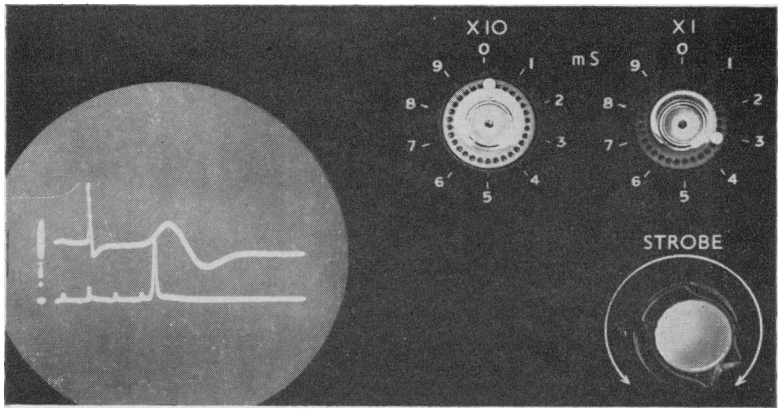


Fig 3 *Illustration of method of latency measurements. The movable pointer on the lower trace indicates the initial deflection of the sensory potential (upper trace). The position of this pointer is controlled by the knob marked Strobe. The decatrons then automatically indicate the latency (in this case 3 msec)*

Measurements of latency were made from the beginning of the stimulus artifact to the initial deflection of both the sensory and motor potentials evoked. A latency of 4 msec or greater was regarded as abnormal for sensory nerve measurements, the length of nerve traversed not exceeding 14 cm, and a latency of 5 msec or greater for motor nerve conduction the distance not exceeding 6 cm. The apparent great difference in conduction rate is accounted for by slowing in the terminal motor nerve fibres, delay at the neuromuscular junction and the time taken for propagation and spread of the muscle fibre action potential. Figs 4 & 5 show typical examples of normal and abnormal sensory and motor latency measurements.

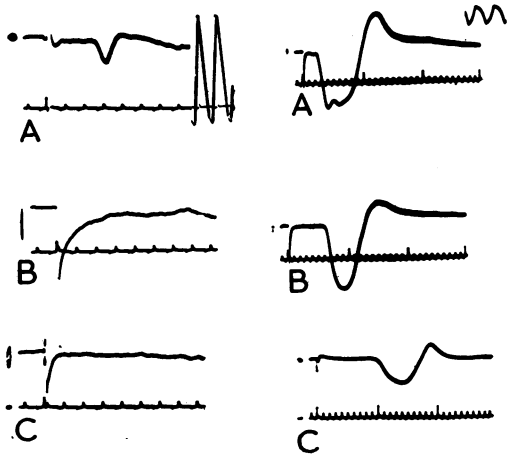


Fig 4




Fig 5

Fig 4 *Sensory potentials showing* (A) *normal,* (B) *diminished and delayed, and* (C) *unrecordable latencies. Calibration 100 μV. Time scale in msec*

Fig 5 *Muscle action potentials showing* (A) *normal,* (B) *moderately, and* (C) *severely delayed latencies. Calibration 500 μV. Time scale in msec*

*Results*

Ninety-six patients with acroparæsthesiæ have been tested by this method and have been followed up for times varying from three to eighteen months.

Forty-one patients (33 female, 8 male) had normal latencies. Two of these, however, had such typical symptoms of carpal tunnel syndrome that they were treated as such and made full symptomatic recoveries; in neither case had symptoms been present for more than three weeks. The remaining 39 were considered to have some other cause for their symptoms and this was either proved by further clinical investigation or inferred by a satisfactory response to specific treatment. Thus 17 patients had cervical spondylosis, 10 thoracic inlet syndrome and the remainder a variety of conditions including diabetes, disseminated sclerosis, post-herpetic neuralgia, tenosynovitis, writer's cramp and mild chilblains. It is worth emphasis that in no case were the symptoms long-standing or severe. The age range of these patients was from 22 to 74; one-third were between 40 and 50, the remainder being evenly dispersed.

Fifty-five patients (46 female, 9 male) were found to have abnormalities of motor and/or sensory conduction and they have been graded into four different categories depending on the severity of the abnormalities detected (*see* Groups

## *Table 1*

**Results of treatment in different categories of carpal tunnel syndrome**

| | *Group 1a* | *Group 1b* | *Group 2* | *Group 3* | *Group 4* |
|---|---|---|---|---|---|
| Total cases | 2● | 17 | 40 | 13 | 6 |
| Sensory conduction | Normal | Delay (4 msec or more) or diminished in amplitude | Delayed or unrecordable ■ | Delayed or unrecordable | Unrecordable |
| Motor conduction | Normal | Normal (less than 5 msec) | Mild delay (5–7 msec) | Moderate delay (8–10 msec) | Severe delay (11 msec or more) |
| *Effect of treatment on sensory and motor conduction:* | | | | | |
| Return to normal | | 17 | 19 | 3 | 1 |
| Improved | | | 11 | 7 | 2 |
| Unchanged | | | 10 | 3 | 3 |
| *Symptomatic relief:* | | | | | |
| Complete | 2 | 15 | 31 | 8 | 2 |
| Partial | | 2 | 7 | 3 | 2 |
| Nil | | | 2 (no treatment) | 2 | 2 |
| *Effective treatment:* | | | | | |
| Splints | | 10 | 14 | 0 | 1 |
| Local prednisolone | 2 | 5 | 14 | 8 | 2 |
| Ultrasound | | | 1 | 1 | |
| Decompression | | 1 | 5 | 2 | |
| No treatment | | 1 | 4 | | 1 |
| Awaiting decompression | | | | 2 | 2 |

●Typical carpal tunnel symptoms
■2 cases (1 patient) in Group 2 had normal sensory findings

## *Table 2*

**Results of treatment in 78 cases of carpal tunnel syndrome**

| *Group totals* | *Motor latency (msec)* | *Symptomatic relief* | | | *Conduction change* | | | *Effective treatment* | *No. of cases* |
|---|---|---|---|---|---|---|---|---|---|
| | | *Complete* | *Partial* | *Nil* | *Normal* | *Improved* | *Unchanged* | | |
| 19 | Normal | 10 | – | – | 10 | – | – | Splints | 25 |
| 40 | 5– 7 | 14 | – | – | 7 | 5 | 2 | | |
| 13 | 8–10 | – | – | – | – | – | – | | |
| 6 | >10 | – | 1 | – | – | – | 1 | | |
| 19 | Normal | 5 | 2 | – | 7 | – | – | Local prednisolone | 35 |
| 40 | 5– 7 | 10 | 4 | – | 7 | 2 | 5 | | |
| 13 | 8–10 | 5 | 3 | 2 | 2 | 5 | 3 | | |
| 6 | >10 | 1 | 1 | 2 | 1 | 1 | 2 | | |
| 19 | Normal | 1 | – | – | 1 | – | – | Decompression | 8 (6 without other previous treatment) |
| 40 | 5– 7 | 5 | – | – | 3 | 2 | – | | |
| 13 | 8–10 | 2 | – | – | 1 | 1 | – | | |
| 6 | >10 | – | – | – | – | – | – | | |
| 19 | Normal | – | – | – | – | – | – | Ultrasound | 2 |
| 40 | 5– 7 | 1 | – | – | – | – | 1 | | |
| 13 | 8–10 | 1 | – | – | – | 1 | – | | |
| 6 | >10 | – | – | – | – | – | – | | |
| 19 | Normal | 1 | – | – | 1 | – | – | No treatment | 8 |
| 40 | 5– 7 | 1 | 3 | 2 | 2 | 2 | 2 | | |
| 13 | 8–10 | – | – | – | – | – | – | | |
| 6 | >10 | 1 | – | – | – | 1 | – | | |

*N.B.* 17 of the 19 patients with normal motor latencies had abnormal sensory latencies. The column 'Normal' under Conduction Change indicates the numbers of patients who have made a full return to normal sensory and motor latencies

1–4 in Table 1). The age range was from 26 to 75, fifty of the patients being over 40 and twenty between 50 and 60. Only one patient was pregnant. Fifteen out of 24 patients X-rayed showed changes in the cervical spine. In 21 patients both hands were affected, so that there were 76 abnormal hands which, together with the 2 cases with normal findings mentioned above, makes a total of 78 cases of carpal tunnel syndrome. Twenty-two patients were affected in the right hand only and 12 in the left.

*Treatment*

In general, patients were treated first by splinting the wrist in the neutral position at night. If this had not given marked improvement in three weeks or complete relief in six weeks, or if symptoms returned after discarding the splints, the carpal tunnel superficial to the median nerve was injected with 20–40 mg prednisolone. If three injections at intervals of ten to fourteen days failed to give satisfactory relief, decompression of the carpal tunnel was advised. Ultrasound to the carpal tunnel was tried in 2 cases. Symptomatic relief was graded into 'complete', 'partial' (where some symptoms remained but the result was acceptable to the patient), or 'nil' if the result was unacceptable.

However, many patients were referred for this investigation from other departments and from other hospitals and this routine was not carried out in every instance. For various reasons (usually domestic) 8 patients had no treatment during the course of this study.

The results of treatment on symptoms and latency measurements are shown in Tables 1 and 2. In these Tables 'effective treatment' is the one credited with giving relief.

These findings are in broad agreement with other views on the treatment of this condition. Thus splinting is shown to be an effective form of treatment in the milder cases, confirming Heathfield's (1957) view. Crow (1960) found splinting to be effective in 15 of 36 cases, a proportion similar to the present series. Kendall (1960, 1961), however, regards nocturnal splinting as virtually useless.

Prednisolone (or its analogues) has been used systemically by Lees & Liversedge (1959) with good and sustained symptomatic relief, and by local injection into the neighbourhood of the carpal tunnel by Crow. The experience of the latter, however, was that excellent immediate relief from 50 mg local hydrocortisone was followed by recurrence of symptoms up to eight months later in three-quarters of the cases. This has not been our experience so far, though 9 of the present series who have responded to prednisolone have been followed up for less than six months: however, 6 of these have shown either improvement, or return to normal, of latency measurements.

Of 9 patients affected in both hands, who were given similar treatment on each side (not decompression), 8 responded less well in the dominant hand as regards latency measurements, though 6 of these are completely symptom free on both sides. Consideration of Table 2 makes it clear that symptomatic cure does not go *pari passu* with return to normal latencies regardless of the treatment used and this finding is in accord with other series (Goodman & Gilliatt 1961). It seems equally clear from these series that decompression is the only treatment for which a virtual guarantee of symptomatic success can be given. Nevertheless, out of 45 patients with motor latencies up to 7 msec in whom splints and/or local prednisolone were tried 39 had complete relief and a further 6 partial relief. However, it should be noted that of 7 patients similarly graded who had no treatment, 2 had complete spontaneous remission and 3 had spontaneous improvement.

Thus the methods described give accurate differentiation between carpal tunnel syndrome and other causes of acroparæsthesiæ. The results of the tests also give some guidance as to the type of treatment likely to be successful: splints give a good chance of success in the milder affected cases; prednisolone is preferred for those moderately affected and decompression for those severely affected.

*Acknowledgments:* I would like to thank the physicians and surgeons who have referred cases for this investigation and who have allowed me to follow up their patients.

REFERENCES

**Crow R S** (1960) *Brit. med. J.* i, 1611
**Dawson G D & Scott J W** (1949) *J. Neurol. Psychiat.* **12**, 259
**Gilliatt R W & Sears T A** (1958) *J. Neurol. Psychiat.* **21**, 109
**Goodman H V & Gilliatt R W** (1961) *Ann. phys. Med.* **6**, 137
**Heathfield K W G** (1957) *Lancet* ii, 663
**Kendall D** (1960) *Brit. med. J.* ii, 1633
(1961) *Brit. med. J.* ii, 1293
**Lees F & Liversedge L A** (1959) *Lancet* ii, 1120
**Lishman W A & Russell W R** (1961) *Lancet* ii, 941
**Sears T A** (1959) *J. Physiol.* **148**, 30P
**Simpson J A** (1956) *J. Neurol. Psychiat.* **19**, 275
**Thomas P K** (1960) *Neurology* **10**, 1045