

# CARPAL TUNNEL SYNDROME DESPITE NEGATIVE NEUROPHYSIOLOGICAL STUDIES

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**The purpose of the present study was to compare the results of conservative and operative treatment for patients with carpal tunnel syndrome having normal neurophysiological studies.**

**We studied 125 patients with normal neurophysiological studies and analysed eight symptoms and signs as “prognostic factors”. Ninety-six patients were treated conservatively (splintage, steroid injection, antiinflammatory medications, activity modification) and 29 were treated surgically (open decompression). One year after initiation of treatment we assessed the outcome and statistically analysed (chi-square test) the differences between the two groups. We did not find any statistically significant correlation between “prognostic factors” and outcome. Twenty four percent of the group treated non-operatively had a good or excellent outcome, whereas 90% of the group treated operatively had a good or excellent outcome. This difference was statistically significant ( $p < 0.0001$ ).**

**Our study supports the view that the diagnosis of carpal tunnel syndrome is clinical and not neurophysiological. We now recommend operative treatment for these patients.**

**Keywords :** carpal tunnel syndrome ; nerve conduction studies ; neurophysiology.

**Mots-clés :** syndrome du tunnel carpien ; tests de conduction nerveuse ; neurophysiologie.

## INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common and well-described nerve entrapment neuropathy in the upper limb (12). Despite the large amount of literature about CTS, several issues

remain unanswered. Controversy exists with regard to the most reliable test that is available to diagnose the condition. No gold standard exists for the diagnosis of CTS (15). In addition, no consensus has been reached regarding the diagnosis and management of individuals presenting with classic or probable symptoms and signs of CTS in combination with negative nerve conduction studies.

We reviewed a group of patients who had symptoms and signs suggestive of carpal tunnel syndrome and negative electrodiagnostic studies. The purpose of our study was to examine the outcome of these specific cases depending on the treatment given (conservative or operative) and to attempt to correlate their initial symptoms and signs with the final outcome.

## PATIENTS AND METHODS

All the patients referred from the Orthopaedic Department of Coventry and Warwickshire Hospital for nerve conduction studies between 1995 and 2000 were identified from a database. These patients had a provisional diagnosis of CTS based on clinical findings. All cases with positive nerve conduction studies were excluded. Patients who had undergone prior carpal tunnel decompression in the same hand, cervical radiculopathy,

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thoracic outlet syndrome or other entrapment neuropathies in the upper limb were also excluded.

Information was collected from a series of 157 patients who had negative nerve conduction studies.

We recorded eight clinical features suggestive of CTS.

1. Dysesthesiae (pain and/or tingling) in the median nerve distribution.
2. Night symptoms relieved by shaking of the hand.
3. Subjective weakness or loss of dexterity.
4. Radiation of symptoms proximally.
5. Decreased sensation in the distribution of the median nerve.
6. Phalen test.
7. Tinel test.
8. Objective weakness of abductor pollicis brevis muscle or thenar muscle atrophy.

The clinical diagnosis was made according to Harrington's criteria (11) drawn together by a wide variety group of specialists. The group concluded the surveillance criteria for carpal tunnel syndrome to be pain or paresthesia or sensory loss in the median nerve distribution and one of the following : positive Tinel's test, nocturnal exacerbation of symptoms, motor loss with wasting of abductor pollicis brevis or abnormal nerve conduction times. Additional features included : no signs and symptoms in the little finger or dorsum of the hand, no other cause apparent and successful steroid injection or surgery.

Electrodiagnostic examination was carried out using the recognised standard neurophysiological guidelines in the UK. The criteria for conventional diagnosis for CTS were :

1. Sensory action potential (SAP) : median peak latency > ulnar by 1 msec
2. Distal motor latency (DML) : median > ulnar by 1.5 msec
3. Palm-wrist technique : median peak latency > ulnar by 0.3 msec

The same neurophysiology department using standard diagnostic criteria performed all the electrodiagnostic studies in our series.

The decision to proceed to operative or non-operative treatment was made on an individual basis after a consensus was reached between the surgeon and the patient. Following the nerve conduction studies the patients were reviewed in the clinic. The findings and the situation were explained to them and the treatment options were discussed. It was made clear that undertaking an operative treatment did not guarantee a satisfactory

result and the patients were left free to decide on what type of treatment they preferred.

Non-operative treatment modalities included non-steroid anti-inflammatory drugs, splintage, physiotherapy, modification of activities, steroid injection or combinations of the above.

Operative treatment was carried out under local anaesthesia. A standard median nerve release was performed with the open technique and signs of nerve compression (hourglass deformity as well as purple discoloration of the median nerve following decompression and tourniquet release) were recorded.

Follow-up data were obtained 12 months after the initial assessment in both the non-operatively and operatively managed groups. The final outcome was considered to be excellent if the patient was completely asymptomatic, good if there was a significant improvement in symptoms, fair if there was no significant change in symptoms and poor if the symptoms were worse.

The data were statistically analysed by Chi-square contingency test including continuity adjustment and by Fischer's Exact test.

## RESULTS

At the time of follow-up the data were incomplete for 22 patients (three managed surgically). Ten patients did not attend the clinic following their initial treatment (one managed surgically). Of the remaining 125 patients (male : 35, female : 90), 96 were treated conservatively (male : 29, female : 67) and the other 29 were treated surgically (male : 6, female : 23). The mean age of the conservatively treated group was 45 years. In the operatively managed group, the mean age was 48 years. The mean duration of symptoms prior to initiation of treatment was nine months, with a minimum of six months. The symptoms and clinical finding distribution as well as the treatment outcomes are presented in table I.

There was no evidence that the two groups were statistically different with regard to age ( $p = 0.71$ ) and sex ( $p = 0.267$ ) as analysed by Chi-square test.

The outcome of the conservatively managed group was good or excellent in 24% of the cases (23 of 96 cases). Good or excellent results were achieved in 90% of the operatively managed cases (26 of 29 cases).

Table I. — Analysis of the symptoms and signs of CTS and outcomes for the non-operatively and operatively treated groups

	Conservative treatment (n = 96)	Surgical treatment (n = 29)
Dysesthesias in median nerve distribution	95 (99%)	29 (100%)
Nocturnal symptom exacerbation	74 (77%)	25 (86%)
Subjective weakness, loss of dexterity	34 (35%)	8 (28%)
Proximal radiation of symptoms	21 (22%)	6 (21%)
Altered sensation in median nerve	33 (34%)	16 (55%)
Phalen test	32 (33%)	16 (55%)
Tinel test	19 (20%)	6 (21%)
Abductor Pollicis Brevis weakness or thenar muscle atrophy	12 (13%)	5 (17%)
Excellent outcome	1 (1%)	19 (66%)
Good outcome	22 (23%)	7 (24%)
Fair outcome	41 (43%)	2 (7%)
Poor outcome	31 (32%)	1 (3%)

Statistical analysis by Pearson Chi-squared test demonstrated a significant difference in outcome between the two groups ( $p < 0.0001$ ). The very small number of surgically treated patients having poor or fair results prevented meaningful statistical analysis by Fisher's Exact test of "prognostic factors" and "outcome" between the two groups.

## DISCUSSION

CTS is a constellation of symptoms and signs that results from compression of the median nerve within the carpal tunnel (19). The syndrome has an incidence of 0.1% to 1.5% in the general population (17, 20) and 1% to 10% in high-risk occupations (7). The mean age at diagnosis is 50 years (18). As with industrial low back pain, it is best to pursue non-surgical management if specific objective evidence of a compression neuropathy is lacking (18).

Many authors believe that CTS is a clinical diagnosis and nerve conduction studies do not contribute significantly to the diagnosis. (6, 8, 9, 10). Others support the idea that the most objective confirmation of median nerve abnormality within the carpal tunnel is the electrodiagnostic evidence (3, 12, 13, 21). In addition, it has been suggested that typical clinical features and positive provocation tests are not sufficient to lead a surgeon to decompress the carpal tunnel and electrodiagnostic confirmation is necessary in every case (1, 5).

We routinely use nerve conduction studies as part of the diagnostic work-up of suspected CTS for a variety of reasons. Electrodiagnostic studies are useful as a baseline if there is no postoperative improvement. They can reveal peripheral neuropathy or multiple nerve compression levels. We also consider these studies to be extremely helpful for cases with unclear clinical features. Finally, neurophysiological evidence may be helpful when medicolegal issues occur.

The two groups were comparable in terms of age, sex and duration of symptoms. The duration of symptoms appears to be a controversial determinant of outcome. Semple and Cargill illustrated that the best results of carpal tunnel release occurred in patients whose symptoms were of short duration (16). If the duration was less than six months the failure rate was 3% and a failure rate of 25% or more was noted if symptoms exceeded six months. In contrast, Choi and Ahn found that there was no correlation between the duration of symptoms and the outcome of surgical treatment (2). All our patients had a minimum duration of symptoms of six months.

The patients in our study fulfilled the Harrington diagnostic criteria (11). Nocturnal exacerbation of pain and/or dysaesthesia in the median nerve distribution was the predominant symptom in both groups (77-86 %). Less frequently the patients complained of (subjective) weakness and loss of

dexterity (35-28%) and proximal radiation of symptoms (21-22%).

On clinical examination the most frequently observed signs were the altered sensation in the median nerve distribution (34-55%) and the positive Phalen test (33-55%). Only 20% of the patients had a positive Tinel test. The relative absence of this sign does not come as a surprise. The majority of the patients studied were experiencing the early stages of the syndrome. This observation is in agreement with the staging of CTS as described by Novak and MacKinnon (14). Provocation tests (Phalen's, Reverse Phalen's, Durkan's, McMurtry's tests) may be positive in the early stages of the disease. Tinel's sign becomes positive in more advanced stages, usually with muscle weakness and abnormal two-point discrimination, when focal segmental demyelination through wallerian degeneration progresses to regeneration of fibers.

The earliest manifestation of low-grade peripheral nerve compression is reduced epineurial blood flow at 20-30 mmHg. When the pressure increases to 30 mmHg the axonal transport becomes impaired. With extended pressure at this level endoneurial fluid pressure increases and between 30 and 40 mmHg neurophysiological changes occur. Higher pressures cause epineurial edema and axonal transport block and at 60 mmHg complete intraneural ischemia and motor and sensory block ensues (18). We suspect that the majority of our patients had pressures between 20 and 40 mmHg, which were significant enough to cause symptoms, but no abnormal neurophysiological findings.

Operative treatment based on clinical presentation either without neurophysiological investigation or for patients with normal nerve conduction studies has been endorsed (6,9). In addition, there is great variability in the adherence of nerve conduction studies to the practice standards and guidelines (4). From our previous experience (based on clinical audit), the results of operative treatment in presence of positive nerve conduction studies are good or excellent in approximately 95% of the cases. A recent study of the value of neurophysiology for typical CTS (6) produced similar results (92.6% good or excellent outcome). These results are comparable with the outcomes in this study

where the nerve conduction studies were normal (good or excellent results in 90% of cases in the operatively treated group).

The conservative treatment in this group of patients has not produced many satisfactory results. Night splintage in combination with a steroid injection in the carpal tunnel provided temporary relief in a small number of patients. The symptoms recurred in all of them within 9 months from the injection. It is worth mentioning that a steroid injection into the carpal tunnel can be a helpful diagnostic manoeuvre in patients with atypical features. Relief from symptoms following the injection, even temporary, confirms the diagnosis. However, only experienced doctors should carry out this procedure, because inadvertent steroid injection into the median nerve may have detrimental and permanent effects.

We are not aware of any studies comparing the outcome of conservative and operative treatment in this subgroup of patients with normal neurophysiological studies. Our results support the view that CTS is a clinical diagnosis. Many patients present with symptoms of CTS and median nerve irritation well before electrodiagnostic evidence appears. There is a distinct value in nerve conduction studies as well as in clinical examination. Both are invaluable in our diagnostic armamentarium. In a world of cost efficiency and managed care it would be fair to say that nerve conduction studies significantly increase the cost of treatment and may prolong the waiting time for the operation. Consequently, one might consider them unnecessary in cases with typical features of CTS. However, within the present climate of litigation, the nerve conduction studies are increasingly being used for purposes of documentation. We establish our diagnosis on clinical grounds supported, if possible, by positive nerve conduction studies, not the reverse. Consequently, we have no hesitation in offering operative treatment to these patients, provided a clear clinical diagnosis is made following meticulous history taking and clinical evaluation.

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## REFERENCES

1. Buch-Jaeger N., Foucher G. Correlation of clinical signs with nerve conduction tests in the diagnosis of carpal tunnel syndrome. *J. Hand Surg.*, 1994, 19-B, 720-724.
2. Choi S. J., Ahn D. S. Correlation of clinical history and electrodiagnostic abnormalities with outcome after surgery for carpal tunnel syndrome. *Plast. and Reconstr. Surg.*, 1998, 102, 2374-2380.
3. Corwin H. M. Relation of preoperative nerve conduction values to outcome in workers with surgically treated carpal tunnel syndrome. *J. Hand Surg.* 1998, 23-A, 354-355.
4. Corwin H. M., Kasdan M. L. Electrodiagnostic reports of median neuropathy at the wrist. *J. Hand Surg.*, 1998, 23-A, 55-57.
5. De Krom M. C. T. F. M., Knipschild P. G., Kester A. D. M., Spaans F. Efficacy of provocative tests for diagnosis of carpal tunnel syndrome. *Lancet*, 1990, 335, 393-395.
6. Finsen V., Russwurm H. Neurophysiology not required before surgery for typical carpal tunnel syndrome. *J. Hand Surg.*, 2001, 26-B, 61-64.
7. Franklin G. M., Hang J., Heyer N., Checkoway H., Peck N. Occupational carpal tunnel syndrome in Washington State, 1984-1988. *Am. J. Public Health*, 1991, 81, 741-746.
8. Gonzalez Del Pino J., DelgadoMartinez A. D., Gonzalez I., Lovic A. Value of the carpal compression test in the diagnosis of carpal tunnel syndrome. *J. Hand Surg.*, 1997, 22-B, 38-41.
9. Grundberg A. B. Carpal tunnel decompression in spite of normal electromyography. *J. Hand Surg.*, 1983, 8A, 348-349.
10. Gunnarson L. G., Amilon A., Hellstrand P., Leissner P., Philipson L. The diagnosis of carpal tunnel syndrome. *J. Hand Surg.*, 1997, 22-B, 34-37.
11. Harrington J. M., Carter J. T., Birrell L., Gompertz D. Surveillance case definitions for work related upper limb pain syndromes. *Occupational and Environmental Medicine*, 1998, 55, 264-271.
12. Jablecki C. K., Andary M. T., So Y. T., Wilkins D. E., Williams F. H. (1993). Literature review of the usefulness of nerve conduction studies and electromyography for the evaluation of patients with carpal tunnel syndrome. *Muscle and Nerve*, 1993, 16, 1392-1414.
13. Johnson E. W. Diagnosis of carpal tunnel syndrome. The gold standard. (Editorial). *Am. J. Phys. Med. Rehabil.*, 1993, 72, 1.
14. Novak C. B., Mackinnon S. E., Brownlee R., Kelly L. Provocative sensory testing in carpal tunnel syndrome. *J. Hand Surg.*, 1992, 17-B, 204-208.
15. Rempel D., Evanoff B., Amadio P. C., de Krom M., Franklin G., Franzblau A. *et al.* Consensus criteria for classification of carpal tunnel syndrome in epidemiological studies. *Am. J. Public Health*, 1998, 88, 1447-1451.
16. Semple J. C., Cargill A. O. Carpal tunnel syndrome. Results of surgical decompression. *Lancet* 1, 1969, 918-919.
17. Stevens J. C. Sun S., Beard C. M., O'Fallon W. M., Kurland L. T. Carpal tunnel syndrome in Rochester, Minnesota 1961 to 1980. *Neurology*, 1988, 38, 134-138.
18. Szabo R. M. in *Hand Surgery Update 2*. Editor: Terry R Light American Academy of Orthopaedic Surgeons, Rosemont Illinois, 1999, pp. 183-195.
19. Szabo R. M., Slater R. R., Farver T. B., Stanton D. B., Sharman W. The value of diagnostic testing in carpal tunnel syndrome. *J. Hand Surg.*, 1999, 24-A, 704-714.
20. Tanaka S., Wild D. K., Seligman P. J., Behrens V., Cameron L., Putz-Anderson V. The US prevalence of self-reported carpal tunnel syndrome: 1988 National Health Interview Survey data. *Am. J. Public Health*, 1994, 84, 1846-1848.
21. Vender M. I., Kasdan M. L., Truppa K. L. Upper extremity disorders: a literature review to determine work-relatedness. *J. Hand Surg.*, 1995, 20-A, 534-541.

## SAMENVATTING

C. K. KITSIS, O. SAVVIDOU, A. ALAM, R. J. CHERRY. *Polskanaalsyndroom zonder electromyografische afwijkingen.*

Deze studie vergelijkt de uitslag van conservatieve en heelkundige behandeling van patiënten met een klinisch polskanaalsyndroom zonder neurofysiologische afwijkingen. De prognostische waarde van 8 klinische tekens en symptomen werd geëvalueerd bij 125 patiënten met een klinisch polskanaal met normale electrodiagnostische verschijnselen. Zesennegentig patiënten werden conservatief behandeld: NSAID, immobilisatie, physio, aanpassen van activiteiten, en steroid infiltratie en 29 werden heelkundig behandeld (open decompressie). De resultaten werden geëvalueerd één jaar na het begin van de behandeling en het verschil tussen beide groepen werd statistisch geanalyseerd (chi-square test). Wij vonden geen statistisch significant verband tussen de prognostische factoren en het resultaat. Bij de conservatief behandelde patiënten waren 21% uitstekend of goed, bij de heelkundig behandelde daarentegen 90% (statistisch significant:  $p < 0.0001$ ). Onze studie ondersteunt de

stelling dat de diagnosis "polskanaalsyndroom" een klinische diagnosis en geen elektrische. Wij raden nu heelkunde aan.

### RÉSUMÉ

C. K. KITSIS, O. SAVVIDOU, A. ALAM, R. J. CHERRY. *Syndrome du tunnel carpien sans anomalie à l'examen neurophysiologique.*

Les auteurs ont entrepris dans ce travail de comparer les résultats du traitement conservateur et du traitement chirurgical chez des patients qui présentaient un syndrome du tunnel carpien, sans anomalie à l'examen neurophysiologique. Ils ont étudié 125 patients dont les examens neurophysiologiques étaient normaux et ils ont évalué la valeur pronostique de 8 symptômes et signes cliniques.

Quatre-vingt-seize patients ont été soumis au traitement conservateur (immobilisation sur attelle, injection de corticostéroïdes, anti-inflammatoires, modification de l'activité) et 29 ont été traités chirurgicalement par dé-compression du nerf. Le résultat a été évalué à un an du début du traitement et les différences relevées entre les deux groupes ont fait l'objet d'une analyse statistique (test du Chi-carré). Les auteurs n'ont relevé aucune corrélation statistiquement significative entre les «facteurs pronostiques» et le résultat. Dans le groupe soumis au traitement conservateur, 24% des patients avaient un résultat bon ou excellent, contre 90% dans le groupe traité chirurgicalement. Cette différence était statistiquement significative ( $p < 0,0001$ ). Cette étude vient conforter l'opinion selon laquelle le diagnostic du syndrome du tunnel carpien est un diagnostic clinique et non neurophysiologique. Les auteurs recommandent actuellement le traitement chirurgical pour ces patients.