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PRACA ORYGINALNA
ORIGINAL PAPER

Which factors affect electrophysiological parameters in patients undergoing surgery for carpal tunnel syndrome?

Jakie czynniki wpływają na elektrofizjologiczne parametry przewodzenia u pacjentów poddanych operacyjnemu leczeniu zespołu cieśni nadgarstka?

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ABSTRACT

INTRODUCTION: Carpal tunnel syndrome (CTS) is an upper limb neuropathy that occurs as a result of compression of the median nerve in the carpal tunnel and is the most common mononeuropathy in the general population. The aim of the study was to assess the electrophysiological parameters of the median nerve before and 6 months after surgical treatment of CTS in patients with a history of smoking and comorbidities.
MATERIAL AND METHODS: 84 patients with CTS who were eligible for surgery were enrolled in this prospective study. Electrophysiological tests were performed in the patients before and 6 months after surgery for CTS.
RESULTS: The results of the study prove that smoking and diabetes significantly worsen the electrophysiological parameters in patients undergoing surgical treatment of CTS.
CONCLUSIONS: Smoking and diabetes cause a significantly worse prognosis in patients after surgery for CTS.

KEYWORDS

carpal tunnel syndrome, diabetes, smoking

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STRESZCZENIE

WSTĘP: Zespół cieśni nadgarstka (ZCN) jest neuropatią kończyny górnej, pojawiającą się w wyniku ucisku nerwu pośrodkowego w kanale nadgarstka. Celem badania była ocena parametrów elektrofizjologicznych nerwu pośrodkowego przed leczeniem i 6 miesięcy po leczeniu operacyjnym ZCN u pacjentów obciążonych nikotynizmem oraz chorobami współistniejącymi.
MATERIAŁ I METODY: Do prospektywnego badania włączono 84 pacjentów z rozpoznanym klinicznie i elektrofizjologicznie ZCN kwalifikowanych do leczenia operacyjnego. U każdego pacjenta przed leczeniem oraz 6 miesięcy po leczeniu operacyjnym ZCN przeprowadzono badanie elektrofizjologiczne.
WYNIKI: Uzyskane wyniki dowodzą, że nikotynizm i cukrzyca istotnie pogarszają parametry elektrofizjologiczne u pacjentów poddanych leczeniu operacyjnemu ZCN.
WNIOSKI: Nikotynizm i cukrzyca są niekorzystnymi rokowniczo czynnikami u osób operowanych z powodu ZCN.

SŁOWA KLUCZOWE

zespół cieśni nadgarstka, cukrzyca, palenie

INTRODUCTION

Carpal tunnel syndrome (CTS), also known as tardy median palsy, is an upper limb neuropathy that results from the compression of the median nerve in the carpal tunnel. The pressure on the median nerve is caused by chronically increased pressure in the carpal tunnel (> 30 mm Hg) due to external and internal mechanical factors. As a result, the size of the carpal tunnel is reduced or the volume of the anatomical structures in the tunnel is increased. As a consequence of the elevated pressure in the carpal tunnel, impaired blood flow is reported in the capillaries that nourish the nerve, and hence ischemic lesions occur in the nerve. Over time, morphological changes (i.e. demyelination and/or axonal degeneration) occur in the nerve structure [1,2]. Currently, CTS is the most common mononeuropathy in the general population. It affects about 3% of the population, mostly women between 40 and 60 years of age. The peak incidence occurs in women over 55 years of age [3,4]. The prevalence significantly increases in the case of occupations that involve repeated palmar flexion and dorsiflexion of the wrist and reaches even 10−30% [5].

The diagnosis of CTS is established based on the history, clinical examination, and median nerve conduction studies [1]. During the physical examination, sensation and muscle function in the median nerve innervation area are assessed. In addition, the examination should include assessment of the strength of selected muscles of the thenar eminence, i.e. opponens pollicis and the abductor pollicis brevis using the Lovett scale and hand grip strength using a dynamometer. Provocation tests are also utilised in the diagnosis of CTS. However, they are of limited diagnostic use owing to the lack of uniform rules for conducting these tests and the subjective assessment and interpretation of the results [6,7]. Ultrasound (US) and electromyography (EMG) are standard diagnostic procedures [8]. Ultrasound is crucial in the diagnosis of CTS because of the availability, non-invasiveness, and short time of the examination. A 3-stage CTS severity scale was developed based on the cross-sectional area (CSA) of the median nerve measured at the entrance to the carpal tunnel (at the level of the pisiform bone) and at the exit of the carpal tunnel (at the level of the hook of the hamate) [9]. Moreover, US allows the visualization of edema of the median nerve in the early period of the disease, which is the cause of subjective complaints, although this has not been yet confirmed by electrophysiological studies [8].

In motor nerve conduction studies, the response from the muscle innervated by the nerve is recorded. The nerve motor point is the place of stimulation, and the evoked potential is recorded in the muscle motor point mostly using surface electrodes. Surface electrodes are placed on finger flexor muscles to assess sensory fibers. In turn, the recording of potentials is possible due to electrodes placed above the nerve. The examination is characterized by high sensitivity and specificity [10]. It allows the extent of nerve damage to be determined and assessment of whether the conduction impairment is associated with axonal injury or a pathological process in the myelin sheath [11].

Electromyography is considered the gold standard in the diagnosis of CTS. Of note, establishing the diagnosis without performing the examination may result in misdiagnosis and inadequate treatment [10,12].

In the early stages of CTS, patients report numbness of the fingers, e.g. when using the telephone [13]. This symptom may be the result of transient ischemia of the median nerve. As the disease progresses, the volume of the carpal tunnel may decrease and result in median nerve fibrosis. The symptoms are usually more severe at night. Some patients may complain of cramps, fatigue, or pain in the forearm or arm [14], although patients with CTS do not always report pain. Nevertheless, it is an important symptom and should not be confused with neuropathic sensory disorders [15]. In a multicenter study on 1123 patients with CTS, pain was reported in 52% of the subjects [16].

The treatment of CTS can be either conservative or surgical. Various methods of non-surgical treatment are available. However, patients should be always

instructed by physicians [17]. Changing habits (e.g. the limitation of wrist movements) and following the principles of ergonomic work can be useful in reducing pressure on the median nerve. Nonetheless, there is little evidence that confirms the success of this approach [18]. Non-surgical treatment is comprised of laser therapy, pharmacotherapy, massage, physical exercise, and ultrasound-based methods. Pharmacological treatment usually includes topical steroid injections [19]. However, they mostly provide only temporary relief. Surgical treatment, which consists in releasing the carpal tunnel content by transection of the transverse carpal ligament, is considered the most effective treatment method of CTS [20]. Studies have demonstrated that conservative treatment is less effective in reducing the symptoms of the syndrome when used for advanced, moderate or severe cases [20,21].

The aim of the study was to ascertain which factors may affect the electrophysiological parameters in different groups of patients undergoing surgery for CTS. It was done by assessing the electrophysiological parameters of the median nerve before and 6 months after surgical treatment of CTS in patients with a history of smoking and comorbidities (i.e. diabetes, renal failure, rheumatoid arthritis and gout).

The analysis of the results in specific patient groups, the variable effectiveness of the surgical procedure depending on comorbidities allow better prediction of the effects of treatment in an individual way for individual patients.

MATERIAL AND METHODS

Eighty-four patients (70 women and 14 men) with clinically and electrophysiologically diagnosed CTS, who were eligible for surgery, were enrolled in this prospective study. The subjects were divided into groups according to sex, age, dominant hand, hand affected by CTS, disease duration, smoking, and comorbidities. Electrophysiological tests were performed using the Nicolet VikingQuest in the patients before and 6 months after surgery for CTS. The analysis included the conduction velocity, the amplitude of the sensory response, the terminal latency, and the amplitude of the motor response of the median nerve. The results were statistically analyzed.

Considering the fact that the conduction velocity, the amplitude of the sensory response, the amplitude of motor response, the distal sensory latency, and the distal motor latency were linearly dependent on age, regression against this variable was performed. Mann-Whitney U tests were employed to compare the medians of the variables due to the lack of normality of the distribution of variables (Shapiro-Wilk test, $\alpha = 0.05$).

RESULTS

The mean age of the patients was 58.7 years. The subjects had a history of smoking (17%), diabetes (9%), renal failure (2%), rheumatoid arthritis (4%), and gout (4%; Table I).

Table I. Patient characteristics
Tabela I. Charakterystyka pacjentów

Patient characteristics		
Mean age	(years)	58.7 ± 9.8
Sex	(W : M)	70 : 14
Dominant hand	(R : L)	81 : 3
Operated hand	(R : L)	52 : 32
Smokers	(%)	17
Diabetes	(%)	9
Gout	(%)	4
Rheumatoid arthritis	(%)	4
Renal insufficiency	(%)	2

When analyzing the electrophysiological effects of treatment for the entire group of patients, as well as by sex, an improvement in the conduction parameters was observed, which confirms the commonly known and repeatedly proven fact of the effectiveness of surgical treatment (Table II).

In the case of terminal latency, both the pre- and post--procedure latency medians were significantly higher in men than in women, as shown in Table II. This may result from the age-inhomogeneous groups. It is worth noting, however, that in the case of age-normalized latency 2, men had significantly better prognosis for the recovery of normal conduction than women.

When the group of smokers was compared with the group of non-smokers, a slight decrease in the amplitude of the sensory response was found before surgery (mean amplitude: 3.6 uV and 4.7 uV, respectively; Table III) and in the postsurgical follow-up examination (median: 4.0 ± 5.09 uV and 5.0 ± 12.3 uV, respectively; Table III), which could suggest a tendency for the occurrence of axonal injury to the nerves in smokers.

The analysis of the terminal latency of the motor response before surgery demonstrated a slight increase in the latency in the group of smokers compared to non-smokers (median: 6.2 ± 2.46 ms and 5.5 ± 2.51 ms, respectively; Table IV). Nevertheless, the mean latency recorded during the postsurgical follow-up examination was similar in both groups (5.49 ms and 5.45 ms, respectively; Table IV), which suggested a similar prognosis for the groups.

Table II. Comparison of latencies (1 and 2) of motor response in women (n = 70) and men (n = 14) using Mann-Whitney U test
Tabela II. Porównanie latencji (1 i 2) dla włókien ruchowych u kobiet (n = 70) i mężczyzn (n = 14) za pomocą testu U Manna i Whitneya

Parameter	Mean – women	Mean – men	Median – women	Median – men	Standard deviation – women	Standard deviation – men	P
Latency 1	6.086	6.992	5.3	6.55	2.46	2.604	0.088
Latency 2	5.264	6.817	4.5	5.95	2.12	2.766	0.005
Absolute difference (Lat 2 – Lat 1)	-0.822	-0.175	-0.8	-0.25	2.295	1.179	0.21
Latency 1 standardized for age	-0.011	0.068	-0.579	-0.415	2.213	2.96	0.903
Latency 2 standardized for age	-0.134	0.804	-0.798	0.028	2.021	2.878	0.083

Table III. Comparison of sensory amplitudes (1 and 2) in smokers (n = 15) and non-smokers (n = 69) using Mann-Whitney U test
Tabela III. Porównanie amplitud czuciowych (1 i 2) u osób palących (n = 15) i niepalących (n = 69) za pomocą testu U Manna i Whitneya

Parameter	Mean – non-smokers	Mean – smokers	Median – non-smokers	Median – smokers	Standard deviation – non-smokers	Standard deviation – smokers	P
Sensory amplitude 1	4.677	3.667	3	3	11.45	2.16	0.761
Sensory amplitude 2	6.585	5.933	5	4	12.36	5.092	0.649
Absolute difference (Lat 2 – Lat 1)	1.909	2.267	1	2	10.42	4.334	0.632
Sensory amplitude 1 standardized for age	0.002	-1.489	-0.852	-1.202	10.09	2.31	0.288
Sensory amplitude 2 standardized for age	0.002	-1.646	-0.489	-0.258	10.46	5.168	0.265

Table IV. Comparison of latencies (1 and 2) of motor response in smokers (n = 15) and non-smokers (n = 69) using Mann-Whitney U test
Tabela IV. Porównanie latencji (1 i 2) dla włókien ruchowych u osób palących (n = 15) i niepalących (n = 69) za pomocą testu U Manna i Whitneya

Parameter	Mean – non-smokers	Mean – smokers	Median – non-smokers	Median – smokers	Standard deviation – non-smokers	Standard deviation – smokers	P
Latency 1	6.20	6.31	5.5	6.2	2.51	2.46	0.670
Latency 2	5.49	5.45	4.8	4.3	2.16	2.80	0.520
Absolute difference (Lat 2 – Lat 1)	-0.70	-0.86	-0.7	-1	2.36	1.07	0.521
Latency 1 standardized for age	0.001	0.437	-0.468	-0.314	2.270	2.587	0.513
Latency 2 standardized for age	0.003	0.203	-0.642	-0.741	2.003	2.908	0.748

The analysis of the median of the terminal latency revealed significantly more severe CTS in diabetic patients compared non-diabetic subjects (median: 7.55 ± 3.7 ms and 5.5 ± 2.1 ms, respectively; p = 0.024). Additionally, after surgery the terminal latency was significantly increased in the diabetic patients (median: 6.65 ± 1.2 ms and 4.5 ± 2.3 ms, respectively; p = 0.001), which showed a worse prognosis in diabetic patients (Table V).

The same analysis was performed in the case of conduction velocity in sensory fibers. Significantly more severe CTS was also found in the diabetic patients compared to the non-diabetic subjects (median: 24 ± 13.1 ms and 28.5 ± 10.5 ms, respectively; p = 0.189). Of note, a significant discrepancy between the values was found postoperatively (median: 24.5 ± 5.8 ms and 35 ± 13.3 ms, respectively; p = 0.016), which revealed practically no postoperative improvement in terms of conduction velocity in the diabetic patients (Table VI).

Table V. Comparison of latencies (1 and 2) in diabetic (n = 8) and non-diabetic (n = 76) patients using Mann-Whitney U test
Tabela V. Porównanie latencji (1 i 2) u pacjentów z cukrzycą (n = 8) i bez cukrzycy (n = 76) za pomocą testu U Manna i Whitneya

Parameter	Mean – patients without diabetes	Mean – patients with diabetes	Median – patients without diabetes	Median – patients with diabetes	Standard deviation – patients without diabetes	Standard deviation – patients with diabetes	P
Latency 1	5.964	8.08	5.5	7.55	2.188	3.708	0.024
Latency 2	5.276	7.04	4.5	6.65	2.304	1.205	0.000
Absolute difference (Lat 2 – Lat 1)	-0.688	-1.04	-0.8	0.4	1.966	3.503	0.38
Latency 1 standardized for age	0.003	1.723	-0.389	0.91	2.072	3.471	0.076
Latency 2 standardized for age	0.002	1.423	-0.68	0.947	2.222	1.167	0.000

Table VI. Comparison of conduction velocities (1 and 2) in sensory fibers in diabetic (n = 8) and non-diabetic (n = 76) patients using Mann-Whitney U test
Tabela VI. Porównanie szybkości przewodzenia (1 i 2) we włóknach czuciowych u pacjentów z cukrzycą (n = 8) i bez cukrzycy (n = 76) za pomocą testu U Manna i Whitneya

Parameter	Mean – patients without diabetes	Mean – patients with diabetes	Median – patients without diabetes	Median – patients with diabetes	Standard deviation – patients without diabetes	Standard deviation – patients with diabetes	P
Velocity 1	27	22.3	28.5	24	10.567	13.141	0.189
Velocity 2	31.338	25.8	35	24.5	13.359	5.846	0.016
Absolute difference (Lat 2 – Lat 1)	4.338	3.5	4.5	0.5	10.374	12.528	0.427
Velocity 1 standardized for age	0.002	-2.254	0.987	1.281	9.631	11.761	0.879
Velocity 2 standardized for age	-0.001	-2.027	1.259	-4.45	11.808	7.836	0.161

No significant correlations were found between the electrophysiological parameters and other comorbidities in the study group, which may be related to the small size of the sample.
Also, the co-occurrence of the analyzed factors could not be considered because of the small size of the analyzed patient groups

DISCUSSION

Diabetes mellitus, menopause, hypothyroidism, obesity, pregnancy in addition to forceful and repetitive hand movements in some professions are probable risk factors for the development of CTS [22,23,24,25,26]. Additionally, other factors that may influence the development of neuropathy include genetics, systemic, autoimmune and inflammatory diseases. They should always be considered when obtaining a history from patients with symptoms of neuropathy [27].
Type 1 and type 2 diabetes mellitus are established risk factors for CTS [24]. Similarly, overweight and obesity, which often co-exist with diabetes, also increase the risk of CTS two-fold [25]. Smoking is also a suspected factor for the development of CTS. The relationship between smoking and CTS has been reported in the literature [28].
Smoking is an established factor for the development of cardiovascular disease, particularly in diabetic patients. Epidemiological studies found that the risk of cardiovascular disease in diabetic patients who were smokers was five-fold higher compared to non-smokers without diabetes [29]. The relationship between smoking and the risk of diabetic neuropathy was confirmed by the European Diabetes Prospective Complications Study, which assessed the severity of neuropathy at the beginning of the study and after a 7-year follow-up. It was found that neuropathy was significantly more prevalent in smokers [30]. In the 1990s, Mitchell et al. [31] demonstrated a significantly higher prevalence of neuropathy in smokers compared to non-smokers (64% vs 42%, respectively) in their retrospective study of over 300 patients with type 1 and type 2 diabetes.
Our study revealed lower mean amplitudes of the sensory response before surgery and during the postoperative follow-up examination. It may be related to axonal injury to the nerves, probably resulting from microangiopathy, which may have been caused by smoking. Studies have found that smoking significantly increased complications resulting from microangiopathy, e.g. in diabetic patients [32]. Therefore, smokers are much more exposed to the occurrence of symptoms of peripheral neuropathy, retinopathy, and

nephropathy since smoking is a highly unfavorable factor, decreasing the quality of life in long-term follow-ups.

Increased terminal latency, which is one of the basic electrophysiological parameters for the diagnosis and severity of CTS, was more evident in smokers in the study group, which indicated slightly more severe CTS at the time of surgery. However, the mean values of terminal latency after surgery were similar in both groups, which allowed the conclusion to be drawn that smoking patients had as good a prognosis as non--smoking subjects in terms of improvement in the electrophysiological parameters. Nonetheless, this conclusion was based only on the analysis of terminal latency, and therefore might not fully reflect the situation. The previously mentioned axonal injury to the sensory fibers of the median nerves should also be considered since it was more severe after surgery in smokers compared to non-smoking subjects. Thus, it is expected that the subjective improvement in the group of smokers will be certainly lower (Figure 1).

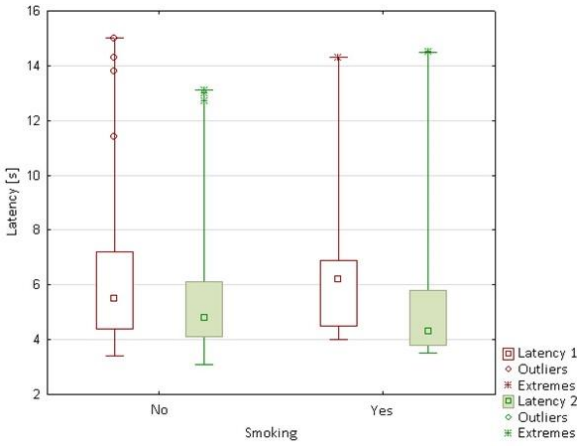

Fig. 1. Comparison of latencies of motor response in smokers and non--smokers before and after reconstructive surgery.
Ryc. 1. Porównanie latencji końcowej odpowiedzi ruchowej u palących i niepalących przed zabiegiem naprawczym i po zabiegu.

In the group of diabetic patients, significantly more severe CTS was found at the time of surgery. The question why diabetic patients are diagnosed later is still open to debate. One of the explanations could be that the accompanying symptoms of diabetic neuropathy delay the diagnosis. It is also suspected that CTS develops more rapidly in diabetic patients based on mechanisms that are not found in individuals without diabetes [33,34].

The ongoing research on diabetic neuropathy, which contributes to the development of CTS, has failed to reliably characterize its pathophysiology and the above mechanisms. However, diabetic neuropathy is a multi-factorial pathology with two main mechanisms, i.e. metabolic and ischemic.

In the former, the excess aldose reductase activity induced by hyperglycemia plays a crucial role. In other words, when the glucose concentration is elevated, an increase in the activity of the alternative pathway of glucose conversion is observed. It is related to the polyol pathway in which glucose is converted into sorbitol by aldose reductase and then sorbitol is partially converted into fructose. High levels of glucose, sorbitol, and fructose adversely affect the normal function of peripheral nerves [35,36].

The latter mechanism is related to pathological changes in the vasa nervorum. Rheological changes in the blood, such as increased blood viscosity owing to hyperglycemia and hyperlipidemia, increased fibrinogen levels, decreased albumin levels and increased red blood cell aggregation result in decreased blood flow in the vasa nervorum and nerve injury [37]. Hence, the underlying microangiopathy can be indirectly reflected by significantly worse parameters of neuromuscular conduction in our cohort of patients.

We found that the terminal latency of the motor response of the median nerve was postoperatively significantly higher in terms of the median in patients without diabetes. In addition, the relative difference, which was statistically significantly lower in patients with diabetes, showed that those subjects had a significantly worse prognosis for the recovery of normal conduction, or had no improvement in the electrophysiological parameters, and hence no subjective improvement in the quality of life. Bearing the above in mind, the usefulness of reconstructive surgery should be considered in this group of patients (Figure 2).

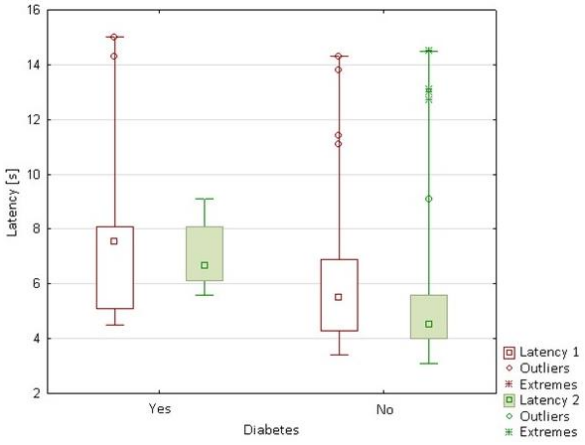

Fig. 2. Comparison of latencies of motor response in diabetic and non--diabetic patients before and after reconstructive surgery.
Ryc. 2. Porównanie latencji końcowej odpowiedzi ruchowej u pacjentów z cukrzycą i bez cukrzycy przed zabiegiem naprawczym i po zabiegu.

Finally, the disproportion between the compared subgroups (smokers 17%, diabetics 9%) we consider the limitation of the study and requires further research on a larger group of patients.

CONCLUSIONS

The results of the study demonstrated that smoking and diabetes significantly worsened the electrophysiological parameters of the nerves in our cohort of patients. Additionally, a significantly worse prognosis was found in patients with diabetes after surgery for CTS.

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Author's contribution

Study design – K. Wierzbicki, C. Linart, M. Adamczyk-Sowa
Data collection – K. Wierzbicki, K. Ślusarz, K. Romanek, B. Tadeusiak
Data interpretation – K. Wierzbicki, M. Adamczyk-Sowa, C. Linart
Statistical analysis – M. Bugdol
Manuscript preparation – K. Wierzbicki, C. Linart
Literature research – K. Wierzbicki, K. Ślusarz, K. Romanek, B. Tadeusiak

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