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Similarities Between Burning Mouth Syndrome and Parkinson's Disease in Selected Electroneurophysiological Studies

Podobieństwa między zespołem pieczenia jamy ustnej a chorobą Parkinsona w wybranych badaniach elektroneurofizjologicznych

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Abstract

Background. Burning mouth syndrome (BMS) is a chronic oral pain condition without any signs of mucosal pathology. It has a complex etiology and constitutes a therapeutic challenge. Nigrostriatal disturbances with dopaminergic hypofunction, similar to those occurring in neurodegenerative diseases such as Parkinson's disease (PD), may play a role in the etiopathogenesis of BMS.
Objectives. To compare the neurophysiological results of patients suffering from BMS and patients with PD.
Material and Methods. The study involved 83 patients: 33 with BMS (Group I), 20 with PD (Group II) and 30 control patients (Group III). In Group I the age range was 41–82 (median age 60.4); the group included 27 females and 6 males. In Group II the age range was 51–79 (median age 65.5); there were 15 females and 5 males. All the patients underwent neurophysiological tests (trigeminal sensory evoked potentials – TSEP, and brainstem auditory evoked potentials – BAEP) and a blink reflex (BR) examination.
Results. In three cases of patients with PD, the coexistence of BMS was confirmed. Group I: The TSEPs were abnormal in 18 patients, mostly bilaterally (15 cases). The BAEPs were disturbed in 18 patients, mostly bilaterally (12 cases). There were central disturbances in 11 patients, mixed (central and peripheral) disturbances in 3 cases, and peripheral disorders in 4 patients. In the blink reflex examination, there was no habituation in 16 patients. Group II: The TSEPs were bilaterally abnormal in 8 patients (prolongation of latencies in 6 cases, pathological asymmetry of amplitudes in 4 cases). The BAEPs were abnormal in 6 patients with PD; they were bilateral and central in all cases. In the blink reflex examination, there was no habituation in 15 patients.
Conclusions. Subjective symptoms suggesting dysfunction of sensory pathways in people with BMS are in most cases confirmed by neurophysiological tests such as TSEP, BAEP and BR. The abnormalities found in the electroneurophysiological studies in patients with burning mouth syndrome are similar to those found in Parkinson's disease. Therefore, it appears that pharmacotherapy for BMS might focus on stimulating the dopaminergic system **(Adv Clin Exp Med 2010, 19, 6, 731–738)**.

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

Streszczenie

Wprowadzenie. Zespół pieczenia jamy ustnej (BMS) jest przewlekłym dyskomfortem w jamie ustnej, niezwiązanym z patologią błony śluzowej. Choroba ta ma złożoną etiologię i sprawia wiele trudności w leczeniu. W rozważaniach nad etiopatogenezą BMS bierze się pod uwagę zaburzenia nigrostriatalnego układu dopaminergicznego, podobne do występujących w chorobach neurodegeneracyjnych, takich jak choroba Parkinsona (PD).
Cel pracy. Porównanie wyników wybranych badań neurofizjologicznych u pacjentów z BMS i u pacjentów z chorobą Parkinsona.
Materiał i metody. Badaniami objęto 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 51–79 lat (średnia wieku 65,5); 15 kobiet, 5 mężczyzn. U wszystkich pacjentów przeprowadzono badania neuroelektrofizjologiczne

(TSEP – potencjały wywołane z nerwu trójdzielnego, BAEP – pniowe słuchowe potencjały wywołane) oraz badanie BR (odruch mrugania).
Wyniki. U trzech pacjentów z PD stwierdzono współistnienie BMS. Grupa I: W badaniu TSEP w grupie osób z BMS u 18 pacjentów uzyskano nieprawidłowe wyniki, w większości o charakterze obustronnym (15 przypadków). W badaniu BAEP u 18 osób z BMS stwierdzono nieprawidłowości, w większości (12 przypadków) miały one charakter obustronny. Zaburzenia ośrodkowe występowały u 11 osób, mieszane (ośrodkowo-obwodowe) u 3, a obwodowe u 4 pacjentów. W badaniu BR u 16 pacjentów z BMS nie stwierdzono habituacji odruchu mrugania. Grupa II: W badaniu TSEP u 8 pacjentów z PD stwierdzono obustronne nieprawidłowości w postaci wydłużonej latencji załamków (6 przypadków) i patologicznej asymetrii amplitud między stronami (4 przypadki). Patologiczne zapisy BAEP dotyczyły 6 pacjentów z PD i we wszystkich przypadkach miały charakter obustronny i ośrodkowy. W badaniu BR w grupie osób z PD brak habituacji stwierdzono u 15 pacjentów.
Wnioski. Subiektywne objawy sugerujące nieprawidłową czynność ośrodkowych dróg czuciowych u osób z BMS w większości znajdują potwierdzenie w wynikach badań BAEP, TSEP, BR. Nieprawidłowości w wykonanych badaniach elektroneurofizjologicznych u pacjentów z zespołem pieczenia jamy ustnej są podobne do stwierdzanych w chorobie Parkinsona. W tej sytuacji wydaje się, że farmakoterapia BMS powinna być skierowana na stymulację układu dopaminergicznego **(Adv Clin Exp Med 2010, 19, 6, 731–738)**.

Słowa kluczowe: zespół pieczenia jamy ustnej, choroba Parkinsona, badanie neuroelektrofizjologiczne.

Burning mouth syndrome (BMS) is a chronic pain syndrome that most frequently affects elderly and middle-aged women. It has a complex etiology and constitutes a therapeutic challenge. In BMS the patients' main complaint is pain in oral mucosa that appear to be healthy in clinical tests. The pain occurs as a burning sensation, possibly with concomitant paresthesia, dysgeusia, xerostomia and oversensitivity to food. The tongue is the most common site of the burning sensation, but burning can occur in other parts of the mouth as well [1–3]. In population studies, BMS vacillated between 0.7–14.8% [4–6]. The difference resulted from different diagnostic criteria assumed for the syndrome.

The etiopathogenesis of burning mouth syndrome is multifactorial. Currently, its etiology is associated with a disturbance in the processing of sensory information on various levels of the central and peripheral nervous system [7, 8] as well as with dysfunctions of the nigrostriatal pathway, similar to those occurring in degenerative diseases and diseases manifesting as dopaminergic dysfunction (for example, Parkinson's disease – PD) [9, 10]. Experimental studies show that presynaptic dopaminergic terminal dysfunction of the nigrostriatal system occurs, which is associated with decreased striatal capture of labeled 6-[18F] fluorodopa [9]. Nigrostriatal dysfunction, on both the presynaptic and synaptic levels, causes insufficient central dopamine-dependent control of pain sensations [10]. Studies show that 40% of patients with Parkinson's disease have sensory complaints, including pain and a burning sensation in the mouth [9]. The theory of neuropathic causes of BMS also seems to be supported by the fact that dysgeusia, dysfunction of sensory processes and chemosensory dysfunction are common among BSM patients [11–14]. It has been proved that the perception of the intensity of subthreshold pain stimuli in patients with burning mouth syndrome may be abnormal, as their perception of nociceptive and non-nociceptive thermal stimuli can be as well [15]. They may have increased trigeminal nerve sensitivity and changes in neuronal transmission [16]; and changes in neurovascular micro-circulation in the oral mucosa [17]. In electroneurological studies Jaaskeleinen et al. [18] observed that patients with BMS demonstrate decreased habituation of the blink reflex. Other studies have recorded hyperexcitability of the trigeminal nerve, or decreased excitability resulting from damage to the brainstem or fiber sensory neuropathy [8, 9, 18].

The objective of this study was to use selected electroneurophysiological examinations to determine whether there are similarities between burning mouth syndrome and Parkinson's disease. A confirmation of such similarities might open a door for new treatment options for patients with BMS.

Material and Methods

The material consisted of 83 patients divided into three study groups:

Group I consisted of 33 patients with burning mouth syndrome who sought help in the Department of Oral Pathology at Wroclaw Medical University. Their age range was 41 to 82 years (mean age 61.5 ± 9.4, median age 60.0); there were 27 women and 6 men.

Group II included 20 patients with Parkinson's disease hospitalized in the Department of Neurology at Wroclaw Medical University; age range: 51–81 years (mean age 65.6 ± 8.4, median age 66.5); 15 women, 5 men.

Group III was the control group of 30 patients

showing no symptoms of burning mouth or Parkinson's disease; age range 42–83 years (mean age 60.5 ± 10.5, median age 59.0), 20 women, 10 men.

Criteria for exclusion were as follows: patients with clinical manifestations of pathological changes in the oral mucosa which could cause pain; patients with secondary BMS as a result of a deficiency in platelet producing factors, vitamin deficiency and/or diabetes; patients with positive fungal cultures of pathogenic *Candida* strains in the oral cavity.

Medical histories were taken, including current medication, the duration of symptoms in years and the intensity of pain (measured on the Visual Analog Scale – VAS). The type of BMS was determined in accordance with the Lamey and Lewis criteria [19].

All the patients included in the study received information about the study project and gave their informed consent. The study was approved by the Bioethical Committee at Wroclaw Medical University.

Studies of brainstem auditory evoked potentials (BAEP) were conducted in a darkened soundproofed room, as described in a previous publication [20]. During the examination the patient lay on a reclining chair. The study was conducted using a Nicolet CA-1000 apparatus. As auditory stimulus, a click of 0.1 ms duration, 20.3 Hz frequency and 65 dB intensity above the individually determined auditory threshold was delivered to one ear. Simultaneously, humming of 35 dB intensity above the hearing threshold was delivered to other (unexamined) ear. Responses were recorded with an electrode placed on the mastoid process on the same side as the examined ear. A reference electrode was localized on top of the head. Analysis lasted 10 ms. For each patient 2000 responses were recorded and averaged in the 150–3000 Hz frequency range. The absolute latencies of consecutive components of BAEP were analyzed: waves I–V and interpeak latencies I–III, III–V and I–V. Prolonged interpeak latencies I–III and/or III–V were viewed as pathological if they were accompanied by prolonged I–V latencies; so was a prolonged latency of wave I, if accompanied by changes in latency of further components of brainstem auditory responses.

The trigeminal nerve somatosensory evoked potentials (TSEP) studies were also conducted in a darkened soundproofed room, as previously described [20]. The patient lay on a reclining chair, and a Nicolet CA-1000 apparatus was used. The evoked potentials were induced by one-sided stimulation of the trigeminal nerve in the area of the lip corner with a rectangular current 0.1 s in duration at a frequency of 3Hz. The applied stimuli were of an intensity three times the perception threshold. The electrode recording the responses was placed contralaterally, in the parietal area (C3 and C4), and a reference electrode was placed on the top of the head (Cz). During the study a curve with a 50-ms base was analyzed after averaging of 200 cortical responses. The latency and amplitude of the cortical response was calculated; it consisted of two positive (P) and two negative (N) peaks: N1, P1, N2, P2. The potential was viewed as abnormal if there was no response or if its latency was prolonged, and/or if the interlatency of the main components was prolonged.

The blink reflex (BR) is a brainstem reflex and consists of bilateral contraction of the orbicularis oculi muscles in response to unilateral placement of stimuli in the supraorbital area. Electromyographic reflex responses to stimulation of the supraorbital nerve with an electric current consists of an early unilateral response (R1), a late bilateral response (R2) and a contralateral response (R2'). The BR tests were conducted as reported in a previous study [20], with the patient being examined patient lying relaxed in a reclining chair. The response electrodes were placed bilaterally over the orbicular muscles of eye, and a reference electrode was placed on the nose. On each side, 8 consecutive electrical stimuli at a frequency of 1 Hz were used. The stimulus intensity was constant during the examination and the induced response had constant parameters with R1 and R2 components. Subsequently, latencies of components R1, R2 and R2' were evaluated bilaterally, as were habituation phenomena. A normal result was at least 50% diminution of the R2 area in responses 3:1 in 8 consecutive responses.

The hypothesis that the means of the parameters in the groups are equal was verified with the ANOVA test and, in groups with non-heterogeneous variance, with the non-parametric Kruskal-Wallis test (the homogeneity of variance was tested with Bartlett's test). For parameters with significant differences when the three groups were compared, a post-hoc test was run – an analysis of contrasts with the Scheffé test. For discrete parameters, the distribution in groups was tested with the χ^2 test with Yates correction. A correlation analysis was run for selected pairs of parameters with Spearmann's or Pearson's correlation coefficient.

For each test a p-value ≤ 0.05 was statistically significant. A statistical analysis was performed using EPIINFO Ver. 3.4.3 statistical software (dated 8.11.2007).

Results

The findings of the analysis of differences between the groups' BAEP examinations are shown in Table 1. The only differences that had no sta-

Table 1. Analysis of the differences between groups in the brainstem auditory evoked potentials (BAEP) examination (using the ANOVA test and in groups with non-heterogeneous "p" variance, the non-parametric Kruskal-Wallis test)

Tabela 1. Analiza międzygrupowa słuchowych pniowych potencjałów wywołanych (BAEP) (test analizy wariancji ANOVA lub dla grup o niejednorodnej wariancji „p*" test nieparametryczny sumy rang Kruskala-Wallisa)

	I						II						III						p
	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	
I-R	1.92***	1.96	0.21	1.36	2.24	33	1.74	1.78	0.14	1.4	1.9	20	1.73	1.75	0.13	1.44	1.94	30	0.000
II-R	3.14**	3.08	0.26	2.64	3.64	29	2.97	3	0.17	2.64	3.28	15	2.94	2.92	0.2	2.65	3.44	29	0.007*
III-R	4.27***	4.28	0.23	3.52	4.64	33	4.05º	4.09	0.21	3.52	4.28	20	3.92	3.94	0.17	3.54	4.14	30	0.000
IV-R	5.59***	5.68	0.29	5	5.96	31	5.35	5.36	0.33	4.36	5.8	20	5.17	5.2	0.26	4.6	5.64	27	0.000
V-R	6.4**	6.48	0.29	5.6	6.96	33	6.15º	6.08	0.37	5.54	6.64	20	5.89	5.96	0.23	5.36	6.2	30	0.000*
I–III-R	2.35**	2.32	0.22	1.84	3	33	2.31	2.33	0.14	1.96	2.48	20	2.19	2.2	0.18	1.72	2.44	30	0.003
III–V-R	2.13**	2.12	0.17	1.72	2.52	33	2.1	2.09	0.24	1.48	2.48	20	1.97	2	0.21	1.46	2.48	30	0.008
I–V-R	4.49**	4.52	0.27	3.92	5	33	4.42º	4.4	0.28	3.9	4.76	20	4.16	4.21	0.19	3.71	4.46	30	0.000
I-L	1.92***	1.96	0.22	1.36	2.32	33	1.69	1.72	0.18	1.24	1.88	20	1.73	1.75	0.14	1.48	1.98	30	0.000
II-L	2.84	3.04	0.94	0	3.56	33	2.99	2.96	0.21	2.6	3.34	20	2.82	2.9	0.57	0	3.32	30	0.668
III-L	4.28***	4.28	0.19	3.72	4.72	33	4.12º	4.18	0.26	3.72	4.44	20	3.9	3.9	0.17	3.52	4.16	30	0.000*
IV-L	4.88	5.52	1.86	0	6.08	33	5.07	5.25	1.23	0	5.8	20	4.84	5.18	1.35	0	5.76	30	0.865
V-L	6.38**	6.44	0.27	5.72	7.04	33	6.16º	6.3	0.44	5.36	6.68	20	5.87	5.9	0.23	5.36	6.18	30	0.000*
I–III-L	2.36**	2.32	0.22	2	2.88	33	2.43º	2.5	0.21	2	2.92	20	2.17	2.2	0.19	1.62	2.48	30	0.000
III–V-L	2.1	2.12	0.16	1.72	2.52	33	2.04	2	0.26	1.46	2.4	20	1.97	1.96	0.22	1.5	2.52	30	0.059
I–V-L	4.46**	4.52	0.26	3.92	4.88	33	4.47º	4.56	0.39	3.71	5.31	20	4.14	4.18	0.18	3.71	4.46	30	0.000*

** – significant difference between Groups I and III.
*** – significant difference between Groups I and II and between Groups I and III.
º – significant difference between Groups II and III.
R – right side. L – left side.

** – istotna różnica między grupą I a III.
*** – istotna różnica między grupą I a II oraz grupą I a III.
º – istotna różnica między grupą II a III.
R – prawa strona. L – lewa strona.

Table 2. Analysis of the differences between groups in the trigeminal sensory evoked potentials (TSEP) examination (using the ANOVA test and in groups with non-heterogeneous "p" variance, the non-parametric Kruskal-Wallis test)

Tabela 2. Analiza międzygrupowa czuciowych potencjałów wywołanych z nerwu trójdzielnego (TSEP) (test analizy wariancji ANOVA lub dla grup o niejednorodnej wariancji „p*" test nieparametryczny sumy rang Kruskala-Wallisa)

	I						II						III						p
	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	X	M	SD	MIN	MAX	N	
N1–R	11.2*	11.2	1	9.2	13	33	10	10.2	2.1	1.6	12.4	20	10.6	10.7	0.6	9.2	11.7	30	0.006
P1-R	21.1**	21.2	1.5	16	23.4	33	19.8	19.5	1.6	16.4	23.2	20	19.9	20	0.9	18.4	21.8	30	0.000
N2-R	32.7**	32.6	2.2	27.8	38.8	33	31.1	31.8	2.1	27	34.6	20	31.4	31.4	1.7	27	34.2	30	0.005
P2-R	42.5**	43.2	2.9	32.6	46.4	33	41.2°	40.1	2.9	36	46.4	20	39.1	39.4	1.1	35.6	40.8	30	0.000*
N1/P1-R	2.72	2	2.2	0.68	10.02	33	2.71	2.72	1.26	0.56	5.9	20	3.35	3.43	0.84	1.95	4.8	30	0.233
N2/P2-R	2.63	2.05	2.01	0.8	10.6	33	2.45	2.02	1.21	0.8	4.65	20	3.07	3	0.9	1.6	5.9	30	0.015*
N1-L	11.4*	11.4	1.2	9.6	15.6	33	9.71	10.8	3.2	0	12.2	20	10.7	10.7	0.7	9.4	11.7	30	0.039*
P1-L	21.1*	21	1.3	17.6	24	33	19.1	19.6	4.7	0	23.4	20	19.8	19.8	0.8	18.6	21.6	30	0.012
N2-L	32.7***	32.4	1.6	28.8	37.6	33	30.7	30.4	1.8	27.4	34	20	31.3	31.5	1.7	27.2	34.2	30	0.000
P2-L	43.1**	43.2	2.2	38.4	47.8	33	41.1°	40.1	3	35.4	45.8	20	39	39.3	1.1	35.8	40.4	30	0.000*
N1/P1-L	2.72**	2.05	2.14	1.03	10.08	33	2.12	2.01	1.49	0	5.45	20	3.42	3.12	1.08	1.8	5.5	30	0.028
N2/P2-L	2.52	2.01	2.03	0.57	11	33	2.77	2.45	1.17	1.36	6.6	20	3.03	3.15	0.94	1.5	5.2	30	0.413

* – significant difference between Groups I and II.
** – significant difference between Groups I and III.
*** – significant difference between Groups I and II and between Groups I and III.
° – significant difference between Groups II and III.
R – right side. L – left side.

* – istotna różnica między grupą I a II.
** – istotna różnica między grupą I a III.
*** – istotna różnica między grupą I a II oraz grupą I a III.
° – istotna różnica między grupą II a III.
R – prawa strona. L – lewa strona.

tistical significance were II, IV and III–V on the left side.

In the BAEP study, abnormalities were found in 18 patients with BMS; in most cases (12) they were bilateral. Central disturbances occurred in 11 patients, mixed (central and peripheral) disturbances in 3 patients and peripheral disturbances in 4. Pathological BAEP recordings were observed in 6 patients with PD; all these cases were bilateral and central disturbances. The patients with BMS had the most prolonged latency and interlatency, significantly different from the control group and average, and higher in every case than in Group II.

The findings of the analysis of intergroup differences in the TSEP examinations are presented in Table 2. The only differences that had no statistical significance were N1/P1 on the right and N2/P2 on the left side.

In the TSEP studies among the patients with BMS, abnormal findings were reported in 18 cases; in most cases (15) they were bilateral. There was prolonged latency of peaks – especially P2. In 8 cases there was a pathological difference in amplitudes between the sides (above 50%). In the TSEP studies in the PD group, 8 patients had prolonged latency of peaks (bilateral in 3 cases) and 4 cases had pathological asymmetry of amplitudes between the sides. In the group of patients with BMS, abnormalities were found in the form of significantly longer latencies of N1, P1, N2, P2 and diminution of amplitude of N1/P1 and N2/P2 compared with the control group. Significantly prolonged latency of those peaks was also recorded in the patients with Parkinson's disease.

Disorders of the blink reflex were reported in 16 patients with BMS and 15 with Parkinson's disease. In Group I and II there was a significantly higher incidence of abnormal BR compared to the control group (Table 3).

In the group of patients with BMS, the correlation between neurological variables and the duration (in years) of the burning sensation (Pearson's correlation) was investigated. The only statistically significant correlation found was a negative correlation between the duration of the burning history and parameter IV on the left-side BAEP ($p = 0.005$). An analysis of the codependency between neurological parameters and pain intensity on the VAS scale (Spearmann's correlation) was performed. A statistically significant negative correlation was found between pain intensity and parameters III on the right-side BAEP ($p = 0.007$), I–V on the right-side BAEP ($p = 0.041$) and I–V on the left-side BAEP ($p = 0.003$). The covariance between the studied neurological variances and polypragmasy (Spearmann's correlation) was also investigated. A significant positive correlation was found between polypragmasy and parameters IV on the right-side BAEP ($p = 0.026$) and III on the left-side BAEP ($p = 0.034$). No significant correlation was found between the type of burning according to the Lamey and Lewis criteria and variances in BAEPs and TSEPs.

Discussion

When analyzing 16 variances describing brainstem auditory evoked potentials, pathologies were found mostly in the patients with BMS (significant differences in 13 variables compared with the controls and in 5 variables compared with the patients with Parkinson's disease). Significant differences (prolonged latency and interlatency of peaks) were found in 7 variables between the patients with Parkinson's disease and the control group. In most of the BMS patients, the BAEP abnormalities were bilateral, usually central; less often peripheral or mixed. In the patients with Parkinson's disease the pathological BAEP recordings were in all cases bilateral and central. Twelve BAEP parameters had the greatest average prolonged latency and interlatency in the BMS patients. Twelve of the variables analyzed for somatosensory potentials induced with stimulation of the trigeminal nerve were abnormal in the patients with BMS (6 variables were significantly different compared to the controls, and 4 variables were significantly different from the PD group). Significant differences between the PD group and the control group were observed in the mean values of two TSEP parameters. In most of the BMS patients, the pathological recordings of TSEP were bilateral; and in the PD patients they were bilateral in all cases. For 8 TSEP parameters the mean rise in the amplitude and prolongation of latency was the greatest in the patients with BMS.

Table 3. Analysis of the frequency of normal and abnormal blink reflexes (BR) in the studied groups

Tabela 3. Analiza częstości prawidłowego i nieprawidłowego odruchu mrugania – BR dla badanych grup

Group (Grupa)	I		II		III		p χ^2
	abnormal	normal	abnormal	normal	abnormal	normal	
BR	16	17	15	5	0	30	$0.000^2 = 31.8$

A lack of habituation of the blink reflex was reported in 16/33 of the BMS patients, and in 15/20 of the PD patients. Abnormal habituation of the blink reflex has been demonstrated in some BMS patients by other authors as well: Jaaskelainen et al. [18] in 4 out of 11 patients, and Forssell et al [8] in 10 out of 52 patients. In Parkinson's disease the most frequent pathognostic findings are prolonged latency and restricted habituation of the blink reflex R2 response [21]. The findings of the analysis of the electrophysiological studies (BAEP, TSEP and BR) in patients with BMS indicate possible central (in most cases) or peripheral damage of the trigeminal nerve. The significant similarities in the BAEP, TSEP and BR findings in patients with BMS and patients with Parkinson's disease indicate possible sensory-information receiving and processing dysfunction on various levels of the central nervous system, particularly the nigrostriatal system.

Positron emission tomography examination confirms impairment of the dopaminergic system in patients with BMS [9]. The scale of abnormalities of the evoked potentials in BAEP and TSEP indicates that in BMS the damage to nervous system structures can be significant.

Surprisingly, the current study demonstrated only two significant correlations between the clinical indications of BMS (the type of burning, the duration of the pain, the VAS scale) and BAEP and TSEP parameters: First, there was a significant negative correlation between latency values and interlatency III on the right, I–V on the right and I–V on the left side in BAEP and pain intensity on the VAS scale; second, there was a significant positive correlation between latency time IV on the right and III on the left side in BAEP and polypragmasy. When interpreting these correlations with all due caution for a two-factor model, in the case of the first correlation mentioned, it can be surmised that the more prolonged latency and interlatency of selected BAEP peaks are, the less intense the burning sensation is. As for the second correlation noted, it is possible that polypragmasy is associated with the prolongation of latency of selected BAEP peaks, which may indicate neurontoxicity of drug-induced interactions in elderly people. These assumptions need to be confirmed by targeted studies.

To conclude, it can be said that the abnormalities found in the electroneurophysiological studies of patients with burning mouth syndrome are similar to those found in Parkinson's disease. Therefore, it appears that pharmacotherapy for BMS might focus on stimulating the dopaminergic system.

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