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CARPAL TUNNEL SYNDROME IN RHEUMATOID ARTHRITIS

A CLINICAL AND ELECTRODIAGNOSTIC SURVEY*

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The term carpal tunnel syndrome (CTS) refers to the clinical condition presenting with paraesthesiae or dysaesthesiae in the median nerve distribution, and coincidental impairment of function of those muscles of the thenar eminence supplied by the median nerve, with or without objective motor and sensory signs. This was first adequately described by Brain, Wright, and Wilkinson (1947) and is attributed to partial or complete interruption of median nerve motor and sensory pathways at the wrist.

While this syndrome may occur *de novo* in some patients, notably young and middle-aged women, certain causative or predisposing conditions are well known, including myxoedema, acromegaly, amyloid disease, ganglion formation (Robbins, 1963), pregnancy, and rheumatoid arthritis.

Estimations of the incidence of CTS in rheumatoid arthritis vary and the true frequency of the association is unknown. Brewerton (1965) considered CTS to be common in the early stages of rheumatoid arthritis, becoming less frequent later in the disease. Moritz (1964) found it an uncommon association.

Clinically this diagnosis may easily be missed—muscle wasting and hand pain being attributed, by both patient and physician, to the underlying rheumatoid arthritis. Yet it is an important diagnosis to make, for treatment is simple, and the function of the rheumatoid hand is probably exceptionally vulnerable to even a mild superadded neurological defect (Currey, 1965).

The diagnosis of the CTS has been aided by electrodiagnostic techniques, which include electromyography of the abductor pollicis brevis muscle and measurement of the motor latency in the segment of the median nerve between the wrist and this muscle, and of sensory conduction between the index finger and the wrist.

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It has been shown that compression of the median nerve at the wrist slows both motor (Simpson, 1956; Thomas, 1960) and sensory (Gilliatt and Sears, 1958) conduction times.

Method

Three groups, each of fifteen patients, with classical or definite rheumatoid arthritis according to the criteria of the American Rheumatism Association (Ropes, Bennett, Cobb, Jacox, and Jessar, 1958), but all having positive tests for rheumatoid serum factor, were selected at random as they attended the Departments of Physical Medicine and Rheumatology at The London Hospital. The first group had had symptoms of their disease for less than 2 years, the second group for between 2 and 10 years, and the third group for over 10 years. Of the 45 patients, 29 were women and 16 men. Twenty control subjects matched for age and sex were chosen at random from patients attending for non-inflammatory arthritic conditions which did not affect the neck or upper limbs. All patients were invited to take part in electrodiagnostic studies before any clinical symptoms or signs suggestive of the carpal tunnel syndrome were elicited.

At the Electrodiagnostic Clinic, the following routine was adopted:

(1) The patient was questioned about symptoms suggestive of the CTS/or of neuropathy in other distributions.

(2) The patient was examined for any neurological deficit in the hands, and the clinical state of the wrist joints was recorded.

(3) The following electrodiagnostic studies were performed, using a GHS type VI electromyograph in a warm well-ventilated room (by one of the authors—C.G.B.)

(*a*) Electromyography of the abductor pollicis brevis muscles, using a bi-polar co-axial needle with both visual and auditory recording.

(*i*) A search was made for fibrillation sampling in at least eight areas in each muscle.

(*ii*) The volitional (maximal) activity was elicited.

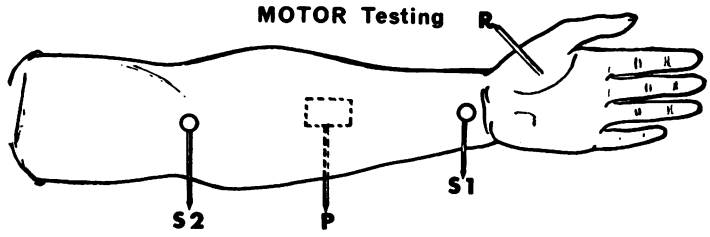

Fig. 1.—Position of recording and stimulating electrodes for motor testing.

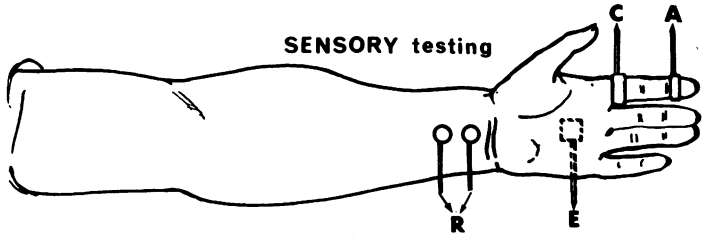

Fig. 2.—Position of recording and stimulating electrodes for sensory testing.

(*b*) Measurement of median nerve motor latencies and conduction velocity in the forearm (Fig. 1).

The recording electrode (R) was a co-axial needle in the abductor pollicis brevis muscle and the stimulus was given through a pad electrode of 2 cm. diameter placed 1-2 cm. above the distal wrist crease (S1) with a remote plate electrode (8 × 5·5 cm.) covered in saline-damped lint on the dorsum of the forearm (P). The stimulus was given × 1 or × 2 per second and was of a fixed duration (square wave) of 0·3 msec. The intensity was a continuous variable up to 200 volts. The threshold stimulus to produce a response in the abductor pollicis brevis was then obtained and the intensity of the stimulus then increased to supramaximal levels. The latency—taken as the interval between the stimulus and the beginning of the action potential—was measured from the face of the oscilloscope which was calibrated in centimetres and a variable time base speed of 1, 5, and 10 msec./cm. being available. Direct measurement from the face of the oscillograph was shown to give results identical to those obtained from a photograph of the tracing (Fig. 3). The zero point of each sweep triggered the stimulus. This was then repeated with the stimulating electrode being placed over the median nerve in the ante-cubital fossa just medial to the biceps tendon (S2). The distance between the two points of stimulation was measured and the conduction velocity of the median nerve in the forearm thus calculated.

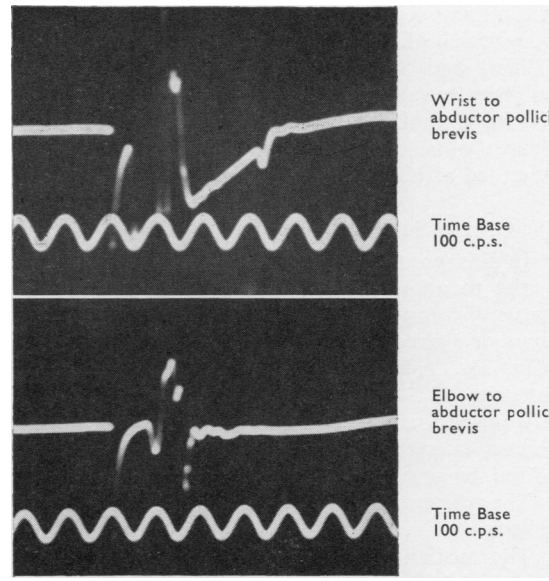

Fig. 3.—Motor latencies in median nerve from wrist and elbow to abductor pollicis brevis of 6·0 and 9·5 msec. respectively (time base 100 c.p.s.)

(*c*) Measurement of ulnar nerve motor latencies (in 26 out of the 45 patients): using the same technique, latencies from the wrist and the elbow to the abductor digiti minimi muscles were measured and the ulnar nerve conduction velocities in the forearm calculated.

(*d*) Measurement of median nerve sensory latencies and amplitudes (Fig. 2). The method described by Dawson (1956) was adopted with only minor modification. The stimulating electrodes were a pair of lint-covered metal strips placed around the index finger, the cathode (C) being placed as close as possible to the metacarpophalangeal joint, and the anode (A) just distal to the terminal interphalangeal joint. An isolating earth plate electrode (E) was placed on the dorsum of the hand using Cambridge electrode jelly. The recording electrodes (R) were a pair of lint-covered metal studs (15 ×

8 mm.) mounted 1·5 cm. apart in Perspex and placed over the median nerve (after skin abrasion) the distal electrode being approximately 2 cm. above the distal wrist crease. Stimuli of constant (0·3 msec.) duration and of variable intensity up to 30-100 volts were given at a frequency of 1 per second. The resultant action potential was measured directly from the oscilloscope screen—the latency being measured on the calibrated time base from the stimulus to the peak of the impulse (again direct measurement from the oscillograph was shown to be identical to measurement obtained from a photographic record), and the amplitude being measured directly (from peak to peak) from the vertical centimetre calibration on the oscilloscope screen using a gain of 10 microvolts/cm. deflection.

Results

The normal values for the median nerve motor latency between the wrist and the abductor pollicis brevis muscle have been assessed on several occasions (Simpson, 1956; Thomas, 1960; Campbell, 1962; Preswick, 1963; Moritz, 1964). In this investigation, a motor latency greater than 5 msec. was regarded as abnormal. Similarly sensory latencies and amplitudes in the median nerve at the wrist after stimulation of the index finger have been measured. Gilliatt and Sears (1958) showed that in normal subjects the upper limit of the latency is 4 msec. and the lower limit of the amplitude is 9 microvolts—figures outside these limits have, therefore, been regarded as abnormal in the present investigations.

(1) Control Subjects

In this group one patient had a motor latency (wrist to abductor pollicis brevis) in one median nerve of 6 msec., without abnormal clinical symptoms or signs and with normal sensory conduction (Fig. 3). The remaining patients all had motor latencies of 5 msec. or less (overall range 3·0 to 6·0 msec.; mean 4·4 msec.; S.D. ± 0·5 msec.), sensory latencies of 4 msec. or less, and an amplitude of the sensory potential of 10 microvolts or more.

Thus these results were in agreement with previous studies.

The median and ulnar nerve motor conduction velocities in the forearms were normal in all subjects (range: *median* 45 to 83 M/sec.; mean 63 M/sec.; S.D. ± 12 M/sec.; *ulnar* 48 to 90 M/sec.; mean 61 M/sec.; S.D. ± 11 M/sec.).

Three patients (15 per cent.) had clinical symptoms and signs suggestive of the CTS but with normal electrodiagnostic tests.

(2) Rheumatoid Arthritic Patients

(*i*) *Electrodiagnostic Tests.*—The number of patients who had abnormal electrodiagnostic tests, and the nature of these abnormalities are shown in Tables I, II, and III (opposite).

There was no statistically significant difference in the incidence of electrical abnormalities between the three groups of patients with rheumatoid arthritis diagnosed by electrodiagnostic techniques as having a carpal tunnel syndrome ($0·2 > P > 0·1$ by χ^2 test). Therefore, in the following discussion patients with rheumatoid arthritis are considered as a single group. The range of motor latencies (wrist to abductor pollicis brevis muscle) in the median nerve was 3·0 to 9·0 msec. (mean 4·5 msec.; S.D. ± 0·8 msec.). (Fig. 4.). This range is not significantly different when compared with the control group ($0·4 < P < 0·5$ by "t" test). However, as shown in Fig. 4, the motor latencies at the wrist in the control group (with a single exception) did not exceed 5 msec., whereas in the rheumatoid arthritics values over 5 msec., were obtained.

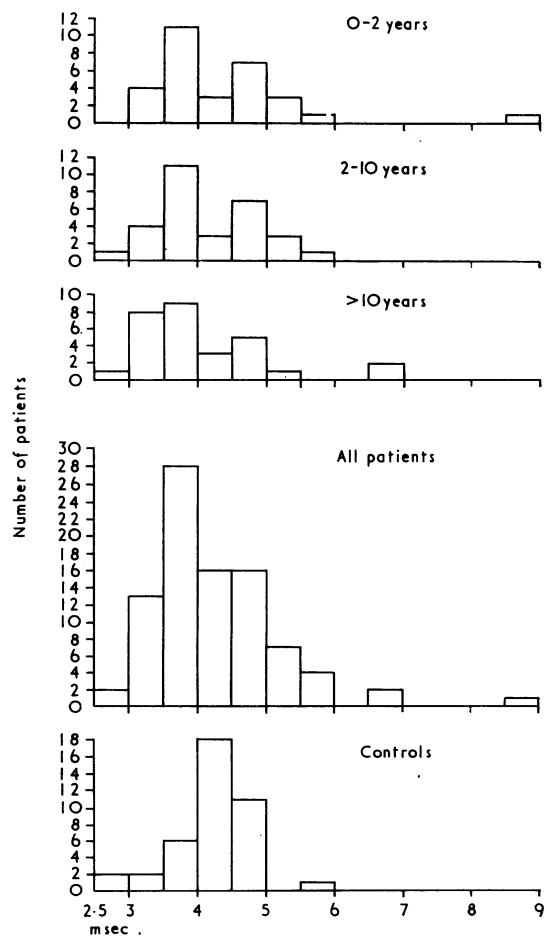

Fig. 4.—Median nerve motor latencies (wrist to A.P.B. muscle).

TABLE I

NUMBER OF PATIENTS WITH RHEUMATOID ARTHRITIS WITH ABNORMAL ELECTRODIAGNOSTIC TESTS

Duration of Disease (yrs)	Total No. of Patients	Abnormal Electrodiagnostic Tests	
		No.	Per cent.
0–2	15	5	33·3
2–10	15	10	66·6
>10	15	7	46·7
	45	22	49

Six of the 45 patients had a motor conduction velocity in the forearm in one median nerve of less than 45 M/sec. (range 37 to 44 M/sec.), the opposite median nerve in each case having a normal conduction velocity. In one of these patients this was associated with clinical symptoms and signs and abnormal electrodiagnostic tests indicating the presence of the carpal tunnel syndrome, and in three patients the slightly slowed conduction velocity was associated with clinical symptoms and signs of the carpal tunnel syndrome but motor and sensory latencies at the wrist were normal.

TABLE III

ANALYSIS OF ABNORMAL ELECTRODIAGNOSTIC TESTS IN PATIENTS WITH RHEUMATOID ARTHRITIS

Abnormality	Patients	
	No.	Per cent.
Motor	3	13·6
Motor and EMG	1	4·5
Motor and Sensory	7	31·9
Sensory	9	41·0
Sensory and EMG	2	9·0
Motor and Sensory and EMG ..	0	0
Total	22	100

TABLE II

SYNOPSIS OF ABNORMAL ELECTRODIAGNOSTIC RESULTS IN PATIENTS WITH RHEUMATOID ARTHRITIS

Duration of Disease (yrs)	Patient No.	Sex	Right				Left			
			Motor Latency Wrist to APB (msec.)	Sensory		EMG of APB	Motor Latency Wrist to APB (msec.)	Sensory		EMG of APB
				Amplitude (μv.)	Latency (msec.)			Amplitude (μv.)	Latency (msec.)	
0–2	1	F							4·5	
	3	F					9·0	Absent		
	5	M					5·5			
	7	F	5·5		4·5					
	13	M	6·0		4·5		5·5		4·5	
2–10	17	F	5·5		4·5			5	5	
	18	M			4·5				4·5	
	20	M						Absent		
	21	M	6·0	Absent			6·0			
	22	M	5·5							
	23	F					6·0			
	25	M						5		
	28	F		5						
	29	F					5·5			Fibrillation
	30	M		5	5			Absent		
>10	31	M					5·5	5		
	32	F		Absent					5	
	35	F		Absent		Fibrillation				
	38	F	7·0		5		6·8		5	
	39	F		5				5		
	41	M		5						
	44	M		Absent		Fibrillation		Absent		Fibrillation

The motor conduction velocities in the forearm in the remaining 84 median nerves were normal. Thus the overall range of median nerve motor conduction in the forearm was 37 to 83 M/sec. (mean 56 M/sec.; S.D. ± 10 M/sec.). These values are significantly lower than in the control group ($P<0.001$ by "t" test).

Ulnar nerve motor testing was performed in 26 of the 45 patients. In all cases the latency (wrist to abductor digiti minimi muscle) was normal (range 1·5 to 5·0 msec.) and the motor conduction velocity in the forearm was also normal (range 45 to 80 M/sec.; mean 57 M/sec.; S.D. ± 9 M/sec.).

The one patient who also complained of paraesthesiae in the feet (and who did not have abnormal symptoms, signs, or electrodiagnostic tests in the arms) had the motor conduction velocity measured in one lateral popliteal nerve which was normal. Thus, of 45 patients with rheumatoid arthritis, 22 (49 per cent.) had abnormal electrodiagnostic tests indicative of functional impairment of the median nerve at the level of the wrist. By motor testing alone, abnormalities would have been detected in eleven of these 22 patients (50 per cent.), whereas by sensory testing alone, eighteen of the 22 (82 per cent.) would have shown some abnormality. Of the 22 patients diagnosed electrically as having the CTS, nine (41 per cent.) had both hands affected.

Although skin temperature measurements were not made, since the recordings were all performed in a warm room and since the results in the control group of patients and of ulnar nerve testing were consistently within the normal range, fluctuations in skin temperature are unlikely to have had any appreciable effect.

(*ii*) *Correlation of Symptoms and Signs suggestive of the Carpal Tunnel Syndrome with Results of the Electrical Tests* (Table IV).—Fifteen patients (33·3 per cent.) with rheumatoid arthritis had clinical symptoms and signs and electrical findings consistent with a diagnosis of CTS, while a further seven patients (15·5 per cent). had electrical findings alone consistent with this diagnosis—a total of 49 per cent. of the patients. In addition to this, nine patients (20 per cent.) had symptoms and/or signs which clinically suggested this diagnosis but their electrodiagnostic tests were normal. Thus, by clinical criteria alone, 24 patients (53·3 per cent.) were diagnosed as having the CTS, and by clinical and electrodiagnostic techniques 31 patients (69 per cent.) were so diagnosed (18 females and 13 males). These figures are highly significant when compared with the control group (Table V).

TABLE IV
CORRELATION OF CLINICAL FEATURES WITH ELECTRODIAGNOSTIC TESTS IN 45 PATIENTS WITH RHEUMATOID ARTHRITIS

Clinical Features	Electrodiagnostic Tests	
	Abnormal	Normal
Symptoms and Signs of the CTS ..	11	7
Motor and Sensory Signs of the CTS ..	4	2
Motor Signs Only	5	9
No Symptoms or Signs	2	5
Total	22	23

Nine patients (20 per cent.) had clinical symptoms and signs of bilateral involvement of the median nerve at the wrist (of whom four had abnormal electrodiagnostic tests bilaterally, and two abnormal tests on one side). Thus, a total of fourteen patients (31 per cent.) were found to have bilateral CTS by clinical and/or electrodiagnostic techniques.

(*iii*) *Correlation of the Clinical State of the Wrist Joint with the Diagnosis of the Carpal Tunnel Syndrome.*—The degree of synovial swelling of the wrist varied greatly. There was no correlation between the clinical state of the wrist joint and the presence of the CTS, whether diagnosed clinically or electrically. Some patients with definite CTS had only minimal synovial swelling, and conversely some

TABLE V
STATISTICAL COMPARISON OF THE DIAGNOSIS OF THE CARPAL TUNNEL SYNDROME IN RHEUMATOID ARTHRITIC AND CONTROL PATIENTS

Diagnosed by	Rheumatoid Arthritics (45)		Controls (20)		Statistical Significance χ^2 Test (Yates' correction)
	No.	Per cent.	No.	Per cent.	
Electrodiagnostic Methods	22	49	1	5	$0.001<P<0.01$
Clinical Methods	24	53·3	3	15	$0.001<P<0.01$
Clinical and Electrodiagnostic Methods	31	69	4	20	$P<0.001$

with gross synovial swelling had no evidence of median nerve compression.

Discussion

These figures support the previously suspected association between rheumatoid arthritis and the CTS. Moritz (1954), however, studying motor nerve conduction in rheumatoid arthritis, discovered only two patients in his series of 32 with objective clinical findings suggestive of the CTS and only one with a slightly delayed median nerve latency (wrist to abductor pollicis brevis muscle). The present figures indicate a far higher incidence of the CTS in rheumatoid arthritic patients. The reason for this discrepancy is not clear.

As has been pointed out previously (Goodman and Gilliatt, 1961), by measuring the motor latency from the point of stimulation to the earliest deflection, the conduction of the fastest and, therefore, least affected fibres are being measured. It is thus possible that mild cases of median nerve compression at the wrist are being missed by this electrodiagnostic technique. This probably accounts for the fact that, in surveys of patients with clinical carpal tunnel syndromes, electrodiagnostic studies have not produced abnormal findings in all the patients. Thomas (1960) found that only sixty of ninety patients with a clinical carpal tunnel syndrome had motor nerve delay at the wrist, and Goodman and Gilliatt (1961) found that 29 of their 33 patients had abnormal sensory conductions. Since the present investigation has revealed that 49 per cent. of these rheumatoid patients had electrical signs of the CTS (including 33·3 per cent. with clinical evidence of this syndrome), and a further 20 per cent. had suggestive clinical symptoms and signs but with normal electrodiagnostic findings (a total of 69 per cent. of the patients), we must consider that the CTS is very frequently associated with rheumatoid arthritis. Secondly, 31 per cent. of the patients had bilateral median nerve involvement at the wrist. Thirdly, since 15·5 per cent. of these patients with the CTS were diagnosed on electrodiagnostic testing alone, this raises the question whether routine testing by nerve conduction studies should be carried out.

Weakness and/or wasting of the abductor pollicis brevis muscles alone in rheumatoid arthritis, does not add to the diagnosis of carpal tunnel syndrome, as this muscle may waste together with the other small muscles of the hand. This view is supported, in this series, since 20 per cent. of the patients showed weakness and/or wasting of the abductor pollicis brevis muscles, but had no symptoms, signs, or abnormal electrical findings suggestive of median nerve compression.

Thomas (1960) demonstrated that, in patients with the CTS, the median nerve motor conduction velocity in the forearm may be slightly delayed. Four cases of the CTS (three based on clinical symptoms and signs alone, and one diagnosed both clinically and by electrodiagnostic tests) in this series also had mild slowing in the forearm (37, 40, 42, and 44 M/sec.), as did two others without objective signs of the CTS (44 and 43 M/sec.). The median nerve motor velocity in the forearm in the group of rheumatoid arthritics was significantly slower than that in the control group ($P < 0{\cdot}001$ by "t" test). The reason for this slowing of conduction proximal to a peripheral nerve lesion is well recognized but of unknown cause.

Tension within the flexor compartment at the wrist (Ficat and Arlet, 1956; Arlet, Ficat, and Malbosc, 1966) is clearly not the only cause of median nerve lesions in rheumatoid arthritis, and it is not possible in every case to exclude a localized rheumatoid neuropathy. A complete electrodiagnostic survey in every patient was not practicable, but the absence of signs or symptoms of "mononeuritis multiplex", and the normal motor conduction velocities in those ulnar nerves tested, make it unlikely that these abnormalities were part of a polyneuropathy.

The fact that the electrical abnormalities were so common, and often only slightly outside the limits adopted as normal, raises the question whether the normal limits used were too stringent. The sharp difference between the rheumatoid patients and the control group statistically makes this unlikely, and one must conclude that conduction defects in the median nerve at the wrist, although often slight, are very common in rheumatoid arthritis. Re-examination of these patients after an interval will be necessary to determine whether the condition is fixed or changing. The present data, however, show that the abnormality occurs at all stages of the disease, and that, clinically, gross wrist joint changes are not a predisposing cause.

The important question to be answered is whether minor electrical changes indicate a functionally significant lesion needing treatment. Function in the rheumatoid hand is achieved by compensating for anatomical, mechanical and inflammatory changes. This delicately balanced state is obviously particularly vulnerable to a superimposed, neurological deficit, whether sensory, motor, or mixed. It is for this reason that surgical relief of well-established CTS is perhaps the most rewarding operation

to be performed on the rheumatoid hand. What of the minor changes which this survey show to be so common—do they justify any form of treatment or routine screening of patients?

The answers may be provided by careful study of selected patients in whom detailed functional assessments are performed before and after surgical or other forms of treatment for median nerve compression. Meanwhile, for those caring for rheumatoid patients, this study underlines the constant obligation to search for this eminently treatable condition.

Summary

45 patients with classical or definite rheumatoid arthritis were examined clinically and by electrodiagnostic methods for evidence of the carpal tunnel syndrome, and the results were compared with those in twenty control subjects.

Electrodiagnostic tests were abnormal in 49 per cent. (of whom 33 per cent. also had clinical evidence of median nerve compression) and a further 20 per cent. had clinical symptoms and signs of the carpal tunnel syndrome alone—a total of 69 per cent. of the patients.

It is concluded that the carpal tunnel syndrome, often mild, is a common complication of rheumatoid arthritis.

We wish to thank Wing Commander C. B. Wynn-Parry for help and advice; Dr. W. S. Tegner and Dr. R. M. Mason for their permission to study patients under their care; and Dr. R. M. Greenwood for his help in the statistical analysis of the results.

DISCUSSION

DR. BACKHOUSE (*London*): I wonder if Dr. Barnes looked at the state of the synovium of the flexor tendon?

DR. BARNES: Examination of the wrist was entirely clinical. This was purely a correlation between clinical signs there and both clinical and electro-diagnostic evidence of the presence or absence of the carpal tunnel syndrome. We have not yet been able to compare these results with the findings at operation.

DR. V. WRIGHT (*Leeds*): We have some patients who presented with a carpal tunnel syndrome and later developed rheumatoid arthritis. Has Dr. Barnes noticed this? Have skin temperatures been taken into account? When we screened out rheumatoid patients we found a number of abnormalities, but discovered that when one recorded skin temperature many conduction times which appeared to be abnormal fell within the normal range. Did the skin temperature have any effect on your results?

DR. BARNES: This was a random series of patients attending on account of arthritis, with no special attention being paid to the onset of this syndrome. We did not measure skin temperature, but the EMG cubicle in the Department here where the tests were performed, is in a warm, non-draughty place. All recordings with a few exceptions have been done during the warm months of the year.

DR. W. S. C. COPEMAN (*London*): Last year, I had two patients who had operations for this syndrome. Symptomatically, operation has given them complete relief, but the cause of the pressure was reported as being a cystocele of the median nerve and not, apparently, the carpal ligament at all. I was wondering whether this has been recorded and if it was an important cause, and also of course whether it is connected with the arthritis or not.

DR. BARNES: I cannot give you the answer, except to say that we have never found this.

DR. M. K. JASANI (*Glasgow*): Could the sympathetic nerves also be compressed in the carpal tunnel and explain Dr. Wright's observations on skin temperature, since sympathetic nerves regulate skin temperature? Was Raynaud's phenomenon more common in your patients?

DR. BARNES: Several patients did complain of symptoms suggestive of Raynaud's phenomenon.

DR. D. A. H. YATES (*London*): The question concerning temperature has perhaps been answered in your paper by the fact that ulnar conduction was normal. Was there any difference whether the patient was right or left-handed, such as a greater incidence of carpal tunnel syndrome in the dominant hand?

DR. BARNES: This is an important point, but I have not the answer. Also, adding to your remarks, I think the most important point of this was the strong statistical conclusion showing a clear difference between the rheumatoid arthritis group and the control group; this would presumably have been made less had temperatures been a relevant factor or had there been a fault in the technique.

DR. M. MCMAHON (*Cork*): Dr. Barnes, what is the sex factor in these rheumatoid patients? I ask this because in the last 2 years I have only come across four men with carpal tunnel syndromes and they all turned out to be rheumatoid patients. In women carpal tunnel syndrome seems to be very common—in Ireland anyway—and I have noticed large numbers drifting on to rheumatoid arthritis; hence I now regard any woman with carpal tunnel syndrome as a potential rheumatoid arthritic. Another thing I notice is that many rheumatoid arthritic patients with carpal tunnel syndrome find the pain as bad by day as by night.

DR. BARNES: The sex ratio in the arthritic group wa roughly as one would expect, about twice as many females as males; it was approximately the same as those with the carpal tunnel syndrome. Thirteen of those diagnosed as having the carpal tunnel syndrome were men. Of those who were diagnosed either clinically or electrodiagnostically, the vast majority said that their pain and paraesthesiae were worse by night.

DR. E. N. COOMES (*London*): What were the clinical criteria?

DR. BARNES: As far as symptoms were concerned, there had to be paraesthesiae or dysaesthesiae in median nerve distribution, and those who had both electrodiagnostic and clinical signs of the carpal tunnel syndrome had symptoms confined to the median nerve distribution. Regarding signs, I only accepted those with both motor and sensory signs and occasionally a positive Tinel test as well. Lone wasting or weakness of the abductor pollicis brevis muscles was not included in the clinical diagnostic criteria.

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Le syndrome du canal carpien dans l'arthrite rhumatismale

RÉSUMÉ

On examina cliniquement et par des méthodes électrodiagnostiques 45 malades atteints d'arthrite rhumatismale classique ou "définie" pour rechercher des signes du syndrome du canal carpien et on compara les résultats avec ceux obtenus chez 20 témoins.

Les tests électrodiagnostiques furent anormaux en 49 pour cent (dont 33 pour cent portèrent des signes cliniques del compression du nerf médian) et, de plus, 20 pour cent des sujets manifestèrent des symptômes et des signes cliniques de syndrome du canal carpien seul—en 69 pour cent des malades en tout.

On en conclut que le syndrome du canal carpien, souvent bénin, est une complication commune de l'arthrite rhumatismale.

El síndrome del canal del carpo en la artritis reumatoide

SUMARIO

Se examinaron clínicamente y por métodos electrodiagnósticos 45 enfermos afectos de artritis reumatoide clásica o "definida" para evidenciar el síndrome del canal del carpo, comparándose los resultados con los obtenidos en 20 testigos.

Los tests electrodiagnósticos fueron anormales en un 45 por ciento (un 33 por ciento de los cuales acusaron síntomas clínicos de compresión del nervio mediano) y, además, un 20 por ciento de los sujetos manifestaron signos y síntomas clínicos de síndrome del canal del carpo sólo—dando un total de un 69 por ciento de los enfermos.

Se concluye que el síndrome del canal del carpo, frecuentemente de poca gravedad, es una complicación común de la artritis reumatoide.