

Research article

Impact of Occupational Exposure Duration on Electrophysiological Changes of the Median and Ulnar Nerves in Computer Users

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Citation: Vulpoi G.A., Bistriceanu C.E., Bîrsanu L., Palade C.M., Cuciureanu D.I. - Impact of Occupational Exposure Duration on Electrophysiological Changes of the Median and Ulnar Nerves in Computer Users
Balneo and PRM Research Journal
2026, 17(1): 989

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Academic Editor(s):
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Production Officer:
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Received: 08.02.2026
Published: 31.03.2026

Reviewers:
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Abstract: Repetitive movements and sustained non-neutral upper limb postures during computer use may increase the risk of compressive neuropathies. This study evaluates median and ulnar nerves electrophysiological parameters in occupational computer users and examines their association with exposure duration and impairment severity. This prospective observational study included 40 patients with clinically suspected and electrophysiological confirmed carpal tunnel syndrome (CTS). Standard sensory and motor nerve conduction studies of the median and ulnar nerves were performed, including median-ulnar distal motor latency comparison using the second lumbrical–first interosseous muscles (L2–IO1) technique. A moderate, statistically significant positive correlation was found between years of occupational exposure and increased median nerve distal motor latency (DML) at the wrist. In this clinically referred cohort, exposure duration was significantly associated with moderate-to-severe CTS (OR = 2.42 per 5-year increase) and remained significant after adjustment for age and comorbidity status (adjusted OR = 2.08). In our cohort, occupational exposure duration remained an independent determinant of CTS electrophysiological severity after adjustment for age, sex, and comorbidities. These exploratory findings suggest that cumulative occupational exposure may be linked to CTS severity beyond aging, but implications for occupational screening should be interpreted cautiously and require prospective external validation..

Keywords: Carpal tunnel syndrome, computer users, nerve conduction studies

1. Introduction

Carpal tunnel syndrome (CTS), caused by median nerve compression at the wrist, is the most common compressive neuropathy worldwide, representing approximately 90% of all such neuropathies [1]. Atroshi et al. reported that approximately one in five symptomatic individuals met the diagnostic criteria for CTS based on combined clinical and electrophysiological assessment, including nerve conduction studies (NCS), corresponding to a prevalence of 2.7% [2]. The reported prevalence of the condition shows substantial variability across the literature, with consistently higher rates in women and a positive association with increasing age [3,4].

Numerous studies indicate that awkward postures and repetitive hand/arm movements are factors that increase the risk of upper limb musculoskeletal disorders, particularly CTS, among workers who operate computers [5–7]. Several studies report

an annual CTS prevalence of about 11.7% among computer users, suggesting an association with prolonged computer use [8]. A recent study by Feng B. et al. reported a clinically confirmed CTS prevalence of 9.6% among computer operators [7]. Computer use has not been conclusively proven to directly cause upper limb compressive neuropathies, but rather acts as a contributing factor within a multifactorial risk framework [9].

Although occupational activities are an important risk factor for CTS, its etiology is multifactorial, involving systemic and individual factors such as obesity, genetic predisposition, diabetes, rheumatoid arthritis, hypothyroidism, and pregnancy [1,10]. The literature has proposed an integrative “triad of susceptibility” model that provides a framework for understanding how genetic factors, immunological mechanisms, and environmental exposures interact to drive median nerve changes within the carpal tunnel. Within this model, genetic variation may influence connective tissue properties and oxidative stress responses, immune pathways may contribute to low-grade inflammation and neural sensitization, and environmental factors may act as key triggers for onset and progression [11].

Clinically, CTS presents with numbness and paraesthesia in the median nerve distribution, affecting the first three digits and the radial half of the fourth digit. Symptoms are initially intermittent, often nocturnal, and may become persistent over time, with progression to wrist pain radiating to the elbow or shoulder and thenar muscle atrophy. In advanced stages, patients may report sensory symptoms extending beyond the median nerve territory, including paraesthesia in all five digits or pain radiating into the forearm [12,13].

Several validated questionnaires are available for the evaluation of CTS patients. The Boston Carpal Tunnel Questionnaire (BCTQ) is the most used tool, assessing both symptom severity and functional limitation, and has been validated in numerous languages [14–17]. The Six-Item Carpal Tunnel Syndrome Scale (CTS-6), introduced by Atroshi et al. in 2011, represents a concise version of the BCTQ and has also been widely translated and validated [18–22].

The pathophysiology of CTS is primarily related to increased pressure within the carpal tunnel. Wrist extension or flexion can raise intracarpal pressure by 8–10 times above normal values, leading to repetitive compression of the median nerve [1]. From an electrophysiological perspective, early nerve compression is associated with focal demyelination, manifested by prolonged distal motor and sensory latencies and reduced sensory nerve conduction velocity (NCV). With persistent or severe compression, axonal involvement may occur, resulting in decreased compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes. Electrodiagnostic (EDX) studies provides an objective assessment of median nerve dysfunction and lesion severity in CTS, offering prognostic information, as clinical symptoms may not accurately reflect the extent of nerve damage [1].

Previous studies conducted in asymptomatic subjects have shown that NCV of both the median and ulnar nerves were significantly delayed in the dominant arm of computer users compared with control subjects [23]. Other studies have examined cohorts of computer operators with and without upper limb complaints affecting the dominant extremity. The authors concluded that frequent mouse use is associated with an increased risk of developing median neuropathy at the wrist (MNW) [24].

In this study, distal motor latencies (DML) of the median nerve at the wrist, as well as median–ulnar distal motor latency differences using the second lumbrical–first interosseous muscles recording technique (L2–IO1 test), were assessed to evaluate the severity of median nerve impairment in relation to the duration of computer use. Given that motor dysfunction generally indicates a more advanced stage of neuropathy, this study aimed to assess the impact of long-term computer use on the median nerve at the

wrist in computer users, presenting with symptoms suggestive of compressive neuropathies.

2. Materials and Methods

Participants

Data were prospectively collected from consecutive patients referred to the EDX laboratory with suspected CTS. Neurophysiological tests were performed by a neurologist specialized in clinical neurophysiology. EDX study was performed using a 3-channel Litebox electromyograph (Neurosoft). Data was collected consecutively from eligible patients who presented clinical suspicion of CTS between May 2024 and December 2025. The inclusion criteria comprised the presence of symptoms compatible with CTS and an occupation involving computer-based work for more than three years, with mouse and keyboard use exceeding four hours per day, five days per week. The exclusion criteria were pregnancy at the time of evaluation, a history of trauma to the wrist or elbow, and age under 18 years.

All examinations were carried out in a temperature-controlled environment, ensuring a skin temperature of 32°C, and the skin was degreased beforehand to minimize potential grounding artifacts. All subjects provided written informed consent for inclusion and participation in the study prior to enrollment. The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of the “Grigore T. Popa” University of Medicine and Pharmacy, Iasi.

Collected data were recorded in an Excel database and included the following variables: age, sex, hand dominance, number of years of computer use, comorbidities, profession, and electrophysiological parameters. Patient history assessment identified the number of years of computer use and known comorbidities at the time of examination. In addition, medical history was used to exclude other known neuromuscular conditions, such as prior cervical radiculopathy, brachial plexus neuropathy, or previous surgical intervention for CTS.

Data were anonymized prior to analysis. Patients who met predefined inclusion and exclusion criteria were enrolled. A total of 40 participants were included over an approximately 18-month period. The clinical diagnosis of CTS was established based on the presence of numbness and/or tingling in at least two of the following digits: thumb, index, middle, and the radial half of the ring finger, persisting for a prolonged period, with or without associated pain. Furthermore, hand numbness was not associated with cervical pain, and symptom onset in the hands were not preceded by similar symptoms in the lower limbs.

The computer user group included the following professions: IT programmers, teachers using computers in their daily activity, accountants, and secretarial staff. These individuals had no history of surgical interventions or traumatic injuries of the upper limbs, and no other musculoskeletal disorders. Most participants were right-hand dominant, with only one participant exhibiting left-hand dominance.

Neurophysiological test

For the neurophysiological study, single-use surface electrodes were used. For the median motor NCS, the recording electrode was placed over the muscle belly of the abductor pollicis brevis (APB) muscle, and stimulations were performed at the wrist and at the cubital fossa. Median nerve stimulation was applied between the tendon of the palmaris longus muscle and the tendon of the flexor carpi radialis muscle, at 7–8 cm proximal to the recording electrode. DML, CMAP amplitude, and median motor NCV were calculated.

For the ulnar motor NCS, recording electrode was placed over the muscle belly of the abductor digiti minimi (ADM) muscle, and stimulations were delivered at the wrist, 7–8 cm proximal to the recording electrode, as well as below and above the elbow.

Sensory responses were obtained using antidromic stimulation at the wrist, with recording from the third digit (for the median nerve) at a distance of 13–14 cm between the stimulation and recording sites, and from the fifth digit (for the ulnar nerve) at a distance of 10–12 cm. To calculate the median–ulnar motor latency difference, recording electrode was slightly lateral to the midpoint of the third metacarpal, over the first interosseous and second lumbrical muscles. Stimulation was performed at the wrist, 8–10 cm proximal to the recording electrode.

EDX diagnosis of CTS was established based on the recommendations and criteria of the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) [25]. The severity of CTS was classified electrophysiological according to the Padua classification [26]

3. Results

Statistical analysis

In the present research, data analysis was performed using the SPSS (Statistical Package for the Social Sciences, Chicago, Illinois version 27.0) program. Statistical analysis included descriptive and inferential methods adapted to the type of data. For continuous variables, mean $\pm$ standard deviation or median (IQR) were calculated. Normality of distribution was tested with Shapiro-Wilk, which determined the selection of parametric or nonparametric tests for comparisons.

Comparisons between independent groups were based on the t-test (for normal data) or Mann-Whitney U (for non-normal data), and for paired samples, the paired t-test or Wilcoxon test was used. Correlations were evaluated with the Pearson coefficient (for linear and normal relationships) or Spearman (for monotonic or non-normal relationships).

Binary logistic regression was used for the analysis of the prediction of CTS severity, with the calculation of OR (Odds Ratio) and 95% CI. Diagnostic performance was analyzed by ROC curves, calculating AUC (Area Under the Curve) and optimal thresholds. Multivariate analysis isolated the independent effect of occupational exposure after adjusting for age, gender, and comorbidities.

Results:

Due to the relatively small sample size, Padua grades were collapsed into two categories for statistical analysis: mild disease (Padua grades 1–2) and moderate-to-severe disease (Padua grades 3–4). No cases of extreme severity (Padua grade 5) were identified in the study cohort. Also, for statistical analysis, comorbidities were treated as binary variables (present/absent). The type of comorbidity (obesity, diabetes, rheumatic diseases, or thyroid conditions) was not considered, nor was their cumulative effect. Thus, patients with one or more comorbidities were classified in the same category (“present”), regardless of their number.

In our patient cohort, the mean age was 50,23 $\pm$ 11,85 years, with a median of 53 years. The Shapiro-Wilk test did not indicate a significant deviation from normality ($p=0.062$). Our results showed that women were significantly older than men (mean difference: 8.33 years; $t(38)=2.14$, $p=0.039$), a factor that may have clinical implications.

In this cohort, there is a positive and significant correlation between the measurement of median DML-APB and the motor median-ulnar comparative latency difference (L2-IO1 test). The positive coefficient indicates that, as median DML-APB increases, the motor latency difference between the median and ulnar nerves tends to increase. This result supports the convergent validity of the two electrophysiological methods in the diagnosis of CTS.

The association between years of professional activity and electrophysiological severity grade was assessed using Spearman’s rank correlation coefficient. These findings support a cumulative effect of long-term occupational exposure on nerve damage

severity. A significant positive Pearson correlation was observed in those cohort, between years of exposure and motor-APB latency in the dominant hand ($r=0.412$, $p=0.013$), indicating that longer occupational exposure is associated with greater impairment of median nerve. In contrast, this association did not reach statistical significance in the non-dominant hand ($r=0.218$, $p=0.244$).

Simple linear regression analysis identified years of occupational exposure as a significant predictor of median DML-APB latency in the dominant hand, explaining 17% of its variance, with an average increase of 0.67 ms for every additional 10 years of exposure ($B = 0.067$).

Participants were stratified into two groups according to exposure duration: <20 years and ≥ 20 years. Clinical and electrophysiological parameters were compared between groups to assess the impact of long-term exposure. Subjects with ≥ 20 years of exposure showed significantly prolonged median DML-APB (4.44 ms) compared to those with <20 years (3.56 ms). Comparison between exposure groups revealed a statistically significant difference in median-DLM-APB latency between subjects with <20 and ≥ 20 years of exposure ($p = 0.034$).

Spearman correlation analysis revealed a significant positive association between years of exposure and the median-ulnar latency difference, indicating progressive median nerve compression with increasing occupational exposure. For the dominant hand, there is a moderately strong and statistically significant positive correlation between the number of years of occupational exposure and the median-ulnar motor difference latency ($\rho=0.501$, $p=0.007$). For the non-dominant hand, there is a tendency for a moderate positive correlation ($\rho=0.382$), but this is not statistically significant at the 0.05 threshold ($p=0.082$). We can say that there is a tendency towards association, but not a clear relationship (Table 1).

Table 1: Spearman correlation between years of exposure and the median latency difference

Analysed association	N pairs	Spearman's correlation coefficient (ρ)	p-value	Correlation strength
Exposure years vs. latency difference (L2-IO1, dominant hand)	27	0.501	0.007*	Moderate-to-strong positive
Exposure years vs. latency difference (L2-IO1, non-dominant hand)	22	0.382	0.082	Moderate, non-significant

Using the Mann-Whitney U test, the median-ulnar motor latency difference differed significantly between exposure groups (<20 vs. ≥ 20 years), with higher values observed in the ≥ 20 -year group ($p=0.014$).

Linear regression analysis for the dominant hand demonstrates that each 10-year increase in exposure was associated with an increase of 0.52 ms in the median-ulnar motor latency difference. The adjusted R^2 indicated that 22% of the variability in the median-ulnar motor latency difference (L2-IO1) in the dominant hand was explained by years of exposure.

A subset of patients within our cohort also presented with concomitant cubital tunnel syndrome (CuTs). Our findings indicate that the mean ulnar nerve conduction velocity across the elbow was preserved within normal ranges (>50 m/s), with only 12–18% of cases showing subnormal values. A statistically significant negative correlation was observed between years of occupational exposure and ulnar NCV at the elbow.

A clear upward trend was observed in the distribution of CTS severity, with increasing exposure duration being associated with a higher proportion of moderate-to-severe cases (Table 2).

Table 2: Distribution of CTS severity according to exposure groups (5-year categories)

Years of exposure (categories)	Total N	Mild/Absent CTS (Grade 0–1)	Moderate–Severe CTS (Grade 2)	% Sever
<10 years	5	4 (80.0%)	1 (20.0%)	20.0%
10–14 years	9	6 (66.7%)	3 (33.3%)	33.3%
15–19 years	6	3 (50.0%)	3 (50.0%)	50.0%
20–24 years	7	2 (28.6%)	5 (71.4%)	71.4%
25–29 years	7	1 (14.3%)	6 (85.7%)	85.7%
≥30 years	6	0 (0.0%)	6 (100.0%)	100.0%

In the logistic regression analysis, duration of exposure was significantly associated with the occurrence of the outcome. For each 5-year increase in exposure, the odds of the outcome increased more than twofold (OR = 2.42, 95% CI: 1.51–3.88; $p < 0.001$). The model demonstrated good explanatory power, with a Nagelkerke R^2 of 0.402. Model calibration was adequate, as indicated by a non-significant Hosmer–Lemeshow goodness-of-fit test ($\chi^2 = 4.32$, $p = 0.827$) (Table 3). This corresponds to an approximately 2.4-fold increase in the odds of moderate-to-severe CTS for each additional 5 years of exposure within our cohort.

Table 3: Logistic regression analysis – Model 1 (Exposure duration only)
Dependent variable: Moderate-to-severe CTS (1) vs. mild/early CTS (0)

Predictor	B	SE	OR (Odds Ratio)	IC 95% pentru OR	p
Intercept	-2.944	0.882	0.053	-	0.001*
Exposure duration (years)	0.884	0.240	2.421	[1.512, 3.875]	<0.001*

To assess potential overlap among predictors included in the model, we examined correlations and multicollinearity. Correlation analysis revealed statistically significant, moderate associations between exposure duration and age ($r = 0.412$), between age and sex ($r = 0.327$), and between age and comorbidity status ($r = 0.379$) (all $p < 0.05$) (Table 4). In addition, VIF values were < 2.5 for all predictors, suggesting the absence of problematic multicollinearity (commonly used thresholds are $VIF > 5$ – 10) (Table 5).

Table 4: Correlation analysis between predictors

	Exposure duration (years)	Age	Sex	Comorbidities
Exposure duration (years)	1.000	0.412*	0.196	0.288
Age	0.412*	1.000	0.327*	0.379*
Sex	0.196	0.327*	1.000	0.157
Comorbidities	0.288	0.379*	0.157	1.000

Table 5: VIF (Variance Inflation Factor) analysis for multicollinearity (no evidence of problematic multicollinearity)

Predictor	VIF	Tolerance (1/VIF)
Exposure duration (years)	1.28	0.781
Age	1.47	0.680
Sex	1.19	0.841
Comorbidities	1.27	0.787

Moreover, compared with the unadjusted model, adjustment for age and comorbidity status (yes/no) attenuated the exposure OR from 2.42 to 2.08 ($\approx 14\%$), indicating partial confounding by these factors. However, the association remained > 1 and statistically significant (Table 6).

Table 6: Comparison between the unadjusted and adjusted models

Model	Predictor	OR (Unadjusted)	OR (Adjusted model)	Adjustment
Unadjusted model	Exposure duration (5 years)	2.421	-	-
Adjusted model	Exposure duration (5 years)	-	2.084	-13.9%
	Age (5 years)	-	1.244	-
	Sex (F)	-	2.321	-
	Comorbidities (Yes)	-	2.500	-

The predicted probability of moderate-to-severe CTS increases progressively with the duration of exposure. At 5 years of exposure, the estimated probability is 12.3%, increasing to 25.7% at 10 years and 44.8% at 15 years. The increase is not linear but increases with the extension of exposure. Between 5 and 10 years, the probability increases by 13.4 %, while between 10 and 15 years the increase is higher, by 19.1%, suggesting a cumulative effect of exposure on the risk of severe CTS.

ROC analysis identified an optimal threshold of >17.5 years of exposure for discriminating patients with severe CTS. At this threshold, sensitivity was 82.6% and specificity was 75.0%, indicating good ability to identify severe cases, with an acceptable level of false-positive results. The area under the ROC curve (AUC = 0.834) suggests a good ability to discriminate the duration of exposure in predicting severe CTS, confirming the usefulness of this variable as a screening tool. For the latency difference, the optimal threshold identified by ROC analysis was >0.8 ms, with a sensitivity of 91.3% and a specificity of 56.3%. The area under the ROC curve (AUC = 0.798) indicates a good ability to discriminate for severe CTS (Table7).

Table 7: Optimal screening thresholds (ROC analysis)

Parameter	Optimal cut-off	Sensitivity	Specificity	AUC
Years of exposure	>17.5 ani	82.6%	75.0%	0.834
Median DML APB	>4.1 ms	78.3%	81.3%	0.816
Median-ulnar latency difference (L1-IO2)	>0.8 ms	91.3%	56.3%	0.798

Indicative thresholds for clinical screening were proposed based on the changes observed in this cohort (Table 8). For a highly sensitive screening approach, a cutoff of more than 15 years of occupational exposure yielded a sensitivity of 91.7% and a specificity of 56.3%. Increasing the exposure threshold to more than 20 years improved specificity to 87.5%, with a corresponding sensitivity of 66.7%. An intermediate cutoff of more than 17.5 years provided a balanced performance, with a sensitivity of 79.2% and a specificity of 75.0%. For electrophysiological assessment, a DML greater than 4.1 ms combined with an median-ulnar latency difference greater than 0.85 ms demonstrated a sensitivity of 91.3% and a specificity of 75.0%

Table 8. Comparative Analysis of Optimal Screening Thresholds

Screening objective	Recommended cutoff	Sensitivity	Specificity	Clinical application
Sensitivity-oriented screening (minimizing missed cases)	>15 years of exposure	91.7%	56.3%	Population-based screening
Specificity-oriented screening (minimizing false-positive results)	>20 years of exposure	66.7%	87.5%	Diagnostic confirmation

Optimal balance (for clinical practice)	>17.5 years of exposure	79.2%	75.0%	Alternative acceptable
For electrophysiological evaluation	median DML-APB >4.1 ms median–ulnar latency difference (L1–I02) >0.85 ms	91.3%	75.0%	Indication for EDX

4. Discussion

Numerous studies have suggested that workers who perform tasks requiring both high force and high repetition have an even higher rate of developing CTS [27]. Prolonged computer use may be associated with upper limb symptoms and electrophysiological nerve abnormalities [23,28]. Previous studies have reported reduced sensory NCV of the median nerve in asymptomatic computer users compared with controls, suggesting the presence of subclinical CTS [23]. Individuals with abnormalities of median NCS but without clinical symptoms appear to have a slightly increased risk of developing symptoms over the following 2–6 years [29]. Reduced NCV of the median motor and sensory nerves, as well as the ulnar motor and sensory nerves, have also been reported in other studies evaluating computer operators [28,30]. In contrast, Sanden et al. found no statistically significant differences in median NCV [31]. The present study aimed to assess the severity of CTS by focusing primarily on motor parameters of the median nerve, which are known to be affected after sensory parameters [29].

In our study, most participants were women, in line with previous literature reporting a higher prevalence of CTS among females [32]. Notably, women were significantly older than men, a difference that may be clinically relevant given the well-established association between increasing age and compressive neuropathies, particularly CTS. Similar results have also been identified in previous studies [33].

Evidence suggests that prolonged computer use increases the risk of repetitive strain injuries, with pain, tingling, numbness, and weakness related to neural involvement of the mouse-operating limb [28]. The finding confined to the dominant hand reinforces the role of repetitive biomechanical load, pointing toward a cumulative occupational impact on the median nerve, predominantly affecting the limb exposed to greater mechanical demand. In other studies, median nerve lesion severity appears to be more strongly related to lifetime hand activity than to computer-based occupational exposure [33,34].

Subjects with 20 years or more of occupational exposure demonstrated significantly prolonged median DML-APB compared to those with less than 20 years of exposure. This observation supports the hypothesis of a cumulative occupational effect on median nerve function, particularly among individuals with long-term exposure. Moreover, the concordant results obtained using both median DML-APB and the median–ulnar latency difference further strengthen the evidence for a cumulative effect.

Also, when assessing CTS in computer users, clinicians should routinely investigate symptoms indicative of concomitant ulnar neuropathy at the elbow. Previous studies have reported electrophysiological changes of the ulnar nerve in computer users, mainly reflected by reduced sensory conduction velocities [23].

The adjusted R^2 of 0.22 suggests that years of exposure account for a meaningful, although not exclusive, proportion of the variability in motor latency differences, highlighting the multifactorial nature of nerve conduction impairment. These findings suggest a real, although not exclusive, cumulative effect of exposure on the median–ulnar motor difference in the dominant hand. The observed association suggests that longer exposure duration may be associated with greater latency differences.

ROC analysis identified an optimal cutoff of >17.5 years of exposure for distinguishing patients with moderate-to-severe CTS. Notably, an accelerated increase in CTS severity was already observed after 10 years of exposure, indicating that this duration may serve as a clinically relevant threshold for initiating comprehensive evaluation in symptomatic computer users. However, when evaluating CTS, it should be borne in mind that

its multifactorial nature may extend beyond repetitive hand movements and ergonomic challenges, also involving factors such as obesity, diabetes, genetic predisposition, and rheumatic diseases [10,35].

Comparative analysis of screening thresholds shows that diagnostic performance depends on the clinical objective. Lower exposure cutoffs increase sensitivity with moderate specificity, favoring case detection, whereas higher thresholds improve specificity at the cost of sensitivity. An exposure duration >15 years provides high sensitivity and is suitable for initial screening, while >20 years offers higher specificity, supporting diagnostic confirmation. The intermediate cutoff of >17.5 years achieves the best balance and is appropriate for routine clinical use. These findings indicate a graded association between cumulative occupational exposure and the risk of moderate–severe CTS. Electrophysiological thresholds also showed high diagnostic performance, and their integration with exposure-based criteria may improve risk stratification and optimize confirmatory testing.

Within our clinically referred cohort, longer exposure duration was associated with higher odds of moderate-to-severe CTS (unadjusted OR 2.42 per 5-year increase). Although the association remained statistically significant after adjustment for age, sex, and comorbidity status (adjusted OR 2.08), the attenuation suggests partial confounding, and residual confounding by unmeasured metabolic factors (e.g., BMI) cannot be excluded. Accordingly, these findings should be interpreted as exploratory and require prospective external validation.

In conclusion, we observed a graded association between cumulative occupational exposure and the severity of electrophysiological impairment. These exploratory results are consistent with a cumulative effect but require confirmation in longitudinal studies. Years of exposure represented the most robust overall predictor, while the motor median–ulnar latency difference (L2–IO1) demonstrated superior sensitivity for the early identification of nerve impairment. These findings support an integrated approach combining exposure history with electrophysiological parameters to optimize screening and prevention strategies.

With respect to the electrophysiological findings involving the ulnar nerve, the observed negative correlation between years of exposure and ulnar nerve conduction velocity should be interpreted as exploratory, as no adjusted multivariable model was performed for this outcome and potential confounding factors cannot be excluded. The observed association between occupational exposure and the electrophysiological parameters of the ulnar nerve suggests that prolonged computer use may contribute not only to median nerve involvement but also to a broader susceptibility to upper limb compression neuropathies. Electrophysiological changes involving both peripheral nerves in computer users have also been reported in previous studies [23,28]. Functional alterations of the ulnar nerve observed in computer users may be attributable to several biomechanical mechanisms. Repetitive hand movements associated with keyboard and mouse use may contribute to muscle imbalance and increased mechanical stress on peripheral nerves, thereby facilitating the development of compression neuropathies. In addition, prolonged elbow support during computer work may lead to compression of the ulnar nerve at the olecranon level, increasing the risk of ulnar neuropathy. Furthermore, sustained elbow flexion, prolonged forearm support on hard surfaces, and repetitive postural loading during computer use may increase mechanical stress within the cubital tunnel, potentially contributing to impaired nerve conduction [36]. Depending on the magnitude and duration of neural loading associated with prolonged computer use, increased local mechanical pressure may contribute to axonal injury and myelin sheath disruption. This process may increase the distance between the nodes of Ranvier and impair impulse transmission, ultimately leading to reduced nerve conduction velocity [28]. Given the exploratory nature of this observation, longitudinal studies are required to clarify the underlying mechanisms.

CTS is a condition of major relevance in occupational health, with significant economic, social, and medical implications, highlighting the critical importance of preventive measures. The burden on patients, employers, and healthcare systems underscores the need for integrated strategies focused primarily on prevention, alongside early diagnosis and effective management of CTS [37]. Moreover, recognition of the contribution of genetic factors (such as COL5A1 polymorphisms and the GSTM1 null genotype) and immunological mechanisms in CTS, may enable more refined risk stratification among individuals engaged in occupations requiring intensive hand use, thereby identifying those who may derive the greatest benefit from targeted ergonomic interventions, task rotation, or proactive workplace modifications [11].

Limitations:

The relatively small sample size represents a limitation of this study, as it may have contributed to an overestimation of the observed effect sizes. Accordingly, the observed associations should be interpreted with caution, being exploratory in nature, and warrant confirmation in larger, preferably multicentre studies.

Another limitation of the analysis is that comorbidities were coded only as a binary variable (yes/no), without accounting for their type or the potential cumulative effect of multiple concurrent comorbidities. Therefore, residual confounding cannot be excluded. Exposure was assessed solely in terms of duration, without accounting for intensity, frequency, or ergonomic factors, which may have influenced nerve conduction outcomes.

Certain limitations regarding the obtained screening thresholds should be acknowledged. The proposed thresholds were derived from a single patient cohort and were not adjusted for the type or cumulative burden of comorbidities, factors that may influence both symptom severity and electrophysiological findings. Therefore, external validation in independent populations is required before broad clinical implementation.

An equally important limitation is that this cohort included patients referred with a clinical suspicion of CTS, representing an already selected population. Therefore, the proposed thresholds should not be directly extrapolated to screening in the general population of computer users, and external validation is needed in occupational cohorts, including both symptomatic and asymptomatic individuals at the time of testing.

Given the absence of a separate control group, the applicability of the proposed thresholds' performance to the wider occupational population of workplace computer users remains limited.

5. Conclusions

Despite these limitations, the study provides valuable insights into the complementary role of exposure history and electrophysiological parameters in early nerve impairment screening. In this clinically selected cohort of participants referred with suspected CTS, occupational exposure duration was independently associated with CTS severity on multivariable analysis. Each 5-year increase in exposure was associated with a two-fold increase in the likelihood of moderate-to-severe CTS. The model showed good calibration and very good discriminative ability, suggesting a clinically relevant effect, although external validation in larger cohorts is required.

Although this study does not evaluate a screening test per se, the observed associations between occupational exposure duration, age, and electrophysiological severity support the concept of risk-based screening strategies for compressive neuropathies. Regular longitudinal monitoring of computer-based workers supports early diagnosis and timely treatment, reducing the risk of advanced disease stages associated with higher costs and incomplete functional recovery.

In the absence of a control group, our analyses rely on internal comparisons and do not support definitive conclusions regarding screening utility at the population level. The association between duration of occupational exposure and CTS severity should be interpreted as hypothesis-generating and requires confirmation in prospective studies with external validation, including individuals who are exposed but asymptomatic.

In conclusion, our findings suggest that, among symptomatic participants in the studied cohort, a longer history of computer work (in years) is associated with more severe CTS, and external validation is warranted. Further studies are required to validate and expand upon these results.

Author Contributions: Conceptualization, G.A.V. and D.I.C.; methodology G.A.V., D.I.C. and C.-M.P.; validation, G.A.V., D.I.C. and L.B.; formal analysis C.E.B.; investigation L.B. and G.A.V.; resources, C.E.B. and C.-M.P.; writing—G.A.V.; writing—review and editing, G.-A.V.; visualization D.I.C.; supervision, D.I.C.; project administration G.A.V. All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of University of Medicine and Pharmacy “Grigore T. Popa” No. 441/12.05.2024.

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data are not publicly available due to ethical and privacy restrictions.

Conflicts of Interest: The authors declare no conflict of interest.

Appendix A: Supplementary data: The difference in paired sample size reported in Table 1 was due to incomplete non-dominant hand recordings: in five participants, a reliable median–ulnar latency comparison could not be obtained because the data were not captured by the software or the examination was terminated early due to discomfort; these cases were excluded from the paired analysis.

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