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Nerve Conduction in Sensory and Motor Fibers of Peripheral Nerves in Burning Mouth Syndrome

Szybkość przewodzenia we włóknach czuciowych i ruchowych nerwów obwodowych w zespole pieczenia jamy ustnej

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Abstract

Background. Burning mouth syndrome (BMS) is defined as a chronic burning sensation in the oral mucosa that can not be attributed to its pathological findings. Current opinion is that the clinical symptoms of BMS can be attributed to neurological disorders caused by disturbances in the processes of sensory information on various levels of the nervous system. In its etiology, dysfunction of the nigrostriatal dopamine-dependent system is also considered. It has been demonstrated that there are similarities between BMS and Parkinson's disease.
Objectives. To evaluate local conditions and disturbances in peripheral nerve conduction in patients with BMS.
Material and Methods. The participants of the study were 83 patients; 33 patients with burning mouth syndrome (group I), 20 patients with Parkinson's disease (group II) and 30 controls. Group I: age range 41–82 years (median age 60.4); 27 women, 6 men, group II: age range 51–81 years (median age 65.5 years); 15 women, 5 men. All patients underwent electroneurography. Motor conduction velocity, motor peroneal nerve and ulnar nerve conduction velocity distribution were analyzed. Sensory ulnar and sural nerve conduction velocity tests were also performed.
Results. Group I: the authors confirmed a statistically significant decrease of both peroneal nerve amplitude and sural nerve amplitude in patients with BMS compared to controls. In patients with BMS, the prolongation of the ulnar nerve latency of sensory action potential was also proved. Negative correlations were revealed between the age of the patients, type of BMS and most of the parameters evaluated in a standard neurography examination. Group II**:** we confirmed a statistically significant decrease of the peroneal nerve amplitude of compound muscle action potential in patients with Parkinson's disease, compared to the control group.
Conclusions. The findings justify further electrophysiological studies as well as neuropathological studies of the peripheral nervous system in patients with BMS. They could help to determine the role of peripheral changes in the disease's pathogenesis (**Adv Clin Exp Med 2011, 20, 6, 753–760**).

Key words: burning mouth syndrome, Parkinson's disease, electroneurography.

Streszczenie

Wprowadzenie. Zespół pieczenia jamy ustnej (BMS) jest definiowany jako uporczywe pieczenie błony śluzowej jamy ustnej, niezwiązane z jej patologią. Uważa się, że objawy kliniczne BMS wiążą się z zaburzeniami neurologicznymi spowodowanymi nieprawidłowym odbiorem i przetwarzaniem informacji czuciowych na różnych poziomach układu nerwowego. W etiologii schorzenia bierze się również pod uwagę dysfunkcję zależnego od dopaminy układu nigrostriatalnego. Wykazano, że istnieją podobieństwa między BMS i chorobą Parkinsona.
Cel pracy. Ocena uwarunkowań miejscowych oraz uszkodzenia nerwów obwodowych w BMS.
Materiał i metody. W badaniach udział wzięło 83 pacjentów; 33 osoby z zespołem pieczenia jamy ustnej (grupa I), 20 pacjentów z chorobą Parkinsona (grupa II) oraz 30 pacjentów z grupy kontrolnej (grupa III). Grupa I: przedział wiekowy 41–82 lat (średnia wieku 60,4); 27 kobiet, 6 mężczyzn. Grupa II: przedział wiekowy 51–81 lat (średnia wieku 65,5); 15 kobiet, 5 mężczyzn. U wszystkich chorych przeprowadzono badania elektroneurograficzne. Oceniano szybkość przewodzenia ruchowego, rozkład prędkości przewodzenia we włóknach ruchowych nerwu łokciowego i strzałkowego, a także szybkość przewodzenia czuciowego w nerwie łokciowym i łydkowym.
Wyniki. Grupa I: wykazano statystycznie istotne obniżenie amplitudy potencjałów ruchowych nerwu strzałkowego oraz amplitudy odpowiedzi czuciowych w nerwie łydkowym w grupie osób z BMS w odniesieniu do grupy kon-

trolnej, a także wydłużenie latencji odpowiedzi czuciowej w nerwie łokciowym u pacjentów z BMS w porównaniu z dwiema pozostałymi grupami. Wykazano ujemne korelacje między wiekiem pacjentów i typem BMS a większością parametrów standardowego badania neurograficznego. Grupa II: u pacjentów z chorobą Parkinsona stwierdzono znamienne obniżenie amplitudy odpowiedzi we włóknach ruchowych nerwu strzałkowego w porównaniu z grupą kontrolną.

Wnioski. Uzyskane wyniki uzasadniają prowadzenie dalszych badań elektrofizjologicznych, a także neuropatologicznych obwodowego układu nerwowego u chorych z BMS. Być może przyczyni się to do ustalenia roli zmian obwodowych w patogenezie tej choroby (**Adv Clin Exp Med 2011, 20, 6, 753–760**).

Słowa kluczowe: zespół pieczenia jamy ustnej, choroba Parkinsona, elektroneurografia.

The term burning mouth syndrome (BMS) describes various types of chronic pain experienced in the mouth by patients without clinical manifestations of oral changes which could explain this kind of discomfort. In most cases, the clinical picture of BMS is marked by a triad of symptoms: incessant pain or burning sensation in the oral mucosa, dysgeusia and xerostomia, with no clinically observable changes in the oral mucosa including the site of the reported pain [1]. In other cases, there is either an oligosymptomatic variant (pain and dysgeusia or pain and xerostomia) or just one symptom occurs – pain. Studies show that burning mouth syndrome is a frequent reason for medical consultation requests [2, 3]. In the United States, about 1.3 million people with BMS have been registered [4].

Three clinical types of BMS are distinguished, depending on the intensity of the burning sensation and its occurrence patterns during the day (Lamey and Lewis criteria) [5]. In type 1, the ailments occur later in the day and are the most intense in the evening. This type is usually associated with systemic diseases such as diabetes and malnutrition [6]. In type 2, the sensations persist throughout the day and make falling asleep difficult. In this group, psychological, neurological and psychiatric disorders are usually observed [7]. In BMS Type 3, varied patterns are reported. Patients report days without symptoms. It is associated with allergic reactions to food additives as well as anxiety [8].

Also, various local and systemic factors are of significance in BMS etiopathogenesis. Among the local factors, the following are named: lichen planus, mechanical irritation, contact allergies, parafunction and dysfunction of the stomatognathic system and prosthetic factors [6–10]. The predisposing factors are: diabetes, hematological disorders, deficiency in platelet producing factors (vitamin B12, iron and folic acid), hormonal disorders (menopause), digestive tract disorders, psychiatric and neurological disorders and polypragmasy [1, 11–15].

Recent studies have attributed burning mouth syndrome to disturbances in the processes of sensory information in the peripheral and central nervous system [16, 17]. Sufferers may experience disturbances in the perception of pain and thermal stimuli [18]. Increased trigeminal nerve sensitivity is also considered [19]. According to some researchers, hyperexcitability of the trigeminal nerve may result from pathologies in the neurovascular micro-circulation in the oral mucosa or damage to the brain stem or nerve V fibers (fiber sensory neuropathy) [17, 20, 21]. When analyzing the etiopathogenesis of BMS, dysfunction of the nigrostriatal system is also considered, similar to that occurring in degenerative diseases, manifesting as dopaminergic dysfunction [22, 23]. Dysfunctions of the nigrostriatal system lead to insufficient, dopamine-dependent, control of pain [23]. Almost half of the Parkinson's disease (PD) sufferers have various sensory complaints, including pain and a burning sensation in the mouth [23] as well as alterations in taste [24, 25].

The aim of the study was to evaluate local conditions and damage to the peripheral nerves in BMS. Seeing as there are suggested similarities between the disturbances in neuronal transmission in BMS and Parkinson's disease, the reference groups were PD sufferers and healthy subjects.

Material and Methods

The subjects were divided into three groups. Group I consisted of 33 patients with burning mouth syndrome without any clinical manifestations of Parkinson's syndrome who sought help in the Department of Oral Pathologies at Wroclaw Medical University. The second group was comprised of 20 patients with Parkinson's disease without burning mouth syndrome, diagnosed in the Clinic of Neurology at Wroclaw Medical University. The third group consisted of 30 patients without symptoms of burning mouth syndrome or Parkinson's disease (controls).

Criteria for exclusion: patients with clinical manifestations of pathological changes in the oral mucosa, with positive fungal culture of pathogenic units of *Candida* in the oral cavity or secondary BMS in accord with Scala et al. [1] criteria as a re-

Table 1. Values of Spearman's correlation ratios between latency, amplitude and conduction velocity in motor and sensory peripheral nerves and the patient's age, type of BMS, disease duration (years), intensity of pain (VAS scale) and polypragmasy in patients with BMS

Tabela 1. Wartości współczynników korelacji Spearmana między latencją, amplitudą i prędkością przewodzenia we włóknach ruchowych i czuciowych nerwów obwodowych a wiekiem pacjentów, typem BMS, czasem trwania dyskomfortu (lata) oraz jego intensywnością (skala VAS), a także polipragmazją u chorych z BMS

	Latency of Ulnar n. (motor fibers)	Amplitude of ulnar n. (motor fibers)	Velocity of ulnar n. (motor fibers)	Latency of ulnar n. (sensory fibers)	Amplitude of ulnar n. (sensory fibers)	Velocity of ulnar n. (sensory fibers)	Latency of peroneal n. (motor fibers)	Amplitude of peroneal n. (motor fibers)	Velocity of peroneal n. (motor fibers)	Latency of Sural n. (sensory fibers)	Amplitude of Sural n. (sensory fibers)	Velocity of Sural n. (sensory fibers)
Patient's age (Wiek pacjenta)	0.13	–0.27	–0.37 p = 0.03	–0.07	–0.53 p = 0.00	–037 p = 0.03	0.23	–0.44 p = 0.01	–0.26	–0.08	0.02	–0.28
Type of BMS (Rodzaj BMS)	0.11	–0.15	0.06	0.28	–0.29	–0.09	0.24	–0.2	–0.08	–0.19	–0.37 p = 0.03	–0.37 p = 0.03
Disease duration (Czas trwania choroby)	–0.04	–0.22	0.07	0.1	–0.17	0.19	0.06	–0.16	0.13	–0.24	0.03	–0.11
VAS scale (Skala VAS)	0.19	–0.1	0.1	0.03	0.03	0.06	–0.13	–0.17	–0.11	0.3	–0.17	–0.03
Polipragmasy (Polipragmazja)	–0.07	–0.25	0.01	0.04	–0.2	0.05	–0.02	–0.04	0.00	0.06	0.18	0.12

Table 2. Values of Spearman's correlation ratios between conduction velocity distribution (CVD) percentage in motor ulnar and peroneal nerve and the age, type of BMS and disease duration (years), intensity of pain (VAS scale) and polypragmasy in patients with BMS

Tabela 2. Wartości współczynników korelacji Spearmana między procentowym rozkładem prędkości przewodnictwa (CVD) w zakresie włókien ruchowych nerwu łokciowego i strzałkowego a wiekiem pacjentów, typem BMS, czasem trwania dyskomfortu oraz jego intensywnością bólu w skali VAS, a także polipragmazją u pacjentów z BMS

	CVD Ulnar nerve 10%	CVD Ulnar nerve 50%	CVD Ulnar nerve 90%	CVD Peroneal nerve 10%	CVD Peroneal nerve 50%	CVD Peroneal nerve 90%
Patient's age (Wiek pacjenta)	0.02	–0.08	–0.27	0.00	0.02	0.12
Type of BMS (Rodzaj BMS)	0.13	0.24	0.07	0.21	0.23	0.27
Disease duration (Czas trwania choroby)	0.02	0.07	0.09	0.14	0.16	0.19
VAS scale (Skala VAS)	0.11	0.18	0.25	0.15	0.2	0.27
Polipragmasy (Polipragmazja)	0.35 p = 0.047	0.28	0.13	0.08	0.13	0.26

sult of platelet producing factor deficiency (vitamin B12, folic acid, group B vitamins: B1, B2, B6, PP), diabetic patients and patients with glucose intolerance.

The age ranges for the groups were as follows: group I: from 41 to 82 years (mean age 61.5 ± 9.4, median 60.0), 27 women, 6 men; group II: 51–81 years (mean age 65.6 ± 8.4, median 66.5), 15 women, 5 men; group III: 42–83 years (mean age 60.5 ± 10.5, median 59.0) 20 women, 10 men. All participants received information about the study project and gave their conscientious consent. The study received a positive opinion from the Bioethical Committee at Wroclaw Medical University. Medical histories were taken which included concomitant diseases, currently taken medication, duration of symptoms in years and intensity of pain (measured on the VAS scale). Type of BMS was determined in accordance with the Lamey and Lewis criteria [5].

Electroneurography was conducted in the Neuroelectrophysiology Laboratory of the Neurology Clinic at Wroclaw Medical University. The apparatus used was a Viking Selekt by Nicolet. The study was conducted in accordance with standard research methods [26]. The researchers assessed ulnar and left peroneal nerve conduction velocity, as well as motor nerve conduction velocity distribution (CVD). Sensory ulnar and left sural nerve conduction velocity was conducted using the antidromic method. Body temperature was kept at 32–33°C.

The hypothesis that the means of the parameters in the groups are equal was verified with an ANOVA test and in groups with heterogeneous variance with a non-parametric Kruskal-Wallis test by ranks (homogeneity of variance was tested with a Bartlett test). For parameters with significant differences when 3 groups were compared, a post-hoc test was run – an analysis of contrasts with a Scheffe test. Correlation analysis was run for selected pairs of parameters with a Spearmann's or Pearson's correlation coefficient. For each test $P \leq 0.05$ was statistically significant. Statistical analysis was performed with a computer program for statistics, EPIINFO ver. 3.4.3 (dated 8.11.2007).

Results

The results of the correlation analysis between electroneurographic variables, age of BMS sufferers, BMS type, duration of discomfort (in years) and its intensity on the VAS scale, as well as applied polypragmasy, are presented in Tables 1 and 2. There was a negative correlation between the majority of parameters of the standard neurographic study and patients' age and BMS type. No significant correlations between other assessed parameters were found.

The results of the analysis of inter-group differences (group I, II, III) for latency, amplitude and motor conduction velocity and sensory conduction velocity are shown in Table 3. There was a statis-

Table 3. Inter-group analysis (group I, II, III) of latency, amplitude and conduction velocity in motor and sensory ulnar, peroneal and sural nerves

Tabela 3. Analiza międzygrupowa (grupa I, II, III) latencji, amplitudy i prędkości przewodzenia we włóknach ruchowych i czuciowych nerwu łokciowego, strzałkowego i łydkowego

	Group I – BMS (Grupa I – BMS)						Group II – Parkinson's disease (Grupa II – choroba Parkinsona)						Group III – control (Grupa III – kontrolna)						p
	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	
lat. ulnar n. (motor f.)	2.6	2.5	0.3	2.1	3.4	33	2.52	2.5	0.5	1.4	3.3	20	2.73	2.7	0.34	2.3	3.4	30	0.116
ampl. ulnar n. (motor f.)	9.4	9.2	2.28	5.1	13.05	33	9.1	8.82	1.79	6.12	13.54	20	9.74	9.71	1.91	6.12	13.32	30	0.538
veloc. ulnar n. (motor f.)	59.8	58.9	4.8	50	71.4	33	55.5	57.8	14	0.6	73.3	20	58	60.6	11.8	0.6	73.3	30	0.353
lat. ulnar n. (sensory f.)	3.63 **	3	1.89	2.3	12.2	33	2.74	2.61	0.46	2.06	3.8	20	2.39	2.3	0.33	2.06	3.3	30	0.000
ampl. ulnar n. (sensory f.)	31.2	26.9	19.9	0	100.4	33	22.4	21.7	8.1	3.2	37.6	20	25.4	25.1	9.3	11.5	45	30	0.305
veloc. ulnar n. (sensory f.)	52	53.1	15.4	0	70.1	32	58.6	57.6	9.4	40.6	76.9	20	54.4	54.2	4.8	47	63.4	30	0.118
lat. peroneal n. (motor f.)	4.34	4.3	0.61	3.4	5.7	33	4.17	4.3	0.65	3.1	5.4	19	4.55	4.5	0.57	3.6	5.4	30	0.103
ampl. peroneal n. (motor f.)	4.56*	4.33	2.42	0.45	10.12	33	5.44***	5.66	3.12	0.66	10.29	19	8.2	8.41	1.6	4.69	11.48	30	0.000
veloc. peroneal n. (motor f.)	49	49.1	4	41.6	56.1	33	49.2	47.5	4.8	43.9	61.5	19	50.9	50.4	3.4	45.7	61.5	30	0.147
lat. sural n. (sensory f.)	2.83	2.85	1.02	0	4.7	24	2.78	2.76	0.35	2.2	3.7	18	2.72	2.69	0.24	2.35	3.28	30	0.114
ampl. sural n. (sensory f.)	16.2 *	15.4	10.4	0	45.7	33	20.7	19.5	10.9	0	45.7	19	21.9	20.5	6.7	12	38	30	0.043
veloc. sural n. (sensory f.)	49	53.9	16.9	0	68.9	32	53.8	54.7	14.3	0	76.2	19	51.9	52.6	3.8	45.3	59.3	30	0.051

* – significant difference between group I and III.
** – significant difference between group I and II and between group I and III.
*** – significant difference between group II and III.

* – różnica istotna statystycznie między grupą I i III.
** – różnica istotna statystycznie między grupą I i II oraz między I i III.
*** – różnica istotna statystycznie między grupą II i III.

Table 4. Inter-group analysis (group I, II, III) of conduction velocity distribution in motor ulnar and peroneal nerves

Tabela 4. Analiza międzygrupowa (grupa I, II, III) procentowego rozkładu prędkości przewodnictwa (CVD) dotyczącego włókien ruchowych nerwu łokciowego i strzałkowego

	Group I – BMS (Grupa I – BMS)						Group II – Parkinson's disease (Grupa II – choroba Parkinsona)						Group III –control (Grupa III – kontrolna)						p
	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	
CVD ulnar n. 10%	44.8	46.2	6.8	21	53.8	32	47.5	49	7.8	33	60.4	18	46.6	47.7	5.1	37.6	59	30	0.318
CVD ulnar n. 50%	52.2	52.7	4.6	39.6	62.7	33	52.4	53.4	7	40	66.8	20	53.4	53	5.2	41.7	65.1	30	0.664
CVD ulnar n. 90%	57.1	57.7	4.6	45.6	68.1	33	57	57.2	6.5	44.8	72.8	20	58.6	58.3	5.5	49.2	70.4	30	0.475
CVD peroneal n. 10%	39.5	40.4	4.8	29.5	48.4	32	36	40	14.4	0	59.5	18	40.6	40.3	2.4	36	46.1	30	0.667
CVD peroneal n. 50%	44.2	45.3	4.6	34.6	55.8	32	41.6	43.6	11.1	0	56.1	18	44.8	45.4	2.7	38.4	50.7	30	0.209
CVD peroneal n. 90%	47.4	48.8	4.6	37.9	60.8	32	46.7	46.9	3.2	39.6	51.5	18	48.2	48.3	2.4	45.3	53.2	30	0.349

tically-significant decrease of the peroneal nerve amplitude of compound muscle action potential as well as of the sensory response in the sural nerve in the BMS group compared to controls. There was also a prolonged latency of sensory response in the ulnar nerve in patients with BMS compared to the other two groups. There was a marked decrease of the peroneal nerve amplitude of compound muscle action potential in Parkinson's sufferers compared to controls. The analysis of motor conduction velocity distribution in the studied groups did not demonstrate any statistically significant differences between the groups (Table 4).

Discussion

As there are no established diagnostic methods and treatment options for patients with BMS, there is need for further studies on the disease's etiology. A great heterogeneity of sufferers may hinder the task. Current literature more and more often emphasizes the connection between BMS and disorders of the central and peripheral nervous system [16–23, 27, 28]. According to Friedman et al. [28], the impairment of both the central and peripheral nervous system in patients with PD is quite often observed. The authors of the current study analyzed neurographic parameters and compared the results with the findings in healthy controls and Parkinson's sufferers in order to exclude the potential effect of central disorders on the function of the peripheral nervous system and neurographic results [29].

Grushka et al. [30] have suggested the hypothesis that damage to peripheral nerves is the underlying cause of BMS. Nevertheless, the authors' findings do not give straight answers as to the presence of neuropathy. The range of neurographic studies was extended to include CVD. In contrast to standard studies, it allows for assessment of the function of small motor fibers (low conduction velocity). Nevertheless, the researchers did not assess small sensory fibers and non-myelinated fibers although such extension of further studies is planned. In their invasive immunohistochemical studies, Lauria et al. [31] demonstrated the presence of sensory neuropathy of small fibers within fungiform lingual papilla innervated by cranial nerves in patients with BMS (lack of epithelium and degenerative changes in sub-papillary nerve bundles). Statistically significant changes demonstrated for singular parameters of a standard sensory conduction study may suggest sub-clinical damage to the peripheral nervous system. The analysis of correlations between sensory conduction parameters and the clinical features of burning mouth syndrome showed a link with BMS type 2, in accordance with Lamey criteria, which may apply to patients with damage to the nervous system [7]. In turn, Eliav et al. [32] demonstrated at least a unilateral raising of the pain and taste threshold in 18 out of 22 patients with BMS when studying the evoked potentials of chorda tympani (containing, among others, sensory fibers innerving fungiform papilla in the anterior two-thirds of the tongue). Furthermore, the researchers noted that in about 2 cm of the tongue tip, the chorda tympani crosses over, which may explain the high frequency of occurrence of a burning sensation on this site in the early stages of the disease. Further electrophysiological studies of the peripheral nervous system using modern techniques are fully justified in patients with BMS. They could help to determine the significance of peripheral changes in the disease's pathogenesis. The researchers plan to extend the electrophysiological assessment of small fibers in peripheral nerves with the application of quantitative sensory tests and analysis of cold, warm and heat pain thresholds.

References

[1] **Scala A, Checchi L, Montevecchi M, Marini I:** Update on burning mouth syndrome: overview and patient management. Crit Rev Oral Biol Med 2003, 14, 275–291.

[2] **Witt E, Palla S:** Mundbrennen. Schmerz 2002, 5, 389–394.

[3] **Pinto A, Sollecito T, DeRossi S:** Burning mouth syndrome. A retrospective analysis of clinical characteristics and treatment outcomes. NYSDJ 2003, 3, 18–24.

[4] **Lipton JA, Ship JA, Larach-Robinson D:** Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993, 124, 115–121.

[5] **Lamey PJ, Lewis MA:** Oral medicine in practice: burning mouth syndrome. Br Dent J 1989, 167, 197–200.

[6] **Lamey PJ, Lamb AB:** Prospective study of etiological factors in burning mouth syndrome. Br Med J 1988, 296, 1243–1246.

[7] **Eli I, Kleinhautz M, Baht R, Littner M:** Antecedents of burning mouth syndrome (glossodynia) – recent life events vs. psychopathologic aspects. J Dent Res 1994, 73, 567–572.

[8] **Lamey PJ, Lamb AB, Hughes A, Milligan KA, Forsyth A:** Type 3 burning mouth syndrome: psychological and allergic aspects. J Oral Pathol Med 1994, 23, 216–219.

[9] **Svensson P, Kaaber S:** General health factors and denture function in patients with burning mouth syndrome and matched control subjects. J Oral Rehab 1995, 22, 887–895.

[10] **Zakrzewska JM:** The burning mouth syndrome remains an enigma. Pain 1995, 62, 253–257.
[11] **Koszewicz M, Budrewicz S, Koziorowska-Gawron E, Ejma M, Kusińska E:** Diagnostyka elektrofizjologiczna w zespole pieczenia jamy ustnej u pacjentów z chorobą Parkinsona. Neurol Neurochir Pol 2008, 42, 31–35.
[12] **Femiano F, Lanza A, Buonaiuto C, Gombos F, Nunziata M, Cuccurullo L, Cirillo N:** Burning mouth syndrome and burning mouth in hypothyroidism: proposal for a diagnostic and therapeutic protocol. Oral Surg Oral Med Oral Pathol 2008, 105, 22–27.
[13] **Carrington J, Getter L, Brown R, MS:** Diabetic neuropathy masquerading as glossodynia. JADA 2001, 132, 1549–1551.
[14] **Cibirka R, Nelson S, Lefebvre C:** Burning mouth syndrome: a review of etiologies. J Prosthet Dent 1997, 78, 93–97.
[15] **Muzyka BC, De Rossi SS:** A review of burning mouth syndrome. Cutis 1999, 64, 29–35.
[16] **Grushka M, Sessle B, Miller R:** Pain and personality profiles in burning mouth syndrome. Pain 1987, 28, 155–167.
[17] **Forssell H, Jääskeläinen S, Tenovuo O, Hinkka S:** Sensory dysfunction in burning mouth syndrome. Pain 2002, 99, 41–47.
[18] **Svensson P, Bjerring P, Arendt-Nielsen L, Kaaber S:** Sensory and pain thresholds to orofacial argon laser stimulation in patients witch chronic burning mouth syndrome. Clin J Pain 1993, 9, 207–215.
[19] **Gao S, Wang Y, Wang Z:** Assessment of trigeminal somatosensory evoked potentials in burning mouth syndrome. Chin J Dent Res 2000, 3, 40–46.
[20] **Heckmann SM, Heckmann JG, Hilz MJ, Popp M, Marthol H, Neundörfer B, Hummel T:** Oral mucosal blood flow in patients with burning mouth syndrome. Pain 2001, 90, 281–286.
[21] **Jääskeläinen S, Forssell H, Tenovuo O:** Abnormalities of the blink reflex in burning mouth syndrome. Pain 1997, 73, 455–460.
[22] **Kimura J:** Disorder of interneurons in parkinsonism. The orbicularis oculi reflex to paired stimuli. Brain 1973, 96, 87–96.
[23] **Jääskeläinen S, Rinne J, Forssell H, Tenovuo O, Kaasinen V, Sonninen P, Bergman J:** Role of the dopaminergic system in chronic pain – a fluorodopa-PET study. Pain 2001, 90, 257–260.
[24] **Grushka M, Sessle B:** Burning mouth syndrome. Dent Clin North Am 1991, 35, 171–184.
[25] **Grushka M, Sessle B, Howley T:** Psychophysical evidence of taste dysfunction in burning mouth syndrome. Chemical Senses 1986, 11, 4, 485–498.
[26] **Oh SJ:** Clinical electroneurography. Nerve conduction studies. Lippincot Williams and Wilkins, 3rd ed., Philadelphia 2003, 86–106.
[27] **Mendak M, Konopka T, Koszewicz M, Koziorowska-Gawron E, Ejma M, Budrewicz S, Kusińska E:** Badanie potencjałów wywołanych i odruchu mrugania u pacjentów z zespołem pieczenia jamy ustnej lub chorobą Parkinsona. Dent Med Probl 2010, 47, 2, 169–176.
[28] **Friedman A:** Choroba Parkinsona – mechanizmy, rozpoznanie, leczenie. Wydawnictwo Czelej, Lublin 2005.
[29] **Koszewicz M:** Elektrofizjologiczny obraz transmisji nerwowo-mięśniowej w ośrodkowym uszkodzeniu układu nerwowego. Praca doktorska, Wrocław 1995.
[30] **Grushka M, Epstein JB, Gorsky M:** Burning mouth syndrome and other oral sensory disorders: a unifying hypothesis. Pain Res Manag 2003, 8, 133–135.
[31] **Lauria G, Majorana A, Borgna M, Lombardi R, Penza P, Padovani A, Sapelli P:** Trigeminal small-fiber sensory neuropathy causes burning mouth syndrome. Pain 2005, 115, 332–337.
[32] **Eliav E, Kamran B, Schaham R, Czerninski R, Gracely R, Benoliel R:** Evidence of chorda tympani dysfunction in patients with burning mouth syndrome. J Am Dent Assoc 2007, 138, 628–633.

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