Acta Neurochir (2005) [Suppl] 92: 47–52 © Springer-Verlag 2005 Printed in Austria

Wrist median nerve motor conduction after end range repeated flexion and extension passive movements in Carpal Tunnel Syndrome. Pilot study

A. Zalaffi, A. Mariottini, B. Carangelo, J. Buric, V. F. Muzii, A. Alexandre, L. Palma, and A. Rovere

Departement of Ophthalmological and Neurosurgical Science, Siena University, Siena, Italy

Summary

Carpal Tunnel Syndrome (CTS) can be due to a variety of different pathological conditions. These etiological and epidemiological differences may explain the non-homogeneous response to ordinary conservative therapeutical options observed in this syndrome. The aim of our study was to investigate on the possibility of identifying different sub-groups of patients among conservatively treatable CTS with different susceptibility to physiotherapeutic treatments. We decided to utilize an objective approach measuring some median motor nerve function parameters.

Short term variations of Compound Motor Action Potential (CMAP) from the thenar eminence were compared in two groups of 55 hands (CTS patients and normal controls) after performance of two different types of end range passive movement.

We found a different distribution of CMAP amplitude modifications within a sub-group of patients that suddenly improved more than the controls after two series of 10 end range passive flexions or after two series of ten end range passive extensions.

Amplitude changes proved to be much more useful than latency variation studies in the provocative test neurophysiological approach. The method we propose appears to be useful for better surgical indication and/or for improvement of conservative therapeutic choice.

Keywords: Carpal Tunnel Syndrome; compound motor action potentials; electromyography; physiotherapy; provocative tests.

Introduction

A variety of conditions may cause carpal tunnel syndrome (CTS) by increasing volume of tissue within the carpal tunnel and/or decreasing of the section area of carpal canal. CTS is associated in fact with inflammatory arthritis, Colles' fracture, amyloidosis, Kienboeck's disease, change of hormonal balance (pregnancy, diabetes, ipotiroidism, steroid or estrogens therapy) [6, 16] and with repetitive and forceful activities that cause thickening of the synovial lining of the tendons that traverse the carpal tunnel along with the median nerve [21]. Controversy still exists regarding the pathophysiology, assessment, diagnosis and treatment of CTS [14, 20]. In a multiperspective, multicentre follow-up study on untreated CTS published in 2001 several affected hands unexpectedly improved spontaneously. This study also showed that cases with initial low severity might tend to get worse, whereas severe patterns may improve [11].

Because of the high prevalence of CTS (reported to be between 2,7% and 5,8% of the general population) [3, 9] accurate diagnosis and effective treatment are important to physician, therapist, employers, and third-party payers.

Especially useful are those criteria that may help to predict the natural clinical course of the disease (toward spontaneous improvement or not) and to select the best individual treatment. In fact, in some cases an earlier surgical decision might save time, pain, further examination, working days and finally money.

The solution of this clinical enigma is often based on a long or short period of conservative treatment with a monitoring of symptoms and signs and sometime neurophysiological parameters.

Several conservative treatments may be used for this purpose: NSAD, Steroids (oral or infiltrative), physical therapy (ultrasound, ionophoresis), immobilization, mobilization, change of daily activities (ergonomical interventions, work changes), tendon and nerve gliding exercises, general conditioning therapies such as yoga or stretching.

Data concerning usefulness of these treatment methods aren't satisfactory: in fact only oral steroids, splinting, ultrasound, yoga and carpal bone mobilization show real short-term benefit. The other nonsurgical treatments don't seem to produce improvements according to the 2002 Cochrane Review which analysed the efficacy of non-surgical treatment (steroid injection excluded) for CTS [10].

Especifically regarding nerve and tendon gliding exercises it has been shown that neurodynamic mobilization doesn't change significantly the symptoms while carpal bone mobilization causes a short term improvement without any statistical difference between the two methods [18].

Moreover, according to other authors there is a tendency towards a decrease in surgical operations (only 57%) in patients who underwent tendon mobilization as opposed to patients that received only conventional treatment (71% surgery) [15, 19].

Akalin prospectively analysed a randomized group of 28 patients treated with splinting alone versus splinting and tendon and nerve mobilization and found better outcomes in the second group but with no statistical difference [2].

Our impression is that the lack of any statistical significance reported in the literature concerning the usefulness of methods that logically would appear effective may be due to a bias in patient selection. In our experience, there are in fact several sub-group of patients that may show a different clinical evolution pattern based on the treatment of tendon and nerve mobilization.

To verify our hypothesis we used a simple neurophysiological method: the study of compound motor action potential (CMAP). Our method evaluates the short-term variations of this parameter following two different types of passive movement and compares these variations in patients and controls. Our aim was to check the possibility of finding a few easy markers correlated with a different sensitivity to treatment of nerve and tendon mobilization in two different movement directions.

Methods

38 consecutive patients (55 hands) who had been referred to the Neurophysiological Laboratory of the Neurosurgical Clinic of the Siena University, Medical School, for evaluation of CTS were studied (*group B*). All of them exhibited symptoms of CTS (eg. Pain, numbness, tingling). A screening history and physical examination was conducted to ensure that the referring diagnosis of CTS was warranted, and to exclude those individuals who were not suitable for the study (i.e. those with peripheral neuropathy or obvious entrapment neuropathy other than median nerve).

55 hands of 32 hospital staff and healthy adult age matched volunteers served as control subjects (*group A*). These individuals did not show any signs or symptoms of CTS and their conventional NCS were within normal limits.

Procedure

A standard electrodiagnostic examination including conventional motor and sensory median and ulnar nerve NCS was performed in both groups.

The active recording position at the motor point of the thenar eminence was carefully controlled for the exact stimulation on the motor point in order to decrease pseudofacilitation phenomena that could be due to change of muscle length [17].

After having measured the baseline medial nerve distal motor latency (DML) and maximal compound muscle action potential (CMAP) from the thenar muscles at rest, 2 series of 10 passive wrist flexions were performed, followed by two series of 10 wrist extensions. After each series of ten passive movements a new measurement of DML and maximal CMAP of the median nerve was obtained. Both passive movements (flexion and extension) were performed until the end range of motion in the required direction.

Amplitude values (negative to positive peak) were measured after exercise performance were normalized and expressed as percentage of rest amplitude. The mean of the two values obtained after the first and the second flexion series and similar mean value after the two extension series were expressed as number value M-test, and values obtained were arbitrarily classified as worsened (lower or equal to 95% of rest value), unchanged (between 95% and 105%) and improved (equal or superior to 105%). Statistical analysis was performed with parametric and non-parametric test (Chi-square, Fisher's exact test and McNemar's) by the SPSS program (SPSS Inc.).

Results

Table 1 and Fig. 1 show mean and standard deviations of Distal Motor Latency and amplitude changes in patients and controls. The first analysis doesn't show any significant difference.

A normalization of values expressed as percentage of rest value (Fig. 2) showed a more elevated standard deviation in the patient group as compared to normal controls. In our opinion, this increased variation in patients is due to the presence of subjects with different disease typologies and therefore with different responses after movement performance.

To test if the distribution of the various responses after repeated movements could be different in subgroup of patients, we calculated the mean of the two values obtained after two series of extension and after two series of flexion movements. Obtained values were classified as worsened if they were lower than 95% of rest value, unchanged if between 95% and 105% and improved if superior to 105%.

The contingency table obtained (Table 2) is composed of three groups, classified according to CMAP change after repeated passive movements. The statistical analysis showed a difference in patient distribution via-à-vis normal controls in both directions of movement.

Table 1. Modifications of mean distal motor latency (DML) and amplitude (AMP) of Compound Motor Action Potential (CMAP) registered from thenar eminence in patients and control subjects. Values were obtained at rest and after each of two groups of 10 repeated end range passive flexion movement (10 Flex) and two groups of 10 repeated end range passive extension movements (10 Ext). Standard deviation (SD)

CMAP Changes	CONTR	CONTROLS					PATIENTS				
	Rest	10 Flex	10 Flex	10 Ext	10 Ext	Rest	10 Flex	10 Flex	10 Ext	10 Ext	
DML ms (SD)	3.49 (0.50)	3.50 (0.51)	3.49 (0.49)	3.53 (0.50)	3.51 (0.51)	4.62 (0.95)	4.62 (0.88)	4.64 (0.89)	4.65 (0.89)	4.66 (0.86)	
AMP ms (SD)	10.0 (2.98)	10.0 (3.19)	10.1 (3.18)	10.1 (2.87)	10.1 (2.94)	6.93 (3.47)	6.72 (2.85)	6.61 (2.77)	6.79 (3.01)	6.57 (2.80)	

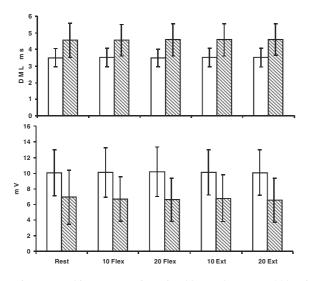


Fig. 1. Graphic representation of Table 1 value. Same Abbreviations

Moreover, comparing distributions of patients after flexion and extension we found a clear difference between the two movement directions. In Fig. 3, in fact, in the histogram of changes after flexion movements we may see a worsening of a wide group of patients without a significant behavioural change in control subjects. End range passive extension on the other hand resulted in a wider distribution also towards improvement in the patient group.

Figure 4 and Table 3 compare the presence of covariations (after flexion and extension) and their consistency between the two groups.

11 hands (32%) of patients and only 3 hands (5%) of controls showed worsening both with repeated flexion and extension movements. In total 31 hands of patients (56%) showed worsening in one and no variation in the other direction of movement or worsening in both directions. In the control group only 8 hands (14,5%) showed a similar response.

Regarding improvements 12 affected hands (22%) were improved after extension and unchanged after flexion against the 12% of controls (7 hands) showing a probable general tendency but without any statistical significance.

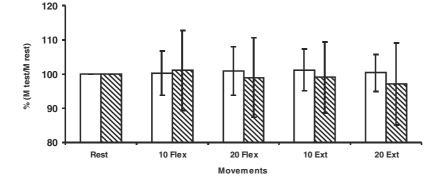


Fig. 2. Normalized Compound Motor Action Potential in Normal Controls (white) and Patients (dashed) shows a wider standard deviation in the patient category

 Table 2. Contingency table of three arbitrary categories of changes of compound muscle action potential after test movement

	Flexion		Extension		
	Control	Patients	Control	Patients	
CMAP < 95%	7	13	8	20	
95% < CMAP < 105%	38	22	37	23	
105% < CMAP	10	20	10	12	

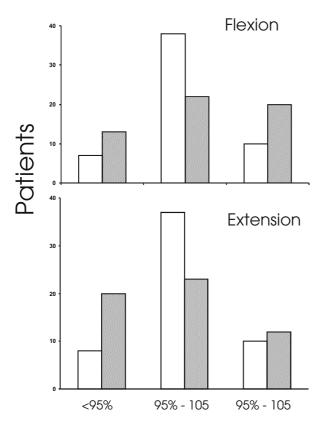
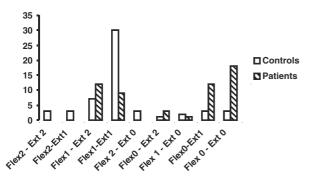


Fig. 3. Histogram of Table 2 values. White: normal control. Dashed: patients



2=improved; 1=no variation; 0=worsened

Fig. 4. As in Table 3. Covariation of compound motor action potential above 105%, below 95% or unchanged with respect to baseline in the two movement directions

Table 3. Co-variation of compound motor action potential above 105% (improved), below 95% (worsened) or unchanged with respect to baseline in the two movement directions

Flexion	Î	Î	\leftrightarrow	Î	\leftrightarrow	Ţ	\leftrightarrow	Ļ	1
Extension	ŕ	\leftrightarrow	Î	Ļ	\leftrightarrow	Ť	Ļ	$\stackrel{\bullet}{\leftrightarrow}$	Ĵ
Controls	3	3	7	3	30	1	2	3	3
Patients	0	0	12	0	9	3	1	12	18
\uparrow improved; \downarrow worsened; \leftrightarrow unchanged									

Discussion

According to the literature, recent prospective studies have shown that in a consistent group of patients affected by CTS a spontaneous improvement can be observed [11]. On the other hand, studies analyzing the efficacy of non surgical treatments in CTS show a very low success rate for the majority of the different conservative methods, [20] even though a tendency towards improvement with the use of non surgical and non infiltrative methods is generally accepted [10].

Most of the studies aimed at verifying the efficacy of the different conservative approaches in the treatment of CTS fail to show a statistical significance: this could be due, in our opinion, to a wide variation of responses in the samples studied.

Owing to a progressively improved health education many patients affected by CTS certainly receive an early diagnosis and seek prompt treatment. On the other hand, many patients still come late to a clinical evaluation [13, 22].

It is well known that CTS can be caused by several different diseases. Therefore, since there are many different stages of the disease and many different physiopathologic entities, it seems logical to suppose that there are wide differences in treatment responses in the group of patients studied.

In fact, the group of patients studied showed important variations in the amplitude of PAMC and significantly different responses as compared to the control group.

Moreover, within the group of patients studied, it seems possible to distinguish single and consistent subgroups of variations. Thirty-two percent of patients as compared to only 5% of controls showed worsening both with repeated movements of flexion and extension. Also, 22% of patients, compared with only 12% of controls, showed improvement after extension exercises and no variations after flexion exercises.

It is surprising that a series of flexion and extension

50

movements actually creating an increase in pressure within the carpal tunnel in the range of 30–110 mmHg [4], can determine an amplitude increase and hence an improvement in the impulse conduction in the motor axons of patients with CTS.

Since only a sub-group of patients showed such a response, it seems to be worth using this method to optimize the criteria for selection of best physiother-apeutic treatment for each patient.

In fact, the conservative treatment with exercises of nerve and tendon mobilization could be a good choice for many patients, but not for all. In our study, for example, a sub-group of patients, showed severe worsening (at least transitory) after a series of these exercises.

In the case reports of other authors individual patient sensitivity to a specific treatment could have been known in advance if a dynamic neurophysiologic evaluation like the one we propose had been performed at the start point of treatment selection in order to obtain a more homogenous group of patients for study with more reliable statistical results.

A dynamic test as proposed by us could also be used as provocative test to discover alterations remaining undetectable by using conventional neurophysiologic studies.

The validity of neurophysiologic methods using provocative tests is still under debate in the literature: the question is whether these tests could really be helpful detecting patients otherwise negative at EMG examination. Currently a significant percentage of patients with CTS (ranging from 16 to 51% according to different authors) are not discovered by common neurophysiologic tests: this is particularly true for the mild forms of CTS [1].

The poor results presently obtained by provocative tests could be due to the fact that they almost always analyse only response latency, which usually shows very weak and poor relevant variations, as we demonstrated in our study [23].

The different contributions of axonal attenuation, ischemia, demyelination and remyelination to the pathophysiology of carpal tunnel syndrome is still unresolved but recent evidence showed that demyelination may not be a critical factor for the slow down of impulse conduction in mild to moderate carpal tunnel syndrome. Hence it is reasonable to suppose that latency variations are not the ideal parameter to be studied by provocative tests.

Sometimes a surgical decision is made only on the

basis of clinical symptoms with negative neurophysiologic examination [7]. Surprisingly, surgical procedures performed without considering the outcome of neurophysiologic examinations don't show different long term results [5, 8]. Anyway, the aim is to reduce as much as possible therapeutic decisions made only on the basis of clinical symptoms, without confirmation of positive objective, reliable, sensitive and specific diagnostic tests so that the failure rate of surgery is reduced and unnecessary surgical risk is avoided.

In our study, only a few subjects showed an amplitude decrease of PAMC both in flexion and extension in the control group (but the normal subjects were chosen in a pseudo-random manner). However, these people when subsequently investigated for the presence of typical symptoms of CTS revealed mild symptomatology in 3 out of 4 cases. These subjects behaved in a way similar to a sub-group of patients treated with early surgical intervention. Probably there is a subgroup of people, like pregnant women [12], who could benefit from a preventive surgical treatment. In such particular cases, a provocative test like the one we propose, could be very useful to best identify those people to be operated on.

References

- (1993) Practice parameter for electrodiagnostic studies in carpal tunnel syndrome (summary statement). American Academy of Neurology, American Association of Electrodiagnostic Medicine, and American Academy of Physical Medicine and Rehabilitation. Neurology 43: 2404–2405
- Akalin E, El O, Peker O, Senocak O, Tamci S, Gulbahar S, Cakmur R, Oncel S (2002) Treatment of carpal tunnel syndrome with nerve and tendon gliding exercises. Am J Phys Med Rehab 81: 108–113
- Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosen I (1999) Prevalence of carpal tunnel syndrome in a general population. Jama 282: 153–158
- Gelberman RH, Hergenroeder PT, Hargens AR, Lundborg GN, Akeson WH (1981) The carpal tunnel syndrome. A study of carpal canal pressures. J Bone Joint Surg Am 63: 380–383
- Glowacki KA, Breen CJ, Sachar K, Weiss AP (1996) Electrodiagnostic testing and carpal tunnel release outcome. J Hand Surg [Am] 21: 117–121
- Katz JN, Simmons BP (2002) Clinical practice. Carpal tunnel syndrome. N Engl J Med 346: 1807–1812
- Kitsis CK, Savvidou O, Alam A, Cherry RJ (2002) Carpal tunnel syndrome despite negative neurophysiological studies. Acta Orthop Belg 68: 135–140
- Longstaff L, Milner RH, O'Sullivan S, Fawcett P (2001) Carpal tunnel syndrome: the correlation between outcome, symptoms and nerve conduction study findings. J Hand Surg [Br] 26: 475–480
- Mondelli M, Giannini F, Giacchi M (2002) Carpal tunnel syndrome incidence in a general population. Neurology 58: 289– 294

- O'Connor D, Marshall S, Massy-Westropp N (2003) Nonsurgical treatment (other than steroid injection) for carpal tunnel syndrome. Cochrane Database Syst Rev 1
- Padua L, Padua R, Aprile I, Pasqualetti P, Tonali P (2001) Multiperspective follow-up of untreated carpal tunnel syndrome: a multicenter study. Neurology 56: 1459–1466
- Padua L, Aprile I, Caliandro P, Mondelli M, Pasqualetti P, Tonali PA (2002) Carpal tunnel syndrome in pregnancy: multiperspective follow-up of untreated cases. Neurology 59: 1643– 1646
- Pienimaki T (2002) Cold exposure and musculoskeletal disorders and diseases. A review. Int J Circumpolar Health 61: 173–182
- Priganc VW, Henry SM (2003) The relationship among five common carpal tunnel syndrome tests and the severity of carpal tunnel syndrome. J Hand Ther 16: 225–236
- Rozmaryn LM, Dovelle S, Rothman ER, Gorman K, Olvey KM, Bartko JJ (1998) Nerve and tendon gliding exercises and the conservative management of carpal tunnel syndrome. J Hand Ther 11: 171–179
- Solomon DH, Katz JN, Bohn R, Mogun H, Avorn J (1999) Nonoccupational risk factors for carpal tunnel syndrome. J Gen Intern Med 14: 310–314
- Stalberg EV, Trontelj JV (1994) Single fiber electromyography: studies in healthy and diseased muscles. Raven Press, New York

- Tal-Akabi A, Rushton A (2000) An investigation to compare the effectiveness of carpal bone mobilisation and neurodynamic mobilisation as methods of treatment for carpal tunnel syndrome. Man Ther 5: 214–222
- Totten PA, Hunter JM (1991) Therapeutic techniques to enhance nerve gliding in thoracic outlet syndrome and carpal tunnel syndrome. Hand Clin 7: 505–520
- Verdugo RJ, Salinas RS, Castillo J, Cea JG (2003) Surgical versus non-surgical treatment for carpal tunnel syndrome. Cochrane Database Syst Rev CD001552
- Werner R, Armstrong TJ, Bir C, Aylard MK (1997) Intracarpal canal pressures: the role of finger, hand, wrist and forearm position. Clin Biomech (Bristol, Avon) 12: 44–51
- Werner RA, Hamann C, Franzblau A, Rodgers PA (2002) Prevalence of carpal tunnel syndrome and upper extremity tendinitis among dental hygienists. J Dent Hyg 76: 126–132
- Wiederien RC, Feldman TD, Heusel LD, Loro WA, Moore JH, Ernst GP, Allison SC (2002) The effect of the median nerve compression test on median nerve conduction across the carpal tunnel. Electromyogr Clin Neurophysiol 42: 413–421

Correspondence: Alessandro Zalaffi, Departement of Ophthalmological and Neurosurgical Science, Siena University, Policlinico "Le Scotte", Viale Bracci, 53100, Siena, Italy. e-mail: Mariottini@ unisi.it