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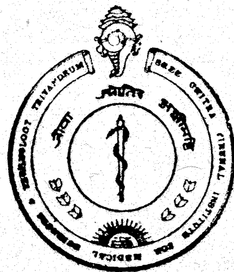

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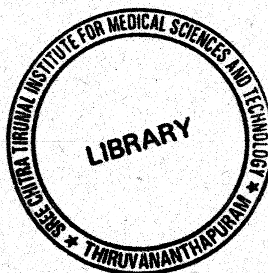

PROJECT REPORT

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NAME : Dr. JOSEPH CHERIAN PERUMPILLICHIRA

PROGRAMME : D.M. NEUROLOGY

MONTH & YEAR OF SUBMISSION : NOVEMBER, 1997

PROJECT REPORT

TITLE OF THE PROJECT: CARPAL TUNNEL SYNDROME-A CLINICAL AND ELECTROPHYSIOLOGICAL STUDY

NAME: Dr. JOSEPH CHERIAN PERUMPILLICHIRA

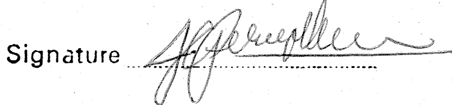
PROGRAMME: D.M. NEUROLOGY

MONTH & YEAR OF SUBMISSION: NOVEMBER 1997

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM 695011

CERTIFICATE

I, Dr. JOSEPH CHERIAN.P. hereby declare that I have actually, performed all the procedures listed / carried out the project, under report.

Signature 

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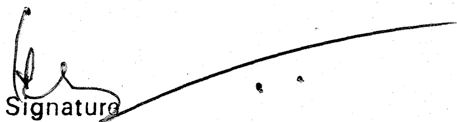

Signature
Head of the Department

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CARPAL TUNNEL SYNDROME - A CLINICAL AND ELECTROPHYSIOLOGICAL STUDY

INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common peripheral mononeuropathy[1]. In a population - based study[2] conducted in Rochester, Minnesota, the age-adjusted incidence rates of this entrapment neuropathy was 88 (cases per 100,000 person-years) during the period of 1961 to 1965 and 125 during the period of 1976 to 1980. In a survey[3] conducted among health care practitioners in Santa Clara county (population: 1.4 million) of California, a total of 7214 CTS cases were reported in the year 1987, of which 47% were believed to be work-related.

No population-based study on CTS has been done in India. In a recent study[4] from South India, out of the 650 cases with peripheral nerve disorders studied in an electrophysiology laboratory during a 2.5 year period, CTS constituted 7%. It formed 2.5% of all the cases of peripheral neuropathies studied over a period of 15 years at NIMHANS, Bangalore[5].

The early detection and treatment of CTS is important in preventing permanent damage to the median nerve, resulting in persistence of symptoms and disability[6]. The correct diagnosis is also important to exclude other causes of acroparesthesia, to avoid costly diagnostic procedures such as MRI, and to establish the most suitable treatment[7].

This study was undertaken to evaluate prospectively, the predisposing conditions, clinical features and electrophysiological parameters in patients referred with a diagnosis of CTS to the electrophysiology lab in a tertiary referral centre in South India.

MATERIALS AND METHODS

PATIENTS:-

We prospectively studied persons referred with the diagnosis of CTS on an outpatient basis for nerve conduction studies to the electrophysiology lab of Sree Chitra Tirunal Institute for Medical Sciences and Technology, during the period of October 1996 to July 1997.

50 persons were referred with a diagnosis of CTS. 39 of them met the electrophysiological criteria for CTS. They constituted 5.53% of the 705 number of electrophysiological studies carried out for suspected peripheral nerve disorders during this period. Out of this, 31 patients (58 hands) fulfilling the clinical and electrophysiological criteria for CTS were the subjects of this study.

Clinical data like age at the onset of symptoms, duration of symptoms, handedness, presence or absence of acroparesthesia, weakness of hand, dryness of skin, provocative factors like sleep, sustained hand posture, repetitive actions of the hand, and relieving factors were recorded using a prepared proforma. Associated conditions

like diabetes mellitus, rheumatoid arthritis, hypothyroidism, pregnancy, cervical radiculopathy and presence of arteriovenous fistula in the limb were looked for.

Patients were examined for the presence of thyroid swelling,bradycardia, slowly relaxing deep tendon reflexes, pedal oedema, acromegalic features and joint changes suggestive of rheumatoid arthritis.

Body Mass Index [Weight in kg/(Height in metres)^2]was measured in all. Examination of the hands consisted of inspection of skin for dryness over thumb, index and middle fingers, atrophy and weakness in the thenar muscles, sensory loss in the median nerve distribution and presence of Tinel's and Phalen's signs. All deep tendon reflexes were assessed and any abnormality noted.

ELECTROPHYSIOLOGY

All studies were done using a Nicolet Viking IV (Nicolet Biomedical Inc., Madison, Wisconsin) and an S 403 electrical stimulator probe. Surface recording and stimulation were used for the nerve conduction studies. Recording electrodes were two 10mm. Silver disk electrodes supplied by Nicolet for motor and orthodromic sensory nerve conduction studies and Medelec digital (ring) electrodes E/DS-K 16639 for the antidromic sensory studies. Temperature of the limb was measured using a temperature probe and studies were done at or above 33°c. The machine settings for motor nerve conduction studies (NCS) were, low frequency filter (LFF) 2 Hz, and High frequency filter (HFF) 10 KHz. The onset latencies were measured at a sensitivity of 1mv and the amplitudes were measured from peak to peak. The sensory NCS were done at LFF 20

Hz, HFF 3 KHz and onset latency of potentials were measured at a sensitivity of 10 micro volts. The F waves were recorded with LFF 2Hz, HFF 5kHz and a sensitivity of 500 microvolts and atleast 20 responses were measured.

Nerve Conduction Studies

Median motor conduction studies were performed by recording the compound muscle action potential (CMAP) from the abductor pollicis brevis (APB) using the belty-tendon technique, with the active electrode(G1) placed over the muscle belly and the reference electrode(G2) over the first metacarpophalangeal (MP) joint. Median nerve was stimulated at the wrist, 2cm proximal to the wrist crease, between the tendons of flexor carpi radialis and palmaris longus muscles. A 0.2 ms supramaximal stimulus was used and distal motor latency (DML) was measured. Proximally, the nerve was stimulated at the antecubital fossa, between the biceps tendon and medial epicondyle, over the brachial artery . To stimulate the median nerve motor fibres in the palm (i.e. recurrent thenar nerve) the cathode was placed at a point 7cm distal to the wrist stimulation point, along a line connecting it with the web space between digits 2 and 3. The anode was kept directed distally towards the base of the 5th digit. This was done to avoid depolarising the recurrent thenar nerve beneath the anode. The nature of the thenar twitch was carefully observed, as stimulation of the deep branch of the ulnar nerve can also generate a motor response recorded over thenar eminence. If an adduction thenar twitch occurred due to ulnar nerve stimulation, the stimulating electrode was repositioned in roughly millimetre increments towards the thenar eminence till an abduction twitch was obtained. The wave forms from palm and wrist stimulation were also compared. An

initial positivity or a change in configuration was taken as being suggestive of stimulation of the deep branch of the ulnar nerve in the palm. Distances were measured using a flexible tape measure.

The median antidromic sensory conduction studies were done by stimulating at the wrist and recording from the second digit with ring electrodes- with G1 over the MP joint and G2 over distal interphalangeal joint.

The ulnar nerve motor NCS were done by recording from the belly of the abductor digiti minimi (ADM) with the G2 placed at the MP joint. The nerve was stimulated at the wrist, 2cm proximal to the wrist crease, radial to the flexor carpi ulnaris tendon, and at the elbow, below and lateral to the medial epicondyle.

The orthodromic sensory nerve action potentials (SNAPs) from the median and the ulnar nerves were recorded at the wrist, with the G1 kept over the motor nerve stimulation point and G2, 3cm proximal to it. The stimulation was done in the palm over equal distances of 8cm each from G1, along a line connecting it to the 2nd interdigital web space for median and the 4th interdigital webspace for the ulnar nerve.

Criteria for diagnosis

The clinical diagnosis of CTS was made if the patient complained of any two of the following symptoms. Dull, aching discomfort in the hand, with paresthesias in the median nerve distribution, precipitated by sleep, sustained hand posture or repititive actions of the hand or wrist and relieved by change in hand posture or shaking the hand.

The following clinical signs, though useful as adjunct were not mandatory - weakness, with or without wasting of thenar muscles, Tinel's sign at the wrist and paresthesias provoked in the median nerve distribution by Phalen's manoeuvre.

The electrophysiological criteria for diagnosis of CTS was abnormality of one or more of the following tests.

1. The distal motor latency from the wrist to APB of >4.1ms

2. Prolonged onset latency of antidromic SNAP to 2nd digit >3.0 ms

3. Orthodromic SNAP onset latency (OSLM) in the median nerve palm - wrist segment >2.0ms.

4. Orthodromic SNAP latency difference (OSLD) in the median palm-wrist segment that was > = 0.4ms than the latency for the analogous segment of the ulnar nerve, using a distance of 8 cm for both.

Both the upper limbs and the peroneal and sural nerves on one lower limb were studied in all patients. Patients were excluded from analysis if the electrophysiological studies were consistent with peripheral neuropathy or multiple mononeuropathies.

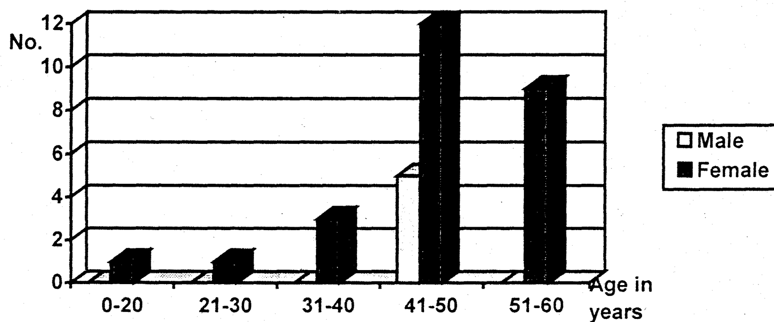
Controls:-

21 age matched controls who did not have symptoms or signs of carpal tunnel syndrome were chosen, from among inpatients at the hospital without peripheral

nerve disorders and healthy volunteers. A volunteer who had an old fracture and deformity of the left wrist and another asymptomatic control who had electrophysiological evidence for CTS were excluded. Thus, 40 hands were studied in 21 subjects and the control values obtained.

RESULTS:

31 patients (male=5, female = 26, female:male ratio 5.2:1) who met the clinical and electrophysiological criteria for CTS were studied (58 hands). Mean age of patients was 46.4 years (S.D = 9.12, range = 18-60)



Of the patients with CTS, 2 were referred to the lab with the diagnoses of peripheral neuropathy and bilateral cervical ribs respectively.

Nine patients who were referred as CTS, were found to have normal nerve conduction studies (8 patients) or an alternate diagnosis (entrapment of the (L) ulnar nerve at the elbow in one patient).

21 control subjects (Male = 13, female = 8, female:male ratio 1:1.63) of mean age 43.91 years (S.D = 12.05, range = 25-67), [40 hands], without peripheral nerve disorders were studied, to establish normative data.

All 31 patients and the 21 controls were right handed.

Mean age at the onset of symptoms was 43.74 years (S.D = 9.311, range = 18-60) the mean duration of symptoms at the time of presentation was 31.08 months (S.D = 37.48, range = 15 days to 144 months).

All patients had dull ache or paresthesias in the affected hands, which was intermittent. Symptoms were on the left in 7(22.6%), right in 9(29%) and bilateral in 15 (48.4%). Provocation of the symptoms by sleep was seen in 20 (64.52%) patients. 14 patients (45.16%) complained of weakness or clumsiness of the hand. 4 (12.90%) had noticed dryness, swelling or colour change in the symptomatic hands. 27 (87.1%) had the symptoms in the median nerve distribution. The 4 others (12.90%) had dull ache or pain involving the foream, arm or shoulder.

All patients had precipitation of symptoms by sustained arm or hand posture or repetitive actions of the hand or wrist. Writing, washing, using a knife or a broom were the common precipitants. Another common precipitant was sustained hand grip on the overhead bar during travel by bus.

Only one patient (3.23%) had history of diabetes mellitus History of thyroidectomy or overt features of hypothyroidism were present in 5 (16.13%).

One patient had pain in the neck, radiating to the shoulder and upper limb, suggestive of cervical radiculopathy.

The mean body mass index (BMI) of the patients was 24.95 (S.D = 4.014, range = 18.42 to 36.89), which was significantly($p < 0.002$) greater than that of controls (mean BMI = 21.72, S.D = 2.95, range = 17.66 to 31.02). Only 2 patients had a BMI greater than 29.

Wasting, weakness or both were seen in the thenar muscles in 19/58 hands (32.76%) [14/31 patients, (45.16%)]. Sensory loss in the distribution of the median nerve was seen in 21/58 hands (31.21%). Phalen's sign was positive in 22(37.93%) hands

ELECTROPHYSIOLOGY:

8 persons who had clinical features suggestive of CTS had normal nerve conduction studies. Of the 31 patients who met the electrophysiological criteria for CTS, the distal motor latency (DML) in the median nerve in the wrist to APB segment was prolonged (>4.1ms i.e, the mean value of controls + 2 S.D) bilaterally in 19 (61.29%) patients, on the right in 5 (16.31%) and in 5 on the left. The mean DML was 5.32 (S.D = 1.47, range = 3.3 to 10.4) milliseconds. The control value for DML was mean = 3.38 ms, (S.D = 0.29, range = 2.7 to 4.0. See table 1) 48/58 CTS hands (82.76%) had prolonged DML. 3/58 had only DML prolongation, with normal SNAPS and OSLD. (5.17%). These patients had normal distal motor and sensory latencies in the ulnar nerves of the same hands.

The motor response was absent in one hand.

Table 1. Median nerve motor NCS

Parameter	Controls	Patients	P value
DML in the Palm-APB segment	1.78 (0.24)ms	1.90(0.45)ms	
DML in the wrist-APB segment	3.38 (0.29) ms	5.32(1.47)ms	
Velocity in the wrist-palm segment	43.94(8.90) m/s	23.72(11.05) m/s	P<0.0001
Velocity in the elbow-wrist segment	56.28(4.87)m/s	54.48(6.06) m/s	P>0.05

Results are reported as mean (standard deviation)
DML = Distal motor latency in milliseconds (ms)
NCS = Nerve Conduction Studies

51 hands (87.93%) had prolonged (>3.0 ms in 43 hands) or absent (8 hands) antidromic median SNAP latency (ASLM) from digit 2. The mean value was 3.76 ms (S.D = 1.04, range = 2.1- 8.6 ms). The mean value for controls was 2.36 ms (S.D = 0.26, range = 1.9 - 3.0) The orthodromic SNAP latency for median nerve (OSLM) in the palm - wrist (P-W) segment was abnormal in 55 hands (94.83%). It was prolonged (>2.0 ms) in 47, and absent in 8 hands. Mean value for patients was 2.86 ms (S.D.=0.78, range= 1.5- 5.5 ms) and for controls, 1.64 ms (S.D= 0.14, range = 1.4 - 1.9 ms).

The orthodromic SNAP latency difference (OSLD) between analogous 8 cm lengths of median and ulnar nerves in the P-W segment was abnormal in 55 (94.83%)

out of 58 hands with the final diagnosis of CTS. It was prolonged (> = 0.4) in 47 and absent in 8. Mean value for patients was 1.29 ms (SD = 0.73, range = 0 - 3.9 ms) and for controls, 0.068 ms (S.D = 0.13, range = -0.2 to + 0.2 ms). Nine hands (15.52%) had SNAP abnormality alone. Four (6.9%) hands had OLSD abnormalities alone. 45 (77.6%) had all 3 parameters(DML,ASLM,OSLD) abnormal.

Table 2. Median nerve sensory NCS

Parameter	Patients	Controls
Antidromic SNAP latency to digit 2	3.76(1.04)ms	2.36(0.26) ms
Orthodromic SNAP latency in P-W segment	2.86(0.78) ms	1.64(0.14)ms
OSLD between median and ulnar in the P-W segment	1.29(0.73) ms	0.07(0.13)ms

Results are reported as mean (standard deviation)
SNAP = Sensory nerve action potential
OSLD = Orthodromic SNAP latency difference between analogous 8cm long segments
P-W = Palm-wrist.

The mean velocity in the motor fibres of the median nerve in the wrist-palm segment for patients was 23.72 m/s (S.D= 11.05, range = 8 - 62 m/s) Which was significantly (P<0.0001) slower than that for controls - mean = 43.94 m/s (S.D = 8.90, range = 30 - 78).(Table 1)

The mean distoproximal ratio (the ratio of CMAP amplitude elicited from APB on palmar stimulation of the median nerve to the CMAP elicited by stimulation at

the wrist) for patients was 2.50 (S.D = 4.19, range = 0.99-20.52). The mean ratio in controls was 1.084 (S.D = 0.11, range =1 - 1.68).(p= 0.023)

Table 3. Distoproximal ratio for Median nerve CMAP

Parameter	Patients	Controls	P value
Palmar CMAP amplitude	12.83(4.99)mv	18.80 (3.85)mv	
Wrist-CMAP amplitude	10.95(4.99) mv	17.13(3.78)mv	
Palm/wrist CMAP ratio i.e. distoproximal ratio	2.50(4.19)	1.08(0.11)	P=0.023

Results are reported as mean (standard deviation)
CMAP = Compound muscle action potential
The palm/wrist CMAP ratio exceeded the control mean + 2S.D in 35.5% patients

DISCUSSION

Carpal Tunnel Syndrome (CTS) is the most common, the best defined and the most carefully studied entrapment neuropathy. It is reported to be more common in middle aged females. In the present study 83.87% of the patients were between the ages of 40 to 60 years. In Phalen's[8] series, 58% of the patients were in this age group, while Murthy[4] reported that 74% of the patients were in the fifth and sixth decade.

Female to male ratio was 5.2 : 1. Phalen[8] found this to be 3:1 and in the population based study[2] from Rochester, Minnesota, it was 3.7:1.

The symptoms were bilateral in 48.4% patients. In a study[9] that used computed tomography to measure the cross-sectional area of the bony carpal canals in controls of both sexes and women with idiopathic carpal tunnel syndrome, it was shown that the women controls had significantly (by 25%) smaller carpal canals. The narrowing was bilateral in patients who had unilateral symptoms. There was no correlation between age and the size of the canal which suggested that symptoms arising later in life was due to acquired changes in the walls or contents of the canals in predisposed individuals.

The mean duration of symptoms at presentation was 31.08 months (range 15 days to 144 months). No significant correlation was found between the duration and severity of symptoms or signs. The motor conduction abnormalities were not significantly greater in patients with longer duration of symptoms. DML prolongation was seen as early as 2 weeks after the onset of symptoms. Thomas et al[10] found that patients who had symptoms for only a short time showed abnormalities of motor nerve conduction less

often than those with symptoms of longer duration. However, they observed prolonged latencies as early as one week after the onset of symptoms.

Table 4. CLINICAL FEATURES IN CTS

Symptoms and signs	Stevens[2] et al		Murthy[4] et al	Present study
	n=1016		n=57	n=31(58 hands)
Dull ache or paresthesias in the hand	100%		100%	100%
Proximal radiation of pain	38%			12.9%
Nocturnal awakening	71%		65%	64.5%
	Right	**Left**		
Tinel's sign positive	55%	54%	24.6%	39.7%
Phalen's sign positive	55	52	17.5%	37.9%
APB wasting	20	15	24.6%	22.4%
APB weakness	20	15	45.6%	32.8%

(APB : Abductor Pollicis Brevis)

The differential diagnosis in patients presenting with sensory and motor symptoms in the hand are cervical radiculopathy (especially $C_{6,7}$), brachial plexopathy, proximal median neuropathy (especially at the pronator teres muscle), peripheral neuropathy, ulnar neuropathy, vascular or neurogenic thoracic outlet syndrome and central disorders like multiple sclerosis and cerebral infarction[11,7]. If most of the clinical criteria are satisfied, the diagnosis of CTS is fairly certain.

Provocation of symptoms by sleep and nocturnal awakening due to acroparesthesias is a characteristic symptom of CTS. It was seen in 64.5% of patients in this study, which was similar to the figures obtained by Murthy[4] and Stevens et al[2]. Sunderland[12] who has done extensive studies and reviewed the literature on the nerve lesion in CTS explains this on the basis of diminished return of blood from the limb due to hypotonia and depression of movements during sleep. In a patient with a compromised canal area this would lead to hyperaemia, venous congestion, and circulatory slowing and rise of pressure in the epineurial and intrafunicular tissues[12,13]. The compressed nerve fibres (the large myelinated fibres are more susceptible) become hyperexcitable and start to discharge spontaneously. Shaking or exercising the hand improves the circulation by aiding venous return and reducing the venous congestion at the periphery[12].

Shaking the hand relieved the symptoms in 13 (14.91%) patients. Pryse-Phillips[14] called this the Flick sign and found it more valid and reliable than Phalen's and Tinel's signs. It predicted the electrodiagnostic abnormality in 93% of cases of CTS and had a false positive rate of under 5% among other neural lesions in the arm. "Flicking" increases the thick fibre proprioceptive input, increases venous pressure and volume and moves the nerve underneath the free distal edge of the flexor retinaculam (i.e it 'untethers' the nerve).

Provocative tests were found to be lacking in specificity and of no additional benefit by many authors[15,16]. A high(20%) incidence of Tinel's and Phalen's signs has been reported in normal subjects[21]. We found Tinel's and Phalen's signs positive in 39.7% and 37.9% of hand respectively. Phalen's test in conjunction with nerve conduction studies, by which slowed conduction on wrist flexion was demonstrated, was

found useful by Schwartz et al[17]. They feel that this method may be of use in diagnosis of borderline cases of CTS.

Features of sympathetic dysfunction in the affected hands were seen in 4 (12.9%) patients in the form of dryness, swelling or colour changes of the skin. This is due to involvement of the sympathetic fibres to the hand, carried with the median nerve. Abnormalities of vasomotor reflex of vasoconstriction produced by inspiration have been found in patients with CTS[6].

The associated conditions found with CTS in this study were diabetes in one (3.23%), definite or probable hypothyroidism in 5 (16.13%) and probable cervical spondylosis in one (3.23%). Conditions producing an increase in the volume of contents of the carpal tunnel, which is a closed space, can result in CTS. The most common cause is nonspecific flexor tenosynovitis. The others include rheumatoid arthritis, granulomatous conditions like tuberculosis, sarcoidosis, gout, amyloidosis, pregnancy, endocrine disorders like acromegaly, hypothyroidism, and presence of arteriovenous fistula for haemodialysis[6,4,18].

In patients with hypothyroidism, CTS is seen in about 10-12% and is often bilateral[4].

Body heights and hand lengths were significantly shorter in patients with CTS as compared to controls in a study by Nakamichi and Tachibana[19]. Warner et al[20] found that obese persons were more likely to develop CTS than non- obese individuals. We found that the mean body mass index (BMI) was significantly ($P < 0.002$) greater in

patients with CTS (mean=24.95, S.D = 4.014) when compared with controls (mean BMI = 21.72, S.D = 2.95)

Electrophysiological studies

Nerve conduction studies (NCS) provide a unique and reliable method for assessing directly the integrity of sensory and motor nerve fibres. Electrodiagnostic studies show good sensitivity (60-84%) and excellent specificity (>95%) in the diagnosis of CTS[21].

Median nerve motor conduction studies showed a prolonged distal motor latency (DML) in 48 hands (82.76%) (Table 5). Previous[21] studies have reported that between 60% and 74% of patients with CTS demonstrate a prolonged median motor distal latency. The higher value in our study was probably due to referral bias and the smaller number of patients studied.

Table 5. Median nerve distal motor latency (DML)

Author (Year)	Delean[21] (1988)	Jackson[21] (1989)	Kimura[21] (1979)	Present Study (1996-97)
No. of normal hands (Subject)	80(43)	38(38)	122(61)	40(21)
Normal subjects' age mean (range)	33(2073)	42(21-69)	43(15-60)	44(25-67)
Technique: conduction distance	6-8 cm	8cm	Anatomical land marks	Anatomical land marks
Stimulation site	wrist	wrist	3cm proximal to wrist crease	2cm proximal to wrist crease
Recording site	APB	APB	APB	APB
Median DML±2S.D (in ms)	3.2±0.4	3.18±0.27	3.60±0.36	3.38±0.29
Abnormal value (Mean+2 SD)(MS)	>4.2	>3.71	>4.4	>4.1
No. of CTS hands (patients)	253(150)	131(123)	172(105)	58(31)
CTS patients age mean (range)	47(20-84)	53(21-85)	48(20-78)	46(18-60)
Percentage symptomatic hands with abnormal studies	60%	74%	61%	82.8%

Because cooling of the limb and lengthening the conduction distance also result in prolongation of the DML, it is important that the limb temperature and the conduction distance be controlled.

The motor conduction velocity of the median nerve in the forearm was not significantly different when compared to controls (Table 1). Some of the previous studies

on CTS have reported mild slowing of conduction in the forearm segment[10,21]. This is probably due to retrograde degeneration of the fastest conducting fibres[23].

Stimulation of the median motor fibres in the palm and recording of the conduction velocity in the wrist-palm segment helps verify that the median nerve involvement is localised to the carpal tunnel[24,25,26]. The motor nerve conduction velocity was significantly ($P<0.0001$) slower in the wrist-palm segment, as compared to the elbow-wrist segment (Table 1). Lesser et al[24] have opined that without stimulation distal to the lesion, the pathophysiology of a reduced CMAP or SNAP amplitude cannot be determined; that is, focal demyelination of median nerve fibres resulting in conduction block or pathologic dispersion with phase cancellation cannot be distinguished from axonal degeneration. They found the palm/wrist amplitude ratio to be 1.6 (S.D = 1.3) in CTS patients and 1.0 (S.D = 0.1) in controls. In the present study, we found this ratio in patients to be significantly ($P=0.023$) greater than in controls [2.50 (S.D = 4.19) Vs 1.08 (S.D = 0.11)] (see table 3).

A problem we encountered during palmar stimulation was the stimulus artifact which made measuring the onset latency difficult in some cases.

The CMAP from the palm stimulation may be more than that from the wrist in case of true conduction block, submaximal stimulation of the median nerve at the wrist or in coactivation of the deep ulnar nerve and the recurrent median nerve in the palm[25].

Median sensory NCS confirm the clinical diagnosis of CTS more often than do median motor NCS[21].

Table 6. Median sensory NCS between wrist and digit2

Author (Year)	Jackson[21] (1989)	Kimura[21] (1979)	Present Study (1996-97)
No. of normal hands (Subjects)	38(38)	122(61)	40(21)
Median sensory onset latency±SD (ms)	2.47 ± 0.12	2.82±0.28	2.36±0.26
Abnormal value (mean +2SD)	Onset >2.73 ms	Onset >3.4 ms	Onset >3.0 ms
Percentage of symptomatic hands with abnormal studies	66%	63%	88%

As in previous studies[21], we found that the orthodromic median sensory NCS over a short distance (8cm) in the palm-wrist segment was more sensitive than the wrist to digit antidromic sensory studies. By the latter method, faster conduction in the more distal segments may mask the proximal slowing in the wrist to palm segment in milder cases. The presence of a muscle twitch artifact is also a problem with this technique.

Table 7. Median sensory and mixed NCS between wrist and palm in CTS

Author (Year)	Jackson[21] (1989)	Present Study
No. of normal hands (subjects)	38(38)	40(21)
Technique: conduction distance	8cm	8cm
Stimulation site	Palm	Palm
Recording site	Wrist	Wrist
Median sensory onset latency± 2SD(normal)	Not reported	1.64±0.14 ms
Abnormal value (mean + 2SD)	Onset>1.78 ms	>2.0 ms
Percentage of symptomatic hands with abnormal studies	69%	94.8%

The orthodromic SNAP onset latency difference between analogous 8 cm lengths of median and ulnar nerves in the palm wrist segment was abnormal in 94.8% of hands in this study. The AAEM quality assurance committee[21] has remarked that if over 90% of the patients with a clinical diagnosis of CTS in a study demonstrate a test abnormality, the results suggest that the patient population was biased with patients with advanced CTS. Since ours is a tertiary referral centre and 2 other electrophysiology labs are there in the same locality, it is possible that patients with milder involvement were not referred to us. Study population also was small in number.

Table 8. Comparison of median and ulnar mixed nerve sensory conduction between wrist and palm in CTS

Author (Year)	Jackson[21] (1989)	Present Study (1996-1997)
Technique: conduction distance	8cm	8cm
Stimulation site	Palm	Palm
Recording site	Wrist	Wrist
OSLD±SDms in controls	0.08±0.12	0.07±0.13
Abnormal value (Mean + 2SD)	Onset >0.32ms	Onset > = 0.4
No. of CTS hands (patients)	131(123)	58(31)
Percentage of symptomatic hands with abnormal studies	66%	94.82%

(OSLD: Orthodromic SNAP latency difference between median and ulnar nerves in the palm wrist segment.)

Techniques which compare sensory or mixed nerve conduction of the median nerve through the carpal tunnel to sensory or mixed nerve conduction of the ulnar nerve or radial nerve in the same hand have been found to be more sensitive than antidromic sensory NCS from wrist to digit. This will eliminate the biologic variation in speed of nerve conductions from person to person due to age and genetic difference and also other confounding factors like limb temperature variation[7,21,27].

Comparison of various nerve conduction study techniques was done by Seror[28], who found that the incremental orthodromic sensory conduction study based on Kimura's[29] and Nathan's[30] methods was the most sensitive. This inching technique is time consuming. Another problem with this stimulation technique was the presence of

stimulus artifact, which was overcome by Kimura using a fast recovery amplifier. Padua et al[31], after comparing various methods, concluded that the ratio of distal to proximal conduction from digit 3 was the most sensitive.

An interesting observation in the present study was the prolongation of the distal motor latency in the median nerve as the only abnormality in 3 symptomatic hands. The normal conduction in ulnar nerves and the normal distal median sensory latencies excluded other confounding factors like a generalized motor neuropathy or low limb temperature. Rarely CTS may selectively involve motor fibres as the motor branch of the median nerve exits the carpal tunnel in a separate channel[25]

Difference in opinion exists regarding the choice of the digit most suitable for sensory conduction studies. Kothari et al[32] found that digit 1 was the most sensitive in milder cases of CTS. They explain it on the basis of fascicular arrangment[12] of the cutaneous sensory fibres within the median nerve at the wrist, where the sensory fibres from Digits 1,4 and medial side of 3 lie more anteriorly, while the sensory fibres from digit 2 lie posteriorly. A similar situation exists for the occurrence of relative lumbrical sparing in CTS[23], as these fibres are located more centrally in the nerve.

In conclusion, we feel that orthodromic sensory latency comparison in the palm-wrist segment between equal lengths of the median and ulnar nerves is highly sensitive in detecting the electrophysiological abnormality in patients with CTS and that palmar stimulation of the motor fibres is useful in deciding the nature of the lesion at the carpal tunnel. This can influence the therapeutic decisions and prognostication. A demyelinating nerve injury as suggested by conduction block across the wrist implies a good prognosis after carpal tunnel decompression. Confirmation of this can be done only by a study undertaking long term follow-up of the patients postoperatively.

References

1. Summary statement - American Academy of Neurology, American Association of Electrodiagnostic Medicine, and American Academy of Physical Medicine and Rehabilitation : Practice parameter for electrodiagnostic studies in carpal tunnel syndrome . Neurology 1993; 43 : 2404-2405

2. Stevens JC, Sun S, Beard CM, O'Fallon WM, and Kurland LT. Carpal tunnel syndrome in Rochester, Minnesota, 1961 to 1980. Neurology 1988; 38 : 134-138

3. Center for disease control. Occupational Disease Surveillance : Carpal Tunnel Syndrome. J Am Med Assoc 1989; 262 : 886, 889.

4. Murthy JMK Carpal Tunnel Syndrome. In : Reviews in Neurology. Murthy JMK(on behalf of Indian Academy of Neurology) Hyderabad. 1995; Vol 2 : 209-219.

5. Gourie Devi M. Some aspects of leprous neuropathy. In: Molecular Mechanisms underlying Neural Damage eds Rao BSS and Body SC, NIMHANS Publication, Bangalore 1990; 1392-1414

6. Dawson DM, Hallet M, Millender LH. Carpal tunnel syndrome. In: Entrapment neuropathies . 2nd ed. Boston: Little, Brown, 1990; 25-92.

7. Uncini A, Di Muzio A, Awad J, Manente G, Tafuro M and Gambi D. Sensitivity of three median-to-ulnar comparative tests in diagnosis of mild carpal tunnel syndrome. Muscle Nerve 1993; 16 : 1366-1373

8. Phalen AS. Reflection on 21 years' experience with the carpal tunnel syndrome. J Am Med Assoc 1970; 212 : 1365-1367

9. Dekel S, Papaioannou T, Rushworth G and Coates R. Idoiopathic carpal tunnel syndrome caused by carpal stenosis. Br Med J 1980; 280 : 1397 - 1299

10. Thomas JE, Lambert EH and Cseuz KA. Electrodiagnostic aspects of the Carpal Tunnel Syndrome. Arch Neurol 1967; 16 : 635-641

11. Report of the Quality Standards Subcommittee of the American Academy of Neurology. Practice parameter for carpal tunnel syndrome (summary statement) Neurology 1993; 43 : 2406-2409

12. Sunderland S. The nerve lesion in the carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 1976; 39 : 615-626

13. Anonymous. Diagnosis of the carpal tunnel syndrome (Editorial). The Lancet 1985; 1 : 854-855

14. Pryse-Phillips WEM. Validation of a diagnostic sign in carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 1984; 47 : 870-872

17. Schwartz MS, Gordon JA, Swash M. Slowed nerve conduction with wrist flexion in carpal tunnel syndrome. Ann Neurol 1980; 8 : 69-71

18. Murthy JMK, Meena AK. Entrapment neuropathies JAPI 1995; 43 : 673-675

19. Nakamichi K, Tachibana S. Small hand as a risk factor for idiopathic carpal tunnel syndrome. Muscle Nerve 1995; 18 : 664-666

20. Werner RA, Albers JW, Franzblau A, Armstrong TJ. The relationship between body mass index and the diagnosis of carpal tunnel syndrome Muscle Nerve 1994; 17 : 632-636

21. Jablecki CK, Andary MT, So YT, Wilkins DE, Williams FH. (AAEM Quality Assurance Committee). Literature review of the usefulness of nerve conduction studies and electromyography for the evaluation of patients with carpal tunnel syndrome. Muscle Nerve 1993; 16 : 1392-1414

22. Wilson JR, Sumner AJ. Immediate surgery is the treatment of choice for carpal tunnel syndrome. Muscle Nerve 1995; 18 : 660-662

23. Logigian EL, Busis NA, Berger AR et al. Lumbrical sparing in carpal tunnel syndrome. Neurology 1987; 37 : 1499-1505

24. Lesser EA, Venkatesh S, Preston DC and Logigian EL. Stimulation distal to the lesion in patients with carpal tunnel syndrome. Muscle Nerve 1995; 18 : 503-507

25. Ross MA, Kimura J, AAEM case report #2: The carpal tunnel syndrome. Muscle Nerve 1995; 18 : 567-573

26. Daube JR. Percutaneous palmar median nerve stimulation for carpal tunnel syndrome. Abstract. Electroencephalogr Clin Neurophysiol 1977; 43 : 139-140.

27. Carroll GJ. Comparison of median and radial nerve sensory latencies in the electrophysiological diagnosis of carpal tunnel syndrome. Electroencephalogr Clin Neurophysiol 1987; 68 : 101-106

28. Seror P. Sensitivity of various electrophysiological studies for the diagnosis of carpal tunnel syndrome. Muscle Nerve 1993; 16 : 1418-1419

29. Kimura J. The carpal tunnel syndrome: localization of conduction abnormalities within the distal segment of the median nerve. Brain 1979; 102 : 619-635

30. Nathan PA, Keniston RC, Meadows KD and Lockwood RS. Predictive value of nerve conduction measurements at the carpal tunnel. Muscle Nerve 1993; 16 : 1377-1382

31. Padua L, Monaco ML, Valente EM and Tonali PA. A useful electrophysiologic parameter for diagnosis of carpal tunnel syndrome. Muscle Nerve 1996; 19:48-53

32. Kothari MJ, Rutkove SB, Caress JB, Hinchey J, Logigian EL and Preston DC. Comparison of digital sensory studies in patients with carpal tunnel syndrome. Muscle Nerve 1995; 18 : 1272-1276

CARPAL TUNNEL SYNDROME - A PROSPECTIVE STUDY

Investigators: Dr.Joseph Cherian.P., Dr. Abraham Kuruvilla,
Prof. K.Radhakrishnan.

PROFORMA

Name ______________________
Address____________________
____

Date of diagnosis at SCTIMST_____________ Occupation----------

CARD 1
COLUMNS CODE ITEM

1--7 _ _ _ _ _ _ _ Hospital number
8-9 _ _ Age at registration
10 _ Gender: Male = 1, Female =2 .
11 _ Income group: A=1, B1=2, B=3, C=4, D=5 .

12-13 _ _ Age in years at the onset of symptoms.
14-15 _ _ Duration of symptoms in months
16 _ Handedness.R=1, L=2,Amphidextrous=3

HISTORY

SYMPTOMS
----

17 _ Dull aching discomfort in the hand, forearm or upper arm.No=1,Yes,R=2,L=3,B/L=4.
18 _ Paresthesia in the hand.No=1,Yes,R=2, L=3,B/L=4 .
19 _ Weakness or clumsiness of the hand. No=1,Yes,R=2,L=3,B/L=4.
20 _ Dry skin, swelling or colour changes in the hand.No=1,Yes,R=2,L=3,B/L=4 .
21 _ Occurrence of any of these symptoms in the medianN.distribution.No=1,Yes,R=2,L=3,B/L=4

Provocative factors.
22 _ Sleep. Yes=1, No=2 .
23 _ Sustained arm or hand posture. Yes=1, No=2.
24 _ Repititive actions of the hand or wrist Yes = 1, No = 2 .

Relieving factors.

25 _ Changes in hand posture. Yes =1, No=2 .

26 _ Shaking the hand. Yes=1, No=2 .

Associated conditions.If yes, specify.

27 _ H/o diabetes mellitus.Yes=1, No=2 .

28 _ H/s/o rheumatoid arthritis.Yes=1, No=2.

29 _ H/s/o hypothyroidism. Yes=1, No=2 .

30 _ Pregnancy. Yes=1, No=2 .

31 _ Pain in the neck, radiating to shoulder and upper limb. Yes=1, No=2 .

32 _ Presence of A-V fistula(shunt)No=1,Yes,R=2 Yes, L=3.

PHYSICAL EXAMINATION

33 _ _ _ Height(cm)

34 _ _ Weight (kg)

35 _ _ Body mass index.

36 _ Thyroid swelling. Yes=1, No=2 .

37 _ Pedal oedma. Yes=1, No=2 .

38 _ Acromegalic features. Yes=1, No=2 .

39 _ Pulse-bradycardia.Yes=1, No=2 .

40 _ Swelling, deformity of small joints of hands and feet.Yes=1, No=2 .

41 _ Deep tendon reflexes present normally. Yes=1, No=2 . (if no, specify_____________)

42 _ Slow relaxing ankle jerks. Yes=1, No=2 .

43 _ Dry skin over the thumb, index and middle fingers.No=1,Yes,R=2,Yes,L=3,Bilat=4.

44 _ Atrophy in the thenar muscles.No=1, Yes,R=2, Yes,L=3, Bilaateral=4

45 _ Weakness of thenar muscles. No=1,Yes,R=2 , Yes,L=3, Bilateral=4.

46 _ Sensory loss in the median nerve distribution.No=1,Yes,R=2, Yes,L=3, Bilateral=4.

47 _ Positive Tinel's sign.No=1, Yes,R=2, Yes,L=3, Bilateral=4.

48 _ Positive Phalen's sign. No=1,Yes,R=2, Yes,L=3, Bilateral=4.

CARD 2

ELECTROPHYSIOLOGICAL TESTS

Motor nerve conduction studies

NERVE & SITE	Lat(ms)	Amp(mV)	Segment	C.V(m/s)
Median nerve R				
Palm				
Wrist				
Elbow				
Median nerve L				
Palm				
Wrist				
Elbow				
Ulnar nerve R				
Wrist				
Below elbow				
Ulnar nerve L				
Wrist				
Below elbow				
Common peroneal nerve				
Ankle				
Below fib.head				

F-waves

	Latency(ms)
Median nerve R	
Ulnar nerve R	
Median nerve L	
Ulnar nerve L	
Peroneal nerve R	

Sensory nerve conduction studies.

Antidromic conduction from wrist

	Med R	Uln R	Med L	Uln L
Latency(ms)				
Amplitude(micro V)				
Segment				

Orthodromic conduction from palm (palm- wrist segment.)

	Med R	Uln R	Diff	Med L	Uln L	Diff
Latency (ms)						
Amplitude(micro V)						

	Amp (micro V)	Lat (ms)
Sural nerve conduction		

EMG:

				MUP			MVA		
	Fibs	+W	Fas	Poly	Amp	Dur	Amp	Pattern	Effort
APB									
FDI									
FCR									