Published in final edited form as:
J Occup Environ Med. 2019 April ; 61(4): 340–346. doi:10.1097/JOM.0000000000001558.

Role of Biomechanical Factors in Resolution of Carpal Tunnel Syndrome among a Population of Workers

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Abstract

Objectives: Identify if CTS symptoms vary by measured biomechanical exposures.

Methods: A nested prospective cohort within a large, multi-center, 8-year cohort study. The CTS case definition was tingling/numbness in 2+ median nerve-served digits plus a nerve conduction study consistent with CTS. Workers were assigned to: 1) low (Strain Index (SI) ≤6.1), and 2) high (SI >6.1) job groups.

Results: Among 1,201 workers, 106 had CTS. Those in the high SI group became and remained symptom-free for at least three months, faster than the low SI group, adjusted **HR= 2.07 (95%CI=1.21 – 3.56, p=0.008)**. Only surgical release trended towards resolving CTS. Light duty had no impact, and job change was associated with delayed symptoms resolution.

Conclusions: High biomechanical exposures paradoxically predicted faster improvement in CTS and light duty did not result in symptom resolution.

Keywords

Carpal tunnel syndrome; Modified duty; Light duty; Biomechanics; Prognosis; Epidemiology; Treatment

Introduction

Carpal tunnel syndrome (CTS) is the most common peripheral nerve entrapment (1). The reported CTS prevalence estimates range from 2.7% to 13.0%(2–14)The incidence rate between 2.3 and 7.5 cases per 100 person-years (15, 16).

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Each author worked at their respective institutions (see above). Level of Evidence: Level II, Prospective Cohort Study

Conflicts of interest: None declared.

Reported, non-occupational risk factors for CTS include age, female sex, body mass index (BMI), biopsychosocial factors, diabetes, smoking, Framingham heart risk scores, wrist depth/width ratio, and genetics (17–26). There are also reported associations with work involving combinations of high force and repetition (27–29), and there is some evidence for increased risk with high amplitude vibrating hand tools (24, 25, 30). In contrast, no consistent associations have been found for work activities that involve awkward postures, keyboards, cold environments, and increasing length of employment (24, 29, 30). Despite being one of the most prevalent musculoskeletal disorders among workers, the natural progression of CTS has not been well described (31), especially in working populations.

The few available reports on the natural history of CTS have somewhat discrepant outcomes, either ignoring or weakly addressing whether job physical factors modify the clinical course of CTS (32–34). Zyluk et. al. (2017), suggested that the clinical progression of CTS is unpredictable and characterized by electrophysiological disturbances (34).Early attempts to describe the natural progression of CTS suggest that severity of its associated symptoms and prognosis are related to the duration of the condition (33, 35). An Italian CTS Study Group followed 196 CTS prevalent individuals from the general population for 15 months and concluded that untreated idiopathic CTS spontaneously improves clinically and electrophysiologically with time when severe, but tends to worsen when the initial presentation is mild (1). This group also found that neurophysiologic measurements followed a similar but non-statistically significant pattern (36). Other studies have found poor correlations between clinical course changes and electrodiagnostic findings (37, 38). A study by Nathan et al. (1998) followed 289 workers for 11 years and found that nerve conduction findings tended to worsen over time without necessarily leading to worsening CTS symptoms or increases in prevalence (39). Silverstein et al. (2010), found that workers with either current symptoms of CTS or positive electrodiagnostic findings at baseline were more likely to meet the case definition for CTS and persist as a CTS case at one year (15).

Thus, it is unclear what role biomechanical exposures might play in the progression of CTS. It is also unknown whether job restrictions to reduce exposures are effective. The objective of this study was to determine whether the clinical symptoms among individuals with CTS vary depending on biomechanical exposures as measured by the Strain Index (SI).

Methods

This report is a nested cohort study within a large, multi-state, multi-center, prospective cohort study (the WISTAH Study). Brief methods follow, as the study methods have been previously published (40). All participants were consented and Institutional Review Board approval was obtained.

Workers were recruited from 17 companies with multiple facilities for a broad variety of tasks in Wisconsin, Utah and Illinois. Workers underwent separate health and job physical exposure assessments at baseline and during follow up by two teams of researchers that were blinded.

Baseline health assessments were performed by a health outcomes assessment team and consisted of a computerized questionnaire, structured interview, measured biometrics (e.g., BMI, blood pressure), two standardized physical examinations and nerve conduction studies (NCS).

Questionnaires included demographics, medical history, psychosocial factors, social factors, and off-work activities. The structured interview and the first of two standardized physical examinations were performed by an occupational medicine resident, occupational or physical therapist. The structured interview included symptoms of: (i) current tingling and/or numbness in each digit, (ii) tingling and/or numbness in each digit in the prior month, (iii) duration of symptoms represented as the percentage of time during the past month, and (iv) any history of other upper extremity musculoskeletal symptoms or neuropathic disorders.

The first of the two standardized physical exams was naturally unblinded to symptoms and immediately followed the structured interview. The second exam was performed by a board-certified occupational medicine physician who was unblinded to the prior results and assessed the symptoms and pertinent examination negatives. Examinations consisted of observation and inspection of the distal upper extremities, palpation, range of motion, and specific examination maneuvers (e.g., Phalen's and Hoffmann-Tinel's).

NCS was administered to all workers at baseline regardless of symptoms using conventional NCS equipment and contemporaneous standards (6). All NCS were conducted by one of two board-certified physiatrists who were blinded to job exposures and symptoms. NCSs were performed according to standard electrodiagnostic testing protocols. NCS data collected included peak median sensory nerve latency, onset median motor nerve latency, and the difference between the median and ulnar nerve sensory latencies across the wrist (transcarpal delta). Sensory latency values were measured at a distance of 14cm. Median sensory measures were antidromic and performed at 6cm. Transcarpal deltas were performed at 8cm. Skin temperature was measured prior to testing, and hands were warmed to a minimum skin temperature of 32°C.

Those few workers with evidence of both median and ulnar neuropathy were excluded as having presumptive evidence of polyneuropathy.

Workers subsequently underwent monthly follow-ups with structured interviews and standardized physical examinations over a period of up to nine years. Monthly structured interviews included queries for treatments, including medications, therapy, exercise, injections, and surgery. Monthly structured interviews also included restricted, or "light duty." Workers leaving employment were not replaced.

Job exposure assessments were conducted by a separate job exposure assessment team of trained ergonomists. Job assessment data included an interview regarding job factors, job physical factors measurements, observation and video recording of all tasks. Videotapes were subsequently analyzed and variables measured (the percentage of time a given hand was meaningfully engaged in work activities) (28). Composite job exposure measures were calculated, including the Strain Index score (41). SI is semi-quantitative and includes 6 variables: force, repetition, duration of exertion, hand/wrist posture, speed of work and shift length. The SI score is weighted with an assigned multiplier for each of the six variables, which are then multiplied together to produce the composite SI score. Force is the most heavily weighted variable in the SI. The SI scores were classified into low risk (SI < 6) and high risk (SI ≥ 6) (42). Jobs were also reassessed quarterly by the job exposure assessment team. If the job changed, it was re-measured and re-analyzed.

The case definition of CTS was (40): (1) presence of tingling and/or numbness in two or more median-served digits; (2) symptoms present on at least 25% of the days during the prior month; (3) symptoms occurring for at least two or more consecutive follow up periods; and (4) abnormal NCS consistent with median mononeuropathy. Additionally, a worker was automatically designated as a case if the worker had undergone surgical CTS release.

For the purpose of this nested cohort study, all workers who met the requirements for the case definition of CTS at baseline were included. Workers with possible polyneuropathy (i.e., abnormal median and ulnar nerve conduction) on NCS were excluded.

Statistical Analyses

Participants were assigned to two groups: 1) low risk (SI ≤6.1), and 2) high risk (SI >6.1). Time-to-event analyses were performed. The main outcome was time to achieve symptom-free status, with a secondary outcome of time to achieve symptom-free status for at least three consecutive months. All analyses were performed using Stata 13.1 (Stata Statistical Software: Release 13, College Station, TX).

Descriptive statistics were performed. Multivariate models were constructed including SI as the main independent variable. Other covariates (BMI, Framingham cardiovascular risk score, history of thyroid disease) were introduced into the model to control for potential confounding. Cox proportional hazards regression was used to model time-to-outcome event.

Source of Funding

This work was supported in part by grants from the National Institute for Occupational Safety and Health (1R01OH010474-01 and 3TC42OH008414). No funding agency contributed to nor was consulted in the research or writing of this manuscript.

Results

Table 1 provides the demographic characteristics of the study sample at baseline. A total of 1,201 workers were initially enrolled. A small number, 15 (1.2%), were excluded because of presumptive polyneuropathy findings at baseline. The mean age of the workers was 42.17 years (CI 41.53 – 42.18). Most, 796 (66.2%), were women. This is a working population and thus it is unsurprising that only 62 (5.1%) had diabetes. A total of 83 (6.9%) had thyroid disease. Many workers, 264 (21.9%), had tingling and/or numbness in two or more median served digits, whereas 225 (18.7%) presented NCS findings consistent with median mononeuropathy. A total of 109 (9.1%) workers met the case definition of both paresthesia and abnormal NCS findings at baseline, however, 3 were excluded due to being lost before the first follow-up structured interview. A total of 106 workers were included in the final statistical analyses, with 48 (45.3%) having mild CTS while 58 (54.7%) having moderate/ severe CTS based on NCS findings.

The 106 workers were assigned to two groups based on their SI scores. A modest majority of 59 (55.6%) were in the low; there were 47 (44.3%) in the high SI group. Survival analyses demonstrated that out of the 106 followed workers, all but 1 eventually became symptom free during follow up.

The median time to symptoms-free status was 235.5 days, with a minimum of 35 days, and a maximum of 1811 days. The 50^{th} percentile for time-to-event was 167 days. (Tables 2–3.)

When analyzing the demographic characteristics of workers with CTS at baseline, we found that there were no differences between groups with respect to sex, ($x^2 = 0.08$, $p = 0.76$), BMI (p=0.76), and tobacco use ($x^2 = 3.61$, p=0.16). Predictably, the high SI group was 6.1 years younger than the low SI group (p=0.001), as younger workers tend to perform more demanding jobs.

Hazard ratios for time to symptom-free status were calculated by Cox proportional hazard analyses (see Figure 1). When controlling for BMI, Framingham score, and thyroid disease, workers in the high SI score group became symptom-free faster, HR= 1.75 (95%CI=1.14 – 2.68, p=0.01).

Figures 2 illustrates time to at least three months of symptom-free status. Similarly, after controlling for BMI, Framingham score, and thyroid disease, workers with high SI scores became and remained symptom-free for at least three months faster than the low SI score group, HR= 1.82 (95%CI=1.10 – 2.99, p=0.01).

When treatment for CTS (NSAID, wrist brace, physical therapy and/or occupational therapy, steroid injections, surgery and "other") and job modification ("light duty" and job change) were included in the model, again, individuals with high SI score became symptom-free faster and remained symptom-free for at least three months, HR= 2.07 (95%CI=1.21 – 3.56, p=0.008).

When analyzing reported treatments among the 106 workers with CTS, 36 (33.9%) denied any treatment. Thirty-one (29.2%) reported NSAID intake, 33 (31.1%) wore a wrist brace, 11 (10.3%) participated in physical or occupational therapy, only 3 (2.8%) acknowledged a steroid injection, and 6 (5.6%) underwent surgical release; and (22.6%) 24 reported other treatments. Among the 106 workers with CTS, 32 received only one type of treatment, 22 workers received two different treatments, 7 received three types of treatment, and 1 had four types of treatment (see Table 4).

When treatment count and surgical release were included in the final model (Table 5), again, individuals with high SI score were more likely to become and remain symptom-free for at least three months compared with the low SI score group, HR= 1.83 (95%CI=1.10 – 3.04, p=0.01). For the final regression model, 7 workers were eliminated because of missing covariate data. Treatments including NSAID intake, wrist brace, physical and/or occupational therapy, steroid injections, and "other" appeared to not influence symptom resolution. The only treatment that appeared to potentially play a role in the resolution of CTS was surgical release, HR 2.91 (95%CI 1.08 – 7.87, p=0.03), however, this was only true when controlling for the other non-surgical treatments. With regards to job modification, light duty had no significant impact on CTS improvement, HR= 1.05 (95%CI=0.38 – 2.92, p=0.91, whereas job change was associated with delayed symptom resolution, HR= 0.56 (95%CI=0.37 – 0.87, p=0.009).

Discussion

This is the first study to describe the impacts of measured job physical factors on the natural course of CTS in a cohort of workers. These findings suggest that despite increasing risk of CTS associated with increasing biomechanical exposures in prior publications (26, 29), rather the exposure to high Strain Index scores was associated with faster resolution of CTS symptoms. Regardless of exposure, nearly all workers became symptom free and 66.7% became symptom-free for at least a three-month duration. While some have advocated modified duty for workers with CTS, this study also failed to find potential benefits in modified or light duty work.

Some of these findings could be compared to those of the Italian CTS Study group (33), which concluded that untreated idiopathic CTS spontaneously improves with time when severe; however, that study did not include biomechanical factors and most of the workers did not have severe CTS. It could be said, however, that regardless of biomechanical exposure, CTS symptoms tend to spontaneously resolve in both working and general populations. Yet, in the Italian study roughly 25% of the subjects spontaneously improved, 25% worsened, and 50% remained unchanged. In terms of neurophysiologic results, the Italian study found that for every unit increment in electrodiagnostic score, the odds of symptoms improving were roughly one-and-half times greater (OR 1.7, CI 1.1–2.6). Our study did not evaluate the course of neuroelectrical activity.

The findings of our study appear to contradict those of (15), which found that more than half (55.6%) of workers with CTS had not recovered after one year. While not studying the impact of job modifications, the authors of that study nevertheless speculated that job modification(s) appear necessary to help such workers improve their symptoms; yet our study suggests that job modification does not influence the clinical course of CTS. Our study did not analyze for subsequent events and thus, we do not know how many and how fast such individuals may have had symptom recurrences. Our study supports the idea that the natural history of CTS is highly variable, also suggesting that only a minority of affected individuals progress to becoming a surgical case.

Our study also found that among the reported treatments only surgical release trended towards providing relief, while other treatments in aggregate appeared to delay resolution of CTS symptoms. NSAIDs use was significantly related to slower resolution of symptoms. That finding is expected as peer-reviewed American College of Occupational and Environmental Medicine’s (ACOEM) evidence-based guidelines have concluded that NSAIDs are ineffective for treatment compared with placebo (24).

We are unable to assess the impacts of the use of steroid injections, which per the ACOEM Guidelines have strong evidence of efficacy in randomized controlled trials for treating CTS. The apparent lack of efficacy of steroid injections in our study is likely due to underpowering, as only 3 workers had steroid injections.

This study's strengths include a large sample size (1,201) drawn from 17 different employers in three diverse states with monthly follow-ups and the objective measurement of individual job exposures provides unique statistical power. This study's demographics (e.g., age, BMI, smoking status, thyroid disease) suggest generalizability to other employed populations. Long-term follow up and relatively low dropouts also improved precision. Computerization of the questionnaires and structured interviews provided nearly complete data. Standardization and blinding of the NCS decreased the risk of operator-dependent results and evaluator biases. This study's analyses again support that cardiovascular disease risk factors as modeled by the Framingham risk score have a meaningful role in the development of CTS (23), although they do not appear to play a demonstrable role in the achievement symptom-free status.

Limitations include potential underpowering for more severe cases, and underpowering of select treatments. The lack of randomization could have resulted in an unrecognized residual confounder being present, although many risk factors were controlled in these analyses including components of the Framingham risk scores. Additionally, this study did not analyze for subsequent events, so it is unclear whether CTS recurrences may subsequently differ by biomechanical exposures. Multiple covariates including sex, smoking, and BMI were not different among comparison groups, which leads us to conclude that they did not influence or confound the results. Ultrasound or other imaging was not included due to fiscal constraints, although it is unclear whether that would have influenced these results.

Conclusion

This study's results are unexpected, as we anticipated that low biomechanical exposures would result in faster resolution of CTS symptoms, especially as there is considerable evidence that the combination of high force and high repetition (including as measured by the SI score) results in higher risk for CTS. On the contrary, the results suggest that high biomechanical occupational exposures are potentially even protective and predict faster improvement in working populations. Among reported treatments, only surgical release trended towards providing relief of CTS symptoms.

The results of this study suggest that modification of job duties, either light duty or job change, may be unsuccessful for treatment of CTS, although a randomized trial would be required to definitively answer this question.

Acknowledgments

Sources of funding: Funded, in part, by CDC/NIOSH Research grant 1 U 01 OH007917-01 and CDC/NIOSH Training grant 3TC42OH008414.

References

1. Padua L, Coraci D, Erra C, et al. Carpal tunnel syndrome: clinical features, diagnosis, and management. The Lancet Neurology. 2016;15:1273–1284. [PubMed: 27751557]
2. Armstrong T, Dale AM, Franzblau A, Evanoff BA. Risk factors for carpal tunnel syndrome and median neuropathy in a working population. J Occup Environ Med. 2008;50:1355–1364. [PubMed: 19092490]
3. Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosen I. Prevalence of carpal tunnel syndrome in a general population. JAMA. 1999;282:153–158. [PubMed: 10411196]
4. Charles N, Vial C, Chauplannaz G, Bady B. Clinical validation of antidromic stimulation of the ring finger in early electrodiagnosis of mild carpal tunnel syndrome. Electroencephalogr Clin Neurophysiol. 1990;76:142–147. [PubMed: 1697242]
5. Hegmann KT, Garg A, Wertsch J, MS T Prevalence of Median Nerve Related Symptoms and Signs in a Cohort at Baseline. Presented at the Fifth International Scientific Conference on Prevention of Work-related Musculoskeletal Disorders ETH Zurich, Zurich, Switzerland July 13, 2004 PREMUS 2004:485–486.
6. Jablecki CK, Andary MT, Floeter MK, et al. WITHDRAWN: Second AAEM literature review of the usefulness of nerve conduction studies and needle electromyography for the evaluation of patients with carpal tunnel syndrome. Muscle Nerve. 2002.
7. Makanji H, Zhao M, Mudgal C, Jupiter J, Ring D. Correspondence between clinical presentation and electrophysiological testing for potential carpal tunnel syndrome. J Hand Surg. 2013;38:489–495.
8. Salerno DF, Franzblau A, Werner RA, Bromberg MB, Armstrong TJ, Albers JW. Median and ulnar nerve conduction studies among workers: normative values. Muscle Nerve. 1998;21:999–1005. [PubMed: 9655117]
9. Seror P Sensitivity of the various tests for the diagnosis of carpal tunnel syndrome. J Hand Surg (British and European Volume). 1994;19:725–728.
10. Stevens JC. AAEE minimonograph #26: The electrodiagnosis of carpal tunnel syndrome. Muscle Nerve. 1987;10:99–113. [PubMed: 3821791]
11. Uncini A, Di Muzio A, Awad J, Manente G, Tafuro M, Gambi D. Sensitivity of three median-to-ulnar comparative tests in diagnosis of mild carpal tunnel syndrome. Muscle Nerve. 1993;16:1366–1373. [PubMed: 8232394]
12. Werner RA, Franzblau A, Gell N, Hartigan AG, Ebersole M, Armstrong TJ. Incidence of carpal tunnel syndrome among automobile assembly workers and assessment of risk factors. J Occup Environ Med. 2005;47:1044–1050. [PubMed: 16217245]
13. Wong S, Griffith J, Hui A, Tang A, Wong K. Discriminatory sonographic criteria for the diagnosis of carpal tunnel syndrome. Arthritis & Rheumatism. 2002;46:1914–1921. [PubMed: 12124876]
14. Wong SM, Griffith JF, Hui AC, Lo SK, Fu M, Wong KS. Carpal Tunnel Syndrome: Diagnostic Usefulness of Sonography 1. Radiology. 2004;232:93–99. [PubMed: 15155897]
15. Silverstein BA, Fan ZJ, Bonauto DK, et al. The natural course of carpal tunnel syndrome in a working population. Scand J Work Environ Health. 2010:384–393. [PubMed: 20352175]
16. Dale AM, Harris-Adamson C, Rempel D, et al. Prevalence and incidence of carpal tunnel syndrome in US working populations: pooled analysis of six prospective studies. Scand J Work Environ Health. 2013;39:495. [PubMed: 23423472]
17. Nathan P, Keniston R. Carpal tunnel syndrome and its relation to general physical condition. Hand Clinics. 1993;9:253–261. [PubMed: 8509465]
18. Nathan PA, Meadows KD, Istvan JA. Predictors of carpal tunnel syndrome: an 11-year study of industrial workers. J Hand Surg. 2002;27:644–651.
19. Kouyoumdjian JA, Zanetta DM, Morita MP. Evaluation of age, body mass index, and wrist index as risk factors for carpal tunnel syndrome severity. Muscle & Nerve. 2002;25:93–97. [PubMed: 11754190]
20. Keir PJ, Rempel DM. Pathomechanics of peripheral nerve loading: evidence in carpal tunnel syndrome. J Hand Therapy. 2005;18:259–269.

21. Van Dijk MA, Reitsma JB, Fischer JC, Sanders GT. Indications for requesting laboratory tests for concurrent diseases in patients with carpal tunnel syndrome: a systematic review. Clin Chem. 2003;49:1437–1444. [PubMed: 12928223]
22. Harris-Adamson C, Eisen EA, Neophytou A, et al. Biomechanical and psychosocial exposures are independent risk factors for carpal tunnel syndrome: assessment of confounding using causal diagrams. Occup Environ Med. 2016;73:727–734. [PubMed: 27466616]
23. Hegmann KT, Thiese MS, Kapellusch J, et al. Association between cardiovascular risk factors and carpal tunnel syndrome in pooled occupational cohorts. J Occup Environ Med. 2016;58:87–93. [PubMed: 26716852]
24. ACOEM Practice Guidelines: Hand, Wrist, and Forearm Disorders Guideline. Elk Grove Village, IL: American College of Occupational and Environmental Medicine; 2018.
25. Talmage JB, Hyman Mark H., Melhorn J. Mark, Ackerman William E.,. AMA Guides to the Evaluation of Disease and Injury Causation. Chicago, IL: American Medical Association; 2013.
26. Harris-Adamson C, Eisen EA, Dale AM, et al. Personal and workplace psychosocial risk factors for carpal tunnel syndrome: a pooled study cohort. Occup Environ Med. 2013:oemed-2013–101365.
27. Palmer KT. Carpal tunnel syndrome: the role of occupational factors. Best Pract Res Clin Rheumatol. 2011;25:15–29. [PubMed: 21663847]
28. Garg A, Kapellusch J, Hegmann K, et al. The Strain Index (SI) and Threshold Limit Value (TLV) for Hand Activity Level (HAL): risk of carpal tunnelsyndrome (CTS) in a prospective cohort. Ergonomics. 2012;55:396–414. [PubMed: 22397385]
29. Harris-Adamson C, Eisen EA, Kapellusch J, et al. Biomechanical risk factors for carpal tunnel syndrome: a pooled study of 2474 workers. Occup Environ Med. 2015;72:33–41. [PubMed: 25324489]
30. Palmer KT, Harris EC, Coggon D. Carpal tunnel syndrome and its relation to occupation: a systematic literature review. Occup Med. 2007;57:57–66.
31. Resende L, Tahara A, Fonseca RG, Sardenberg T. The natural history of carpal tunnel syndrome. A study of 20 hands evaluated 4 to 9 years after initial diagnosis. Electromyography Clinical Neurophysiol. 2003;43:301–304.
32. Carlson H, Colbert A, Frydl J, Arnall E, Elliot M, Carlson N. Current options for nonsurgical management of carpal tunnel syndrome. Int J Clin Rheumatol. 2010;5:129.
33. Puchalski P, Zyluk A, Zyluk-Gadowska P. An analysis of the course of carpal tunnel syndrome before operation. Acta Orthopaedica Belgica. 2017;83:22–29. [PubMed: 29322890]
34. Zyluk A, Puchalski P. Natural history of carpal tunnel syndrome--a review. Chirurgia narzadow ruchu i ortopedia polska. 2010;75:261–266. [PubMed: 21375035]
35. DeStefano F, Nordstrom DL, Vierkant RA. Long-term symptom outcomes of carpal tunnel syndrome and its treatment. J Hand Surgery. 1997;22:200–210.
36. Padua L, Padua R, Aprile I, Pasqualetti P, Tonali P. Multiperspective follow-up of untreated carpal tunnel syndrome A multicenter study. Neurology. 2001;56:1459–1466. [PubMed: 11402101]
37. Ortiz-Corredor F, Enríquez F, Díaz-Ruíz J, Calambas N. Natural evolution of carpal tunnel syndrome in untreated patients. Clin Neurophysiol. 2008;119:1373–1378. [PubMed: 18396098]
38. Hardoim DGV, Oliveira GBd, Kouyoumdjian JA. Carpal tunnel syndrome: long-term nerve conduction studies in 261 hands. Arquivos de neuro-psiquiatria. 2009;67:69–73. [PubMed: 19330215]
39. Nathan PA, Keniston RC, Myers LD, Meadows KD, Lockwood RS. Natural history of median nerve sensory conduction in industry: relationship to symptoms and carpal tunnel syndrome in 558 hands over 11 years. Muscle & Nerve. 1998;21:711–721. [PubMed: 9585324]
40. Garg A, Hegmann KT, Wertsch JJ, et al. The WISTAH hand study: a prospective cohort study of distal upper extremity musculoskeletal disorders. BMC Musculoskel Disord. 2012;13:90.
41. Steven Moore J, Garg A. The strain index: a proposed method to analyze jobs for risk of distal upper extremity disorders. Am Indus Hyg Assoc J. 1995;56:443–458.
42. Moore J, Vos G, Stephens J, Stevens E, Garg A. The validity and reliability of the Strain Index. Proceedings of the 16th World Congress on Ergonomics (IEA’06); 2006.

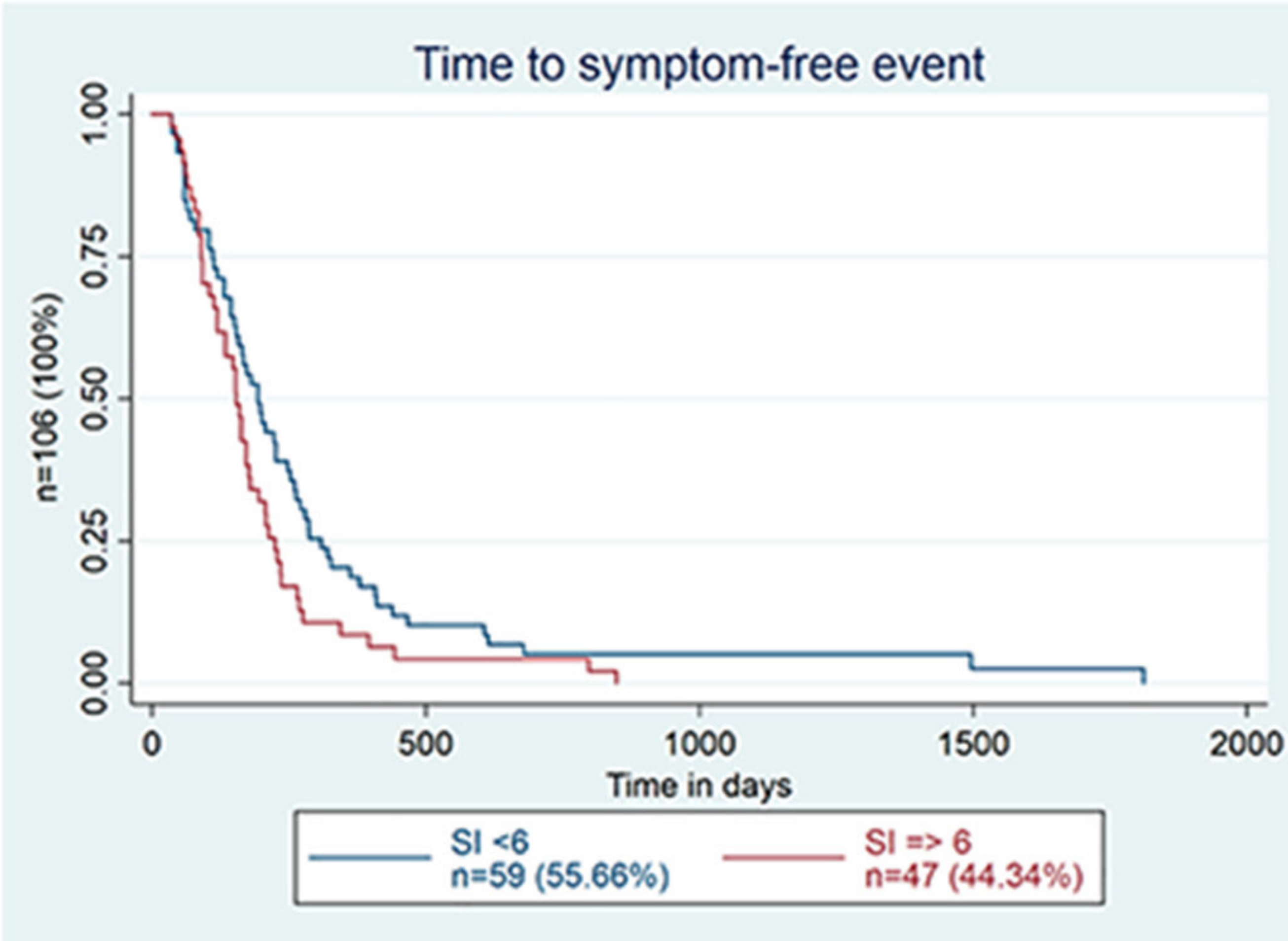

Figure 1.

Time to One-month Symptom-free Duration Stratified by High (=>6.0) compared with Low (<6.0) Strain Index.

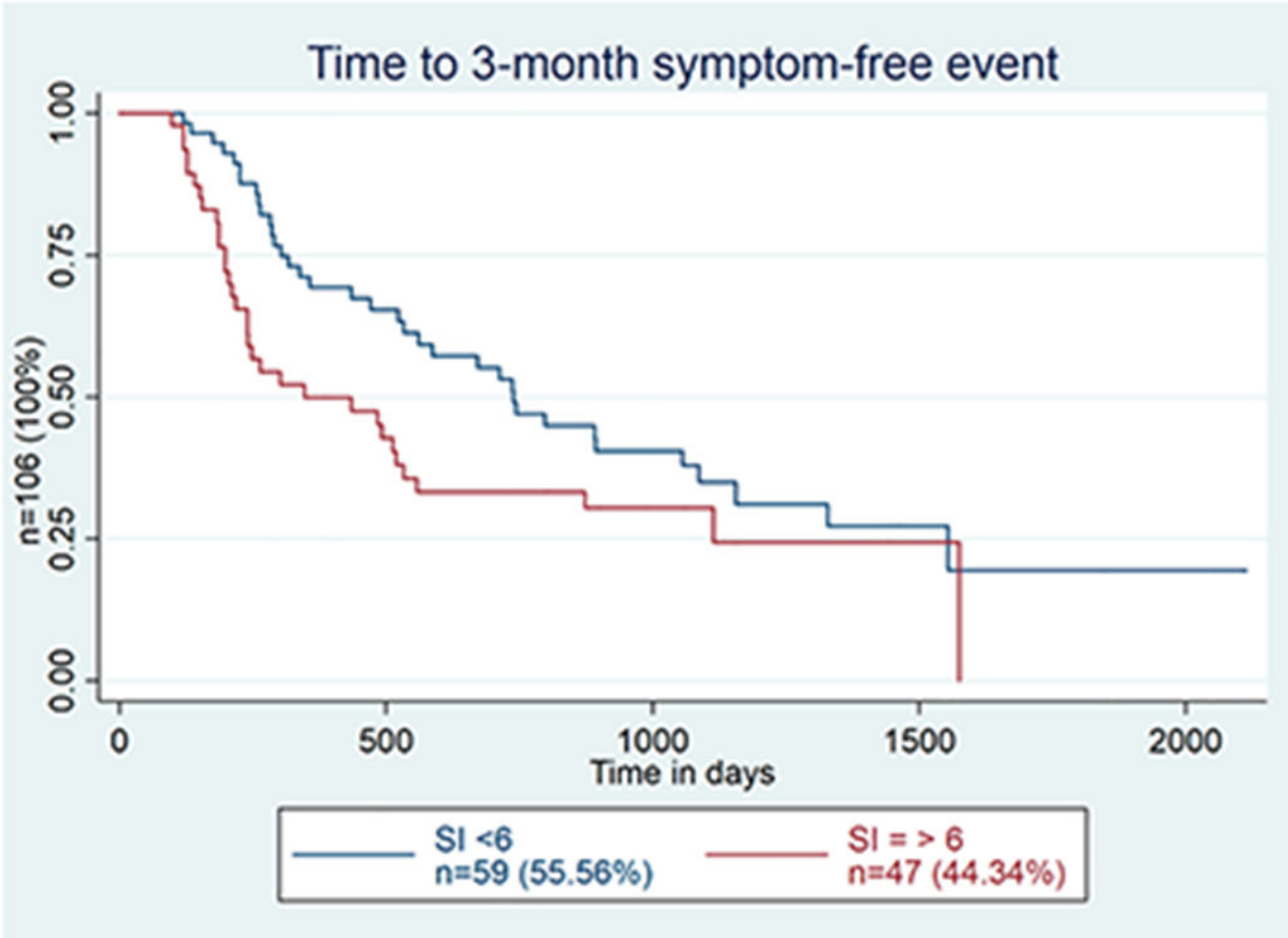

Figure 2.

Time to Three-month Symptoms-free Duration Stratified by SI Exposure.

Table 1.

Demographic variables for the total cohort and the workers with CTS.

	Total n (%)	No CTS at baseline n (%)	CTS at baseline n (%)
	n=1201 (100%)	**n= 1092 (91%)**	**n=106[*] (9%)**
Age mean (CI)[1]	42.17 (41.53 – 42.81)	41.70 (41.02 – 42.38)	46.87 (45.00 – 48.74)
Sex			
Female	796 (66.28%)	712 (65.20%)	82 (77.36%)
Male	405 (33.72%)	380 (34.80%)	24 (22.64%)
Strain index score			
Low risk <6	647 (53.87%)	586 (53.66%)	59 (55.66%)
High risk ≥6	554 (46.13%)	506 (46.34%)	47 (44.34%)
Paresthesia[^]			
No	937 (78.02%)	937 (85.81%)	0
Yes	264 (21.98%)	116 (10.62%)	106 (100%)
Nerve conduction study			
Normal	976 (81.27%)	976 (89.38%)	0
Abnormal	225 (18.73%)	116 (10.62)	106 (100%)
Body mass index mean kg/m^2 (CI)	29.47 (29.08 – 28.85)	29.07 (28.69 – 29.46)	33.43 (31.87 – 34.98)
Medical history			
Diabetes mellitus	62 (5.16%)	49 (4.49%)	13 (11.93%)
Thyroid disease	83 (6.91%)	71 (6.5%)	12 (11.01%)
Framingham score mean (CI)	6.37 (6.08 – 6.64)	6.11 (5.82 – 6.41%)	8.93 (7.8 – 9.93)

[1] Confidence interval

[*] 106 after 3 workers excluded for incomplete data.

[^] Tingling and/or numbness in ≥ 2 median nerve-served digits

Table 2.

Cox regression analyses for time to one-month symptom-free status.*

	HR	*p*	95% CI
Strain Index Score	1.75	0.01	1.14 – 2.68
Framingham Score	1.04	0.02	1.00 – 1.08
Thyroid Disease	1.32	0.3	0.71 – 2.47
Body Mass Index	1.00	0.8	0.98 – 1.02

*Framingham score includes age, sex and diabetes mellitus.

Table 3.

Time to symptoms free status of at least three-months duration.*

	HR	*p*	95% CI
Strain Index Score	1.82	0.01	1.10 – 2.99
Framingham Score	1.03	0.1	0.98 – 1.08
Thyroid Disease	1.11	0.7	0.56 – 2.19
Body Mass Index	1.00	0.4	0.98 – 1.03

*Framingham score includes age, sex and diabetes mellitus.

Table 4.

Effects of CTS treatments on attainment of 3-month symptoms-free status.

	n= total	Symptom-free for three months	No Symptom-free for three months
	n= 105* * one worker missing	n= 70	n=35 (1 missing)
Sex			
Female	81*	57	24*
Male	24	13	11
Missing data	1		1
Strain index			
Low risk <6	58*	37	21
High risk ≥6	47	33	14
Missing data	2		2
NSAID's			
No	68	55	13
Yes	31	13	18
Missing data	7	2	5
Wrist brace			
No	66	50	16
Yes	33	18	15
Missing data	7	2	5
Physical/occupational therapy			
No	88	63	25
Yes	11	5	6
Missing data	7	2	5
Steroid injection			
No	96	67	29
Yes	3	1	2
Missing data	7	2	5
Other treatment			
No	74	50	24
Yes	24	18	6
Missing data	8	2	6
Surgical release			
No	93	63	30
Yes	6	5	1
Missing data	7	2	5
Number of treatments received			
None	36	32	4
One	32	20	12
Two	22	13	9
Three	7	3	4

	n= total	Symptom-free for three months	No Symptom-free for three months
	n= 105* * one worker missing	n= 70	n=35 (1 missing)
Four	1		1
Missing data	8	2	6

Table 5.

Time to three-month symptom-free status controlling for female sex, BMI, Framingham cardiovascular risk score, number of types of treatment(s) and surgical release.*

	HR	*p*	95% CI
Strain Index	1.834	0.0189	1.105 – 3.045
Female sex	1.096	0.774	0585 – 2.052
Framingham score	1.059	0.0252	1.007 – 1.114
BMI	1.032	0.0281	1.003 – 1.062
Treatment count	0.470	<.0001	0.338 – 0.654
Surgical release	2.916	0.0346	1.081 – 7.870

*Framingham score includes age, sex and diabetes mellitus.