

# ASSOCIATIONS BETWEEN FAMILY HISTORY OF CARPAL TUNNEL SYNDROME AND ABNORMAL MEDIAN NERVE CONDUCTION WITH AND WITHOUT SYMPTOMS OF CARPAL TUNNEL SYNDROME

Hannah Edwards, University of Utah  
Matthew S. Thiese, University of Utah  
Kurt T. Hegmann, University of Utah

[kurt.hegmann@hsc.utah.edu](mailto:kurt.hegmann@hsc.utah.edu)

## ABSTRACT

**Background:** Few studies have examined the relationship between family history and carpal tunnel syndrome (CTS) on a population basis.

**Methods:** Workers (N=841) completed a questionnaire, structured interview, two standardized physical examinations, and a bilateral nerve conduction study (NCS). Self-reported family history of CTS was recorded from the questionnaire.

**Results:** 37.0 % of subjects with CTS by a case definition (abnormal median nerve conduction at the wrist consistent with CTS plus tingling/numbness in at least two median nerve served digits) reported a family history of CTS compared to 21.3% of subjects without CTS. Family history was also significantly associated with bilateral NCS abnormalities consistent with CTS in multivariate models with an odds ratio of 1.81 (95% CI 1.09-3.01) when controlling for age, BMI, gender, and diabetes mellitus. Family history was also associated with the case definition of CTS with an OR of 2.44 (95% CI 1.43-3.27) for right sided and 2.25 (95% CI 1.23-4.10) for bilateral CTS. The associations persisted after controlling for age, body mass index, gender, diabetes mellitus, hypertension, high cholesterol, thyroid disorders, gout, osteoarthritis, and inflammatory arthritis. The mean age of the subjects with and without a family history of CTS did not differ.

**Conclusions:** Self-reported family history of CTS appears to be associated with bilateral median nerve conduction abnormalities in multivariate models, as well as with a case definition of CTS.

## INTRODUCTION

Carpal Tunnel Syndrome (CTS) is thought to occur as a result of any mechanism causing compression or entrapment of the median nerve as it travels through the carpal tunnel at the wrist. Theoretically, any factor that contributes to an increase in the size of the contents of the tunnel, increased pressure in the tunnel, decrease in the overall size of the tunnel, or changes in the function of the median nerve could lead to CTS. Research into the causes of CTS has focused on work-place exposures, personal characteristics, hobbies, medical conditions, personal psychosocial, and work-related psychosocial factors that appear to be associated with CTS. Few

studies have considered a possible familial or genetic contribution to the development of CTS in the general population.

Several case reports documenting CTS in multiple generations of affected families indicate that a familial form of CTS may exist (Danta, 1975; Folik, 1998; Gossett, 1998; Gray, 1979; Leifer, 1992; Mahjneh, 2001; Michaud, 1990; Stoll, 1998; Vadasz, 1997). In these families, CTS often developed bilaterally at young ages and appeared to be transmitted as an autosomal dominant trait. The most frequently identified abnormality in these families was thickening of the transverse carpal ligament. The overall prevalence of this familial form of CTS is unknown.

A few studies have described the genetic or familial contribution to CTS on a population basis. Hakim et al. estimated a heritable fraction for CTS of 46% in a population of monozygotic and dizygotic female twins in the St. Thomas' UK Adult Twin Registry (Hakim, 2002). This heritable estimate did not change after controlling for age, body mass index, menopausal status, reported occupation, and leisure or home activity.

Radecki collected information on family history from patients referred to an electrodiagnostic and physical medicine practice (Radecki, 1994). He found that, overall, 21.7% of patients with CTS reported a family history of CTS as compared to 13.3% of patients without CTS. Of the patients who had undergone a prior carpal tunnel release, 39.3% were aware of a family history. Radecki proposed that 39.3% more closely reflected the true proportion of people with CTS who have a family history because he assumed that people who had undergone surgery for CTS would have the strongest motivation to learn their family history.

Alford et al. sought to account for an effect from recall bias when collecting information on family history by comparing reported family history in patients seen at a hand surgery clinic with unilateral and bilateral CTS. These authors assumed that family history's influence on CTS would affect both wrists equally, generating more bilateral disease, and that patients with one sided CTS would have the same motivation to learn their family history as patients with bilateral CTS. They found that 45% of patients with bilateral CTS reported a family history of CTS, compared to 27% of patients with unilateral CTS and 22% of patients without CTS (Alford, 2004).

The purpose of this study is to describe relationships between a family history of CTS and unilateral and bilateral abnormal median nerve conduction at the wrist that is consistent with CTS with or without symptoms of CTS in an industrial population. This study also examined the mean ages of affected subjects with and without a family history, and sought to estimate the possible effect of recall bias on results.

## **MATERIALS AND METHODS**

### **Subjects**

A total of 869 workers from twelve different manufacturing operations in Wisconsin and Utah enrolled in a prospective cohort study examining the causes of upper extremity musculoskeletal disorders. These employees worked in a variety of industries, including electric motor

manufacturing, assembly, apparel manufacturing, meat processing, electric light manufacturing, small motor manufacturing, and office work. Complete data were available from 841 subjects for the purposes of these analyses.

### **Data collection, Examination, and Evaluation of Symptoms**

Each participant completed a laptop administered questionnaire that collected information on demographic characteristics, medical conditions, hobbies, exercise, and psychosocial factors. Anthropomorphic data were measured, and each participant underwent a laptop administered structured interview and standardized physical examination by either a hand therapist or occupational medicine resident. A second examiner, board certified in occupational medicine, confirmed all pertinent examination findings, as well as re-examined pertinent negative findings.

### **Family History of Carpal Tunnel Syndrome**

One question in the questionnaire inquired about family history of CTS. Participants provided a “yes” or “no” response to the question “Has anyone in your family (blood relatives only) ever been diagnosed with carpal tunnel syndrome?”

### **Nerve Conduction Studies**

All subjects underwent nerve conduction testing of both hands following an established protocol conducted by board certified physical medicine and rehabilitation physicians. These examiners were blinded to other measures, including the presence of symptoms, physical examination findings and occupational exposures. Hand temperature was measured. All hands were warmed to at least 34 degrees centigrade prior to evaluation. One physician (JW) reviewed all NCS to interpret the studies as normal, abnormal but not consistent with CTS, and abnormal and consistent with CTS. Those consistent with CTS were further categorized as mildly moderately and severely abnormal. Abnormal median nerve conduction was defined by the following parameters: transcarpal delta  $\geq 0.4$  msec, motor latency  $> 4.4$  msec, and sensory latency  $> 3.7$  msec. Participants with an abnormal NCS that was felt to be inconsistent with CTS were excluded from the analyses used in this report.

### **Outcomes**

A prevalent case of CTS was defined as an abnormal median nerve NCS consistent with CTS plus tingling/numbness in at least two median nerve served digits (thumb, index middle and/or ring finger) in the past month. This study also assessed a second outcome of an abnormal NCS consistent with CTS, but irrespective of symptoms. The two outcomes were thought to differ in their objectivity. The NCS is an objective measure of median nerve conduction, as compared to the case definition of CTS that required the addition of participant self-report of symptoms.

### **Statistical Analysis**

All data was analyzed using SAS 9.1 (SAS Institute Inc., Cary, NC, USA). The proportions of subjects reporting a positive family history for various outcomes were expressed as percentages.

Odds ratios for potential associated factors were measured with logistic regression for both outcomes of abnormal NCS and the case definition of CTS. Models were constructed to control for age, measured body mass index (BMI), gender, and self reported past diagnosis of diabetes mellitus. Additional models were constructed to control for self reported past diagnoses of hypertension, high cholesterol, thyroid disease, gout, and wrist osteoarthritis as diagnosed by a physician. T-test comparisons were used to assess differences in age between subjects with and without a family history.

## RESULTS

Table 1 summarizes the demographics of the study population. The average age of study participants was 41.4 years and they tended to be overweight with a mean BMI of 29.5 kg/m<sup>2</sup>. Approximately two-thirds were female. Forty-five subjects (5.3%) reported having diabetes mellitus. Overall, 256 of 841 subjects (31.3%) had an abnormal NCS consistent with CTS. Of the 256 subjects with abnormal NCS, 119 (14.0%) also had tingling/numbness to meet the case definition for CTS. Of the 256 subjects with abnormal NCS, 147 had right sided only abnormalities and 103 had bilateral abnormalities. Of the 147 subjects with right-sided median nerve conduction abnormalities, 61 experienced tingling/numbness that met the case definition for CTS. Of the 103 subjects with bilateral median nerve conduction abnormalities, 48 experienced tingling/numbness that met the case definition. Very few subjects had left sided only abnormal median nerve conduction abnormalities (15 of 256) or met the case definition (10 of 15).

Overall, 37.0 % of subjects meeting the CTS case definition reported a family history of CTS, compared to 21.3% of workers who did not meet the case definition (Table 2). Of the 156 subjects who had abnormal NCS and no tingling/numbness, 32 (20.5%) reported a family history of CTS. Of the 104 subjects who had normal NCS but reported tingling/numbness, 28 (26.9%) reported a family history of CTS. Twenty-one percent of subjects with a normal NCS and no tingling/numbness reported a family history of CTS.

None of the participants with left sided only CTS reported a family history of CTS. Due to the small number of subjects with left sided only abnormalities, logistic regression analyses are shown (Tables 3, 4, and 5) for subjects with right sided and bilateral abnormalities.

Table 1. Demographic characteristics of the study population (N=847)

Variable	n (%) or mean +/- S.D.
Age	41.4 ± 11.2 years
Body Mass Index	29.5 ± 6.9 kg/m <sup>2</sup>
Sex	
Male	285 (32.7%)
Female	584 (67.3%)
Handedness	
Right	743 (85.5%)
Left	81 (9.3%)
Ambidextrous	45 (5.2%)
Smoking status	
Current smoker	247 (28.4%)
Ex-smoker	197 (22.7%)
Lifelong non-smoker	425 (48.9%)
Diabetes Mellitus	45 (5.3%)
Hypertension	134 (15.9%)
High cholesterol	131 (15.5%)
Thyroid disease	58 (6.9%)
Gout	14 (1.7%)
Osteoarthritis	78 (9.2%)
Inflammatory arthritis	37 (4.4%)
<u>Outcomes</u>	
Abnormal NCS	
Right	147 (17.4%)
Left	15 (1.8 %)
Bilateral	103 (12.2%)
Total	265 (31.3%)
Case Definition	
Right	61 (7.2%)
Left	10 (1.2%)
Bilateral	48 (5.7%)
Total	119 (14.0%)

Table 3 shows results from univariate logistic regression analyses of the relationship between a self-reported family history of CTS and the outcomes of all cases, right sided and bilateral abnormal NCS and the case definition of CTS. A self-reported family history of CTS was associated with the case definition with an odds ratio (OR) of 2.16 (95% CI 1.43-3.27). The relationship was not stronger for bilateral cases than right sided cases. The OR for right CTS was 2.44 (95% CI 1.42-4.17) and 2.25 (95% CI 1.23-4.10) for bilateral CTS. The relationship between a self-reported family history and right sided and bilateral abnormal NCS did not reach statistical significance.

Table 2. Workers reporting a family history of carpal tunnel syndrome

Outcome	Number of workers	Number reporting a positive FH for CTS <sup>1</sup>	(Percent)
Abnormal NCS with tingling/numbness (Case Definition)			
Right	61	25	(41.0%)
Left	10	0	(0.0%)
Bilateral	48	19	(39.6%)
Total	119	44	(37.0%)
Abnormal NCS without tingling/numbness			
Right	95	18	(19.0%)
Left	16	5	(31.3%)
Bilateral	45	9	(20.0%)
Total	156	32	(20.5%)
Total Any Abnormal NCS <sup>2</sup>	265	73	(27.6%)
Normal NCS with tingling/numbness			
Right	49	11	(22.5%)
Left	55	17	(30.9%)
Bilateral	0	0	(0.0%)
Total	104	28	(26.9%)
Normal NCS without tingling/numbness	447	94	(21.0%)
Total not meeting the Case Definition	722	154	(21.3%)

1: Self-reported family history of CTS.

2: Ten people with unilateral CTS by the case definition had an abnormal NCS but no symptoms on the other side.

Table 3. Univariate analysis of the relationship between a self-reported family history of CTS and abnormal NCS consistent with CTS or Case Definition of CTS

	Odds Ratio (95% CI)	
	<u>Abnormal NCS</u>	<u>Case Definition</u>
All	1.37 (0.98-1.92)	2.16 (1.43-3.27)
Right	1.28 (0.86-1.92)	2.44 (1.42-4.17)
Bilateral	1.47 (0.93-2.32)	2.25 (1.23-4.10)

\*Abnormal NCS consistent with CTS plus tingling/numbness in at least 2 median nerve served digits.

In a multivariate analysis controlling for age, BMI, gender, and diabetes mellitus, a self-reported family history of CTS appeared more strongly associated with bilateral than right sided abnormalities on NCS (Table 4). The association between family history and bilateral abnormal NCS reached statistical significance with an odds ratio of 1.81 (95% CI 1.09-3.01). Family history was not more strongly associated with bilateral CTS as defined by the case definition. Of

note, age, BMI, and diabetes mellitus also demonstrated stronger associations with bilateral disease, which is consistent with the systemic influences of these variables. Gender was not associated with abnormal median nerve conduction in these data.

Table 4. Multivariate analyses of the relationship between a family history of CTS and right sided or bilateral abnormal NCS or case definition of CTS.

	Odds Ratio (95% CI)			
	Right		Bilateral	
	<u>Abnormal NCS</u>	<u>Case Definition</u>	<u>Abnormal NCS</u>	<u>Case Definition</u>
Family History	1.29 (0.85-1.95)	2.53 (1.46-4.40)	1.81 (1.09-3.01)	2.48 (1.31-4.69)
Age (per year)	1.02 (1.00-1.04)	1.02 (0.99-1.04)	1.06 (1.03-1.08)	1.04 (1.01-1.07)
BMI (kg/m <sup>2</sup> )	1.05 (1.02-1.07)	1.04 (1.00-1.07)	1.11 (1.08-1.14)	1.07 (1.03-1.12)
Female Gender	0.91 (0.61-1.35)	1.52 (0.79-2.91)	0.92 (0.55-1.54)	1.52 (0.71-3.28)
Diabetes Mellitus	0.64 (0.27-1.50)	1.36 (0.49-3.74)	3.55 (1.76-7.13)	2.59 (1.06-6.31)

In an extended multivariate model that additionally controlled for hypertension, high cholesterol, thyroid disorder, gout, osteoarthritis, and inflammatory arthritis, the association between family history and abnormal NCS remained essentially unchanged, suggesting that family history may influence the development of CTS through a mechanism not accounted for in the model (Table 5).

Table 5. Extended multivariate analyses of the relationship between a family history of CTS and right sided or bilateral abnormal NCS or case definition of CTS

	Odds Ratio (95% CI)			
	Right		Bilateral	
	<u>Abnormal NCS</u>	<u>Case Definition</u>	<u>Abnormal NCS</u>	<u>Case Definition</u>
Family History	1.26 (0.83-1.92)	2.51 (1.43-4.41)	1.68 (1.00-2.83)	2.33 (1.16-4.30)
Age (per year)	1.01 (0.99-1.03)	1.01 (0.98-1.04)	1.05 (1.02-1.08)	1.03 (1.00-1.07)
BMI (kg/m <sup>2</sup> )	1.04 (1.01-1.07)	1.03 (1.00-1.07)	1.11 (1.07-1.14)	1.08 (1.03-1.12)
Female Gender	0.95 (0.63-1.43)	1.63 (0.84-3.16)	0.85 (0.50-1.45)	1.35 (0.62-2.99)
Diabetes Mellitus	0.59 (0.24-1.40)	1.39 (0.48-4.01)	3.06 (1.49-6.29)	2.04 (0.81-5.15)
Thyroid Disorder	1.10 (0.54-2.23)	0.48 (0.14-1.64)	2.03 (0.98-4.22)	2.79 (1.19-6.53)
High Cholesterol	1.08 (0.65-1.78)	1.92 (1.00-3.69)	1.27 (0.73-2.21)	1.49 (0.73-3.04)
High Blood Pressure	1.21 (0.72-2.01)	0.97 (0.46-2.03)	1.32 (0.76-2.30)	1.13 (0.53-2.39)
Gout	3.24 (1.01-10.40)	2.03 (0.38-10.98)	0.77 (0.18-3.36)	1.44 (0.26-8.03)
Osteoarthritis	1.05 (0.57-1.95)	1.49 (0.66-3.34)	1.04 (0.51-2.10)	0.55 (0.18-1.70)
Inflammatory Arthritis	1.40 (0.63-3.09)	0.55 (0.12-2.49)	1.35 (0.57-3.20)	1.52 (0.52-4.49)

Comparison of the mean ages of subjects with abnormal NCS or case definition of CTS with and without a reported family history did not reveal any statistically significant differences in ages (Table 6). There was, however, a trend towards younger age among those subjects with bilateral

CTS by the case definition and a family history than those with bilateral CTS and no family history (p-value of 0.07).

Table 6. Mean ages of subjects with abnormal median nerve conduction consistent with CTS or case definition of CTS with and without a self-reported family history of CTS

	Mean Age		
	<u>+FH</u>	<u>No FH</u>	<u>p value</u>
Abnormal NCS			
Right	43.5	43.4	0.20
Bilateral	46.2	47.5	0.51
Case Definition			
Right	43.4	43.8	0.89
Bilateral	43.1	48.1	0.07

## DISCUSSION

### Overall Results

This study found that a family history of CTS was associated with a right sided and bilateral case definition of CTS that was defined as a NCS consistent with CTS plus symptoms of tingling/numbness in at least two digits in a median nerve distribution. Family history was more strongly associated with bilateral abnormal NCS regardless of symptoms in multivariate models. Family history was not more strongly associated with bilateral than unilateral CTS as defined by the case definition. The data did not clearly demonstrate that subjects with a family history and abnormalities were younger than subjects without a family history. However, there was a trend for subjects with bilateral CTS as defined by the case definition to be younger if they reported a family history (p-value of 0.07), suggesting a lack of power.

### Unilateral and Bilateral Abnormalities

Genetic or environmental factors that might mediate family history's influence on the development of CTS would be predicted to involve both wrists and possibly lead to an excess of bilateral CTS. A genetic predisposition to CTS could also lead to early development of disease. Associations between family history and unilateral versus bilateral abnormalities and the mean ages of individuals with abnormalities with and without a family history were assessed to test these predictions. In contrast to the report from Alford et al. that indicated that people with bilateral CTS were more likely to report a family history of CTS, this study found similar proportions of subjects with right sided and bilateral abnormal median nerve conduction with or without tingling/numbness reported a family history. A self-reported family history of CTS was more strongly associated with bilateral abnormal median NCS, but not more strongly with bilateral CTS as defined by the case definition. Overall, these results indicated a trend towards increased association with bilateral disease, but the associations were inconsistent. Comparison of mean ages of subjects with abnormalities also identified a non-statistically significant trend toward subjects with bilateral CTS by the case definition being younger if they reported a family history.

## **Limitations**

Results from this study suggest an association between family history and CTS. However, the number and type of relatives affected were not recorded. The associations between family history and CTS could have been apparently increased through the effect of recall bias. The models also did not take into account possible occupational contributions to the development of CTS. However, there is no reason to suspect differential distribution of subjects with and without a family history based on occupational factors.

Because CTS is a well known term for a disease that requires tingling/numbness for diagnosis, and there are widespread scientific and non-scientific publications describing hand pain, subjects may have incorrectly assumed that any relative with tingling/numbness or pain in his or her hands had CTS. This incorrect assumption could have led to misclassification of subjects' family histories in the study. Lack of detail regarding the number, ages, and types of relatives reported to have CTS precluded examination of a possible dose-response type relationship in which individuals with multiple close relatives with CTS might have been more likely to have abnormal median nerve conduction.

Recall bias may have influenced the results of the study. Subjects with tingling/numbness or a diagnosis of CTS may have been more likely to learn their family history of CTS than those without symptoms. This differential knowledge of family history could have led to increases in the odds ratios between family history and abnormal NCS, particularly in the group with tingling/numbness. The finding that only 20.5% of subjects with abnormal NCS and no symptoms reported a family history suggests that recall bias could have generated large odds ratios between family history and the case definition of CTS. However, only 26.9% of the group of subjects with normal NCS and tingling/numbness reported a family history, which is less than the 37.0% of subjects with abnormal NCS and tingling/numbness (case definition of CTS). The presence of symptoms in this group with normal NCS may have motivated this group to learn their family history, but they did not report a family history as often as subjects with CTS. The lower proportion reporting a family history as compared to the group meeting the case definition suggests that recall bias is unlikely to account for all of the association between self-reported family history and CTS.

A family history of CTS may confer a predisposition to median nerve compression or dysfunction that manifests as CTS in the presence of additional individual, environmental, work place, personal psychosocial or non-occupational psychosocial factors. Because the models in this study did not account for the physical exposures, no conclusion can be made about the interactions between family history and environmental or physical exposures.

## **Future Analysis**

In future analyses from this occupational cohort, incident cases of CTS may allow for a better understanding of how recall bias may have influenced results. Many or most of the subjects with incident cases of CTS would likely have been asymptomatic at the beginning of the study when information on family history was recorded. Comparing associations between reported family

history and the incident cases of CTS should provide insight into the effect recall bias has had on the cross-sectional data. Future analyses should also include leisure and work place ergonomic exposures.

Family history could influence risk for developing CTS through anatomical changes in the shape or size of the carpal tunnel, changes in collagen formation, or through psychosocial mechanisms. Conceivably, shared environmental factors within families or shared psychological influences on symptom perception could account for part of family history's influence, and may explain why family history appeared most strongly associated with risk for CTS in subjects who perceived tingling/numbness. Previous researchers have identified associations between anatomical factors such as increased wrist depth to width ratio and abnormal median nerve conduction (Kamolz, 2004, Kouyoumdjian, 2000). Further analysis of the influence of family history on CTS could focus on identifying anatomical differences in individuals with a family history of CTS.

### ACKNOWLEDGEMENTS

This research was supported (in part) by the Rocky Mountain Center for Occupational and Environmental Health at the University of Utah. The Rocky Mountain Center, an Education and Research Center, is supported by Training Grant No. T42/OH 008414 from the Centers for Disease Control and Prevention/National Institute for Occupational Safety and Health. The contents are solely the responsibility of the authors and do not necessarily represent the official views of the National Institute for Occupational Safety and Health.

### REFERENCES

- Alford, J.W., Weiss, A., Akelman, E. The familial incidence of carpal tunnel syndrome in patients with unilateral and bilateral disease. *Am J Orthop*. 2004; 33(8): 397-400.
- Braddom, R.L. Familial carpal tunnel syndrome in three generations of a black family [abstract]. *Am J Phys Med*. 1985; 64(5): 227-234.
- Danta, G. Familial carpal tunnel syndrome with onset in childhood. *J Neurol Neurosurg Psychiatry*. 1975; 38(4): 350-355.
- Folik, A., Modai, D., Pervin, R., Marcus, E.L., Fried, K. Autosomal dominant carpal tunnel syndrome in a Karaite family. *Isr J Med Sci*. 1998; 24(6): 295-297.
- Gossett, J., Chance, P.F. Is there a familial carpal tunnel syndrome? An evaluation and literature review. *Muscle Nerve*. 1998; 21(11): 1533-1536.
- Gray, R.G., Poppo, M.J., Gottlieb, N.L. Primary familial bilateral carpal tunnel syndrome. *Ann Intern Med*. 1979; 91(1): 37-40.
- Hakim, A.J., Cherkas, L., El Zayat, S., Macgregor, A., Spector, T.D. The genetic contribution to carpal tunnel syndrome in women: a twin study. *Arthritis Rheum*. 2002; 47(3): 275-279.

- Kamolz, L.P., Beck, H., Haslik, W., Hogler, R., Rab, M., Schrogendorfer, K.F., Frey, M. Carpal tunnel syndrome: a question of hand and wrist configurations? *J Hand Surg* (British and European Volume). 2004; 29B:4: 321-324.
- Kouyoumdjian, J.A., Morita, M.P.A., Rocha, P.R.F., Miranda, R.C., Gouveia, G.M. Wrist and palm indexes in carpal tunnel syndrome. *Arq Neuropsiquiatr*. 200; 58(3-A): 625-629.
- Leifer, D., Cros, D., Halperin, J.J., Gallico, G.G., Pierce, D.S., Shahani B.T. Familial bilateral carpal tunnel syndrome: report of two families. *Arch Phys Med Rehabil*. 1992; 73(4): 393-397.
- Mahjneh, I., Saarinen, A., Siivola, J. Familial carpal tunnel syndrome: a report of a Finnish family. *Acta Neurol Scand*. 2001; 104: 377-379.
- Michaud, L.J., Hays, R.M., Dudgeon, B.J., Kropp, R.J. Congenital carpal tunnel syndrome: case report of autosomal dominant inheritance and review of the literature. *Arch Phys Med Rehabil*. 1990; 71(6): 430-432.
- Radecki, P. The familial occurrence of carpal tunnel syndrome. *Muscle Nerve*. 1994; 17(3): 325-330.
- Stoll, C., Maitrot, D. Autosomal dominant carpal tunnel syndrome. *Clin Genet*. 1998; 54(4): 345-348.
- Vadasz, A., Chance, P.F., Epstein, L.G., Lou, J. Familial autosomal-dominant carpal tunnel syndrome presenting in a 5-year-old-case report and review of the literature. *Muscle Nerve*. 1997; 20(3): 376-378.
- Vallat, J.M., Dunoyer, J. Familial carpal tunnel syndrome [abstract]. *Sem Hop*. 1978; 54(17-20): 661-662.

