NÖRO PSİKİYATRİ ARŞİVİ

Research Article

Arch Neuropsychiatr 2015; 52: 151-6 • DOI: 10.5152/npa.2015.7601

Sympathetic Skin Responses from the Neck Area in Patients with Unilateral Migraine

Bektaş KORKMAZ[1], Serpil YILDIZ[2], Nebil YILDIZ[2]

[1]Department of Neurology, İstanbul University Cerrahpaşa Faculty of Medicine, İstanbul, Turkey
[2]Department of Neurology, Abant İzzet Baysal University Faculty of Medicine, Bolu, Turkey

ABSTRACT

Introduction: In this study, in patients with unilateral migraine headache and in normal controls, it was aimed to assess the sympathetic function during attack, post attack, and interval periods and to compare these findings by recording sympathetic skin responses from the neck area, which was not studied before.

Methods: A total of 37 unilateral patients with migraine (30 women, seven men) who fulfilled the criteria of International Headache Society (2004) were recruited from our outpatient clinic. The control group consisted of 21 healthy individuals (16 women, five men) who are employees or students of our Medical Faculty. Mean latency and maximum amplitude values of sympathetic skin responses obtained from neck areas of the patients during attack, post attack, and interval periods were calculated. We compared the mean latency and the maximum amplitude values of the symptomatic side with the data of the asymptomatic side and with the data of the control group. We also compared the responses of the patients with right-sided headache with the responses of the patients with left-sided headache. All statistical analyses were performed using SPSS.

Results: On the neck area, we observed sympathetic hypo-function in the attack and interval periods and a relative hyper-function in the post attack period bilaterally, regardless of the symptomatic side.

Conclusion: These findings suggest that there is ongoing bilateral sympathetic hypo-function in the neck area and there occurs a temporary increase in the function of sympathetic sudomotor activity in the recovery period of headaches.

Keywords: Primary headache, migraine, autonomic dysfunction, sympathetic skin response, neck

INTRODUCTION

A migraine is the most common type of headache in the society and has a negative effect on the daily activities and emotional status of those who have it. In the light of information obtained recently, it is considered that migraine is a primary neuronal process (1,2). Neuronal depolarization composed of internal or external triggers in a highly excitable cerebral cortex and the spreading depression wave lead to aura in migraine and activation of the trigeminovascular system (3).

The autonomic dysfunction in patients with migraine has been demonstrated in many studies and it has been reported that migraine is associated with sympathetic hypo-function (4,5,6,7,8,9,10), sympathetic hyper-function (11,12), sympathetic and parasympathetic hyper-function (13), or sympathetic irregularity (14,15). In order to investigate the sympathetic dysfunction in patients with migraines, various processes such as the examination of the sympathetic skin responses from the hand (SSR), superior ophthalmic nerve stimulation with soap or salt eye drops, of the resulting trigeminovascular response, pupil functions, thermographic asymmetry, frontal blood flow measurement, heart rate variables, etc., have been performed. The SSRs obtained from the hand and foot have been broadly examined. There are only a few studies on the functional differences of the sympathetic nerve system in the facial and nape regions (16,17,18,19). None of these studies are about the sympathetic activity of the neck region in a migraine headache (MH).

In this study, we aimed to examine the differences in SSRs of symptomatic and asymptomatic neck regions during and after migraine attacks and in intervals of the attacks by recording bilateral neck SSRs (N-SSR) in healthy individuals (controls) and patients with unilateral migraines.

METHODS

In this study, the N-SSRs of the bilateral neck regions in the control group and patients with unilateral migraines was recorded. We aimed to examine the differences in N-SSRs during and after a migraine attack and in the intervals of attacks in patients with unilateral migraine as compared with the control group.

Correspondence Address: Dr. Bektaş Korkmaz, Department of Neurology, İstanbul University Cerrahpaşa Faculty of Medicine, İstanbul, Turkey Phone: +90 212 414 31 47 E-mail: bektaskorkmaz@hotmail.com

Received: 31.12.2013 **Accepted:** 24.03.2014

In this study, 58 patients were in compliance with the migraine criteria of International Headache Association 2004. All the patients included in the study stated that most of their headaches were repeated on the same side. Twenty-one patients who had unilateral headaches but had a similar repeating headache on the other side were excluded from the study. In total, 37 patients (30 women and seven men) were evaluated. Routine biochemistry, cranial computed tomography, electromyography (EMG), and neurologic examinations were normal. They had no other known diseases other than migraines. Patients diagnosed with any other diseases as a result of the examinations were excluded from the study.

The recording times of the patients included in the study were categorized as follows: during attack, after attack, and during intervals between attacks. The period during the attack was considered as the period with headache, the period after the attack was considered as 3 days after the headache finished, and the period during intervals between attacks was considered as at least 1 week after the most recent headache. None of the patients received any treatment other than migraine treatment and no patient had any attack preventing treatment before and after the recordings of the periods with attacks. No patients used drugs with long-lasting effects and that were effective on the autonomic nervous system. Patients receiving treatment after recording the attacks were treated with a nonsteroid anti-inflammatory drug (Naproxen sodium 550 mg p.o. 1×1) with the same dose and name. Patients were asked to rate the severity of their headaches on a numeric scale between 0 and 10 during the attacks. Before recording these ratings, patients were informed of the gradation of the scale, in which 0 indicated no headache and 10 indicated unbearable headache.

Twenty-one fully healthy individuals from the faculty staff and students who did not experience any headache such as migraines, did not have any known diseases, did not undergo any drug treatment, and did not demonstrate polyneuropathy on EMG were classified into the control group.

Approval of the Ethics Committee of İzzet Baysal Medical Faculty and informed consent of all study participants were obtained. The examinations were performed via Nicolet Viking IV channel electromyography device. SSR recordings were performed via standard Ag–AgCl electrodes (10 mm in diameter NihonKohden, NM-312S). The recording method was similar to that of Yıldız et al. (19). Our frequency interval was adjusted between 0.2 and 100 Hz. The time window for recording was 5 s and the screen sensibility was 500 μV/division.

In one day, recordings were performed between 09:00 and 17:00. The room temperature where the recordings were performed was 24±1°C. In the well-ventilated, normally illuminated room, examinations were performed when the patient lied on his back on the EMG stretcher and had a skin temperature of over 32°C. During the examination, the participants were asked to keep their eyes open, not to inspire deeply, not to cough, and not to speak or move their heads during the process.

The recording points are presented in Figure 1. Each participant underwent bilateral neck examinations, bilateral as right and left sides. After the first response was obtained, 10–15 stimulants in total were provided. Depending on the response pattern, each participant was group as either "bilateral response," "unilateral response," or "no response." Those giving bilateral responses were those with SSR on each side, those giving unilateral responses were those with SSR on one side, and those not giving any responses were those with no responses. Responses of those with spontaneous responses were recorded in 20 s. In those with no responses or with unilateral responses, these data were verified through SSR recordings by providing audio stimulants at the end of each session and having them take deep inspiration.

The electrical stimulant (0.2-ms time pulse square and with a 25-mA severity) was applied on the right median nerve of the wrist. The stimulant was applied irregularly, with intervals of at least 20 s. In the participants who did not give any response, the power of the stimulant was increased in increments of 10 mA until a response was seen and took a reliable form (a maximum electrical stimulant of 100 mA was applied). The participants receiving 10 consecutive stimulants at 100-mA power and not giving any responses on either side were considered as participants without responses.

If the amplitude was over 50 μV and had similar latencies with at least two consecutive stimulants, SSR was considered to have "occurred." In each response recorded, the peak-to-peak amplitude and beginning latency were measured. Maximum amplitude and average latency values were calculated for right and left sides from the first 10 response pairs.

Statistical Analysis

In the statistical analysis, the symptomatic and asymptomatic side responses of the patient group and the right and left sides of the control group were compared in terms of average latency and maximum amplitudes via two related sample tests (Wilcoxon marked sequence test). In the comparison tests, data of only patients with bilateral responses were used. The correlation constants between the symptomatic and asymptomatic sides of patients and between the right and left sides of the control group were calculated (Spearman test). The data of the control group and the patients were separately compared in terms of the period during the attack, after the attack, and during intervals between attacks using Mann–Whitney U test. A P-value of <0.05 was considered statistically significant.

RESULTS

Thirteen of 37 patients complied with the criteria of migraine with aura and 24 complied with the criteria of migraine without aura. The average age of the patients was 31.06±9.02 (between 18 and 50) years. The control group comprised 16 female and five male participants. The average age of the control group was 29.58±7.63 (between 19 and 45) years.

The illness period, side of the headache, frequency of the headache, the term of the headache during examination, and the headache scores during the attack recordings are presented in Table 1 for each patient. Fifteen patients could be examined in two or three different periods.

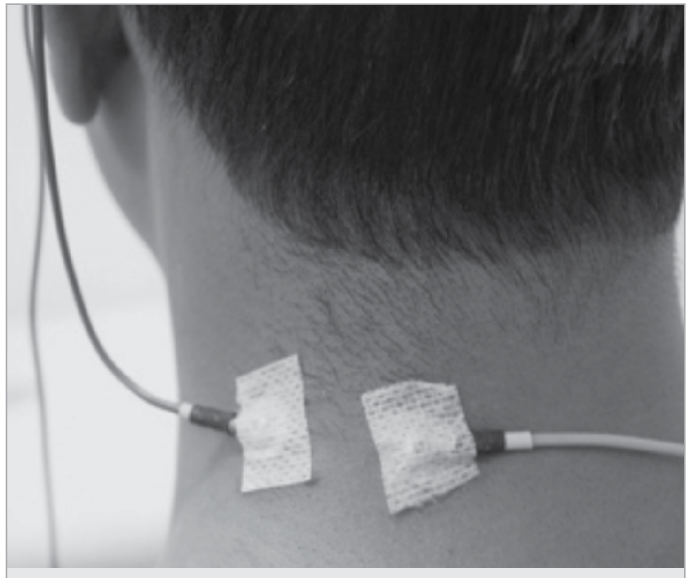

Figure 1. N-SSR recording point
N-SSR: neck sympathetic skin responses from the hand

Table 1. The illness period, side of the headache, frequency of the headache, the term of the headache during examination, and the headache scores during recording of the attack for patients whose N-SSRs were examined

Patient no.	Side	Headache score	Period (year)	Frequency (per month)	A	PA	I
9*;13*;	L;R;	7;7;	5;1/6;	5;3;	+		
5;8;10;12;	R;R;R;L;		20;2.5;2;2;	3;5;4;2;		+	
18;22;27;33;	R;R;L;R;		2;3;1;5;	5;4;5;4;			
34;35	L;R		2;10	3;2			
2;3;17;19;20	L;R;R;L;L		2;1;8.5;3;5	2;5;6;1;3			+
23;28;31,37*	L;R;L;R		2;4.5;7;3	4;2;1;3			
11*; 14**	L;R	5;6	15;15	1;2	+	+	+
4;21**;25;29;36**	R;L;L;R; R	6;9;7;8; 8	9;15;2;1;10	3;4;4;5; 3	+	+	
1;6;7**;15;16**;	R;R;L;L;R;	5;6;10;8;7;	28;2,5;13;20;3;	3;6;4;4;1;	+		+
24**;26	L; L	8; 8	4;20	3;5			

*with unilateral responses; **with no responses. Those not marked have bilateral responses.
The numbers written in bold indicate the patients who were examined within the same headache cycle.
A: attack; PA: after attack; I: interval

Table 2. N-SSRs of the control group

Controls	Right N-SSR	Left N-SSR
Latency (ms)		
Mean±SD, SE	1082±115, 26	1085±99, 22
Median	1058	1075
Interval	926-1380	891-1400
Statistic	Wilcoxon marked sequence test, p=0.614	
Correlation	Spearman's rho: 0.570, p= 0.009	
R+L Mean±SD, SE	1084±106, 17	
Amplitude (μV)		
Mean±SD, SE	772±525, 117	730±480, 107
Median	651	641
Interval	160-1800	210-2400
Statistic	Wilcoxon marked sequence test, p=0.332	
Correlation	Spearman's rho: 0.725, p=0.000	
R+L Mean±SD, SE	751±497, 79	

N-SSR: Sympathetic skin responses in the neck region; R+L: combined data of right and left sides; SD: standard deviation; SE: standard error

On the other hand, no patient demonstrated the responses of the same headache cycle. Please see Table 1 for patients whose recordings could be taken in the same attack cycle. N-SSRs during the attack were observed in 16 patients in total, all of whom had medium and severe headaches during recording (=5 or >5). The average headache score was 7.3±1.5 (between 5 and 10). N-SSRs after the attack were recorded for 17 patients. B-RRS recordings were performed for 18 patients during the period between attacks.

Recordings from the neck could not be made in one of the 21 healthy individuals (Figure 2). There was no significant difference between the right and left side of the remaining 20 individuals in terms of maximum amplitudes and average latencies of N-SSRs. Thus, the data of the right and left sides could be combined. The average latencies, maximum amplitudes, and correlation values between the sides obtained when two sides were evaluated together are presented in Table 2.

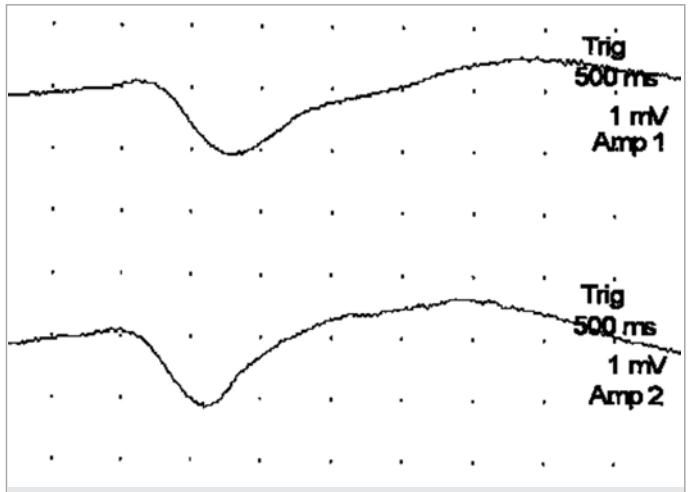

Figure 2. Neck SSRs of a normal individual
SSR: sympathetic skin responses from the hand

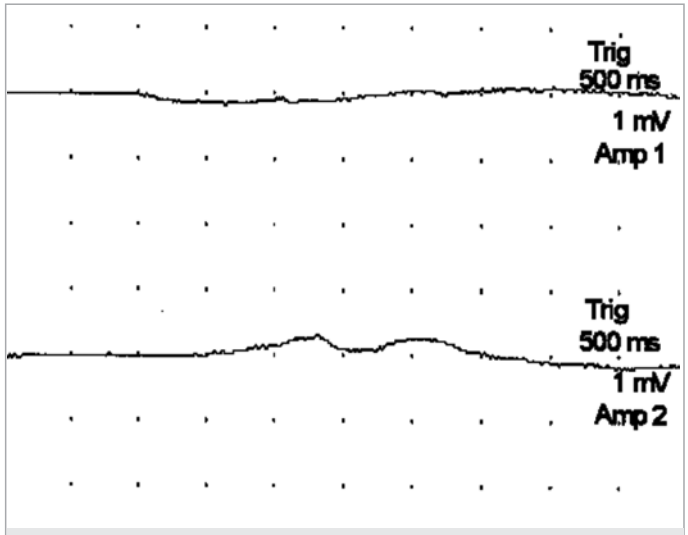

Figure 3. Neck SSRs during attack in a patient with unilateral migraine
SSR: sympathetic skin responses from the hand

N-SSRs during the Attack in Patients with Unilateral Migraine

During the attack period, the N-SSRs of 16 patients were examined (Figure 3). During this period, there were six patients without responses. Two of the remaining 10 patients had unilateral responses. The side where no response could be obtained in those giving unilateral responses was the asymptomatic side.

The average latencies, maximum amplitudes, and correlation values between the sides obtained during attack are presented in Table 3.

In patients with bilateral responses, there was no significant difference between the symptomatic side and the asymptomatic side in terms of average latencies and maximum amplitudes.

The maximum the N-SSR amplitudes of the symptomatic and asymptomatic sides were significantly lower compared with those of the

Table 3. Sympathetic skin responses in the neck regions of unilateral patients with migraine in the period during, after, and between attacks

Period	*Attack (n=10)		*After attack (n=17)		*Interval (n=18)	
Side	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic	Symptomatic	Asymptomatic
Latency (ms)**						
Mean±SD, SE	1169±138, 44	1179±106, 37	1095±107, 27	1115±102, 25	1117±96, 23	1089±116, 28
Median	1199	1219	1085	1106	1131	1111
Interval	931-1330	1010-1282	877-1300	910-1263	911-1257	902-1280
Statistic**	Wilcoxon: 0.123		0.393		0.339	
Correlation	Spearman's rho: 0.619, p=0.102		Spearman's rho: 0.764, p=0.001		Spearman's rho: 0.252, p=0.347	
Amplitude (μV)*						
Mean±SD, SE	412±379, 120	420±490, 154	855±787, 191	781±706, 171	415±386, 91	449±295, 70
Median	286	220	527	549	288	371
Interval	88-1180	0-1340	110-2880	0-2695	0-1190	0-940
Statistic**	0.445		0.687		0.913	
Correlation	Spearman's rho: 0.857, p=0.007		Spearman's rho: 0.779, p=0.000		Spearman's rho: 0.638, p=0.008	

*The data of the patients bilaterally with no responses were not included. **Only the data of patients with bilateral responses were included. SD: standard deviation; SE: standard error

control group. When there were no significant differences between average latency values of the symptomatic side and of the N-SSRs of the control group, the average latency values of the asymptomatic side were significantly longer compared with those of the control group.

N-SSRs in the Period after the Attack in Patients with Unilateral Migraine

Seventeen patients in the period after attack were examined in terms of N-SSR. There was no patient without a response. The side without any response in a patient with a unilateral response was the asymptomatic side.

The average latencies, maximum amplitudes, and correlation values between the sides obtained after attack are presented in Table 3.

In patients with bilateral responses, there was no significant difference between the symptomatic side and the asymptomatic side in terms of average latencies and maximum amplitudes.

There was no significant difference when the maximum amplitude and average latencies in the symptomatic and asymptomatic sides were compared with those of the N-SSRs of the control group.

N-SSRs in the Interval Period without Headache in Patients with Unilateral Migraine

Eighteen patients in this period were examined in terms of N-SSRs. There was no patient without a response. There were two patients with a unilateral response. There was no response in the symptomatic side in one of them and in the asymptomatic side in the other.

The average latencies, maximum amplitudes, and correlation values between the sides obtained during the interval without headache are presented in Table 3.

There was no significant difference between the symptomatic side and the asymptomatic side in terms of average latencies and maximum amplitudes in the interval period without headache.

The maximum amplitudes of the symptomatic and asymptomatic sides during the interval without headache were significantly lower compared with those of the N-SSRs of the control group. There was no significant difference between the average latencies of the symptomatic and asymptomatic sides and those of the N-SSRs of the control group.

Comparison of the Neck Responses of Patients with Migraine having Headaches on the Left or Right Sides in the Periods during, after, and between Attacks

The average latencies, maximum amplitudes, and results of the comparison of two groups pertaining to the SSRs recorded in the neck regions of the patients with migraine with headaches on the right and left sides in the periods during, after, and between attacks are presented in Table 4.

When the maximum amplitudes of the SSRs obtained from the neck regions of the patients with migraine with headaches on the right and left sides during the attack were compared, the maximum amplitudes of those whose left sides were symptomatic were lower, whereas there was no significant difference between their average latencies.

When the maximum amplitudes of the SSRs obtained from the neck regions of the patients with migraine with headaches on the right and left sides after the attack were compared, there was no significant difference. However, the average latencies of those whose right sides were symptomatic were significantly longer.

There was no significant difference in the maximum amplitudes and average latencies of the SSRs recorded in the neck regions between those with migraine with headaches on the right and left sides.

DISCUSSION

In summary, it was observed that the findings obtained from N-SSRs were in compliance with the sympathetic hypo-function during the headache, particularly in the period when the headache was medium or severe. It was detected that a relatively hyper function developed within 3 days immediately after the headache in the region where hypo-function was observed before had disappeared and there was no significant difference any more compared with normal cases; again, hypo-function was observed in this region in the period at least 1 week after the headache.

Table 4. Total values of two groups pertaining to the SSRs recorded in the neck regions of patients with headaches on the right and the left sides in the period during, after, and between attacks

	Attack		After attack		Interval	
	Headache on right side	Headache on left side	Headache on right side	Headache on left side	Headache on right side	Headache on left side
Latency*						
Mean±SD, SE	1148±124, 41	1199±120, 40	1139±76, 17	1051±120, 35	1108±126, 33	1099±91, 21
Interval	931-1290	1000-1330	1023-1300	877-1290	913-1280	902-1257
Statistic	Mann–Whitney U test: p=0.297		Mann–Whitney U test: p=0.005		Mann–Whitney U test: p=0.891	
Amplitude**						
Mean±SD, SE	424±495, 124	96±119, 30	953±840, 179	571±423, 122	409±344, 86	451±344, 77
Median	193	84	595	417	349	355
Interval	0-1340	0-432	0-2880	190-1540	0-1150	0-1190
Statistic	Mann–Whitney U test: p=0.001		Mann–Whitney U test: p=0.027		Mann–Whitney U test: p=0.648	

*Data of one patient with no unilateral response was not included. **Data of patients with unilateral responses were included. In a patient in the post-attack group with a headache on the right side, no response could be obtained of the asymptomatic side. No unilateral response could be obtained of the asymptomatic side of one patient with a headache on the right side during the interval and of the symptomatic side of one patient with a headache on the left side. SD: standard deviation; SE: standard error

It has been reported that the amplitudes of the SSRs obtained from the homologous regions in the two sides of the body must be relatively similar because the anatomic and physiological factors in these homologous regions are similar (19,20,21,22,23). Additionally, it has been argued in many case reports and studies that sudomotor innervation can be undertaken by parasympathetic fibers when the sympathetic fibers recessed (20,24). However, as a common view, in diseases with failure of the sympathetic nervous system or, more generally, the autonomous nervous system, for example, in diseases such as pure autonomic failure, multisystem atrophy etc., it has been reported that sympathetic hypo-function supports the inability to obtain the response in skin responses from the hands and feet (5,25,26,27).

In this study, six of the patients undergoing SSR recordings from their neck regions during attacks were bilaterally without responses (6/16, 38%). Two of the remaining 10 patients had unilateral responses (2/16, 13%). Although there was one patient with a unilateral response after the attack and two patients with unilateral responses during the attack, there were no participants with unilateral responses in the control group. However, there was one participant without a response in the control group. It was considered that the patients from whom we could not obtain N-SSRs may have sympathetic hypo-function. In certain previous studies, it has been asserted that the sympathetic hypo-function in the facial blood vessel and pupil in unilateral migraine could be bilateral (8,28,29). Similarly, in this study, the changes in the neck responses observed in both the symptomatic and asymptomatic sides in patients with a unilateral response were in line with the presence of bilateral pathology.

Drummond demonstrated that the pupil diameter was smaller during headache in patients with unilateral migraines (30). The same researcher stated in different studies that there was unilateral sympathetic hypo-function during migraine attacks in patients with unilateral migraines (8,28). In this study, although there was no significant difference between the symptomatic and asymptomatic sides in patients with bilateral responses, it is interesting that the side where no response could be obtained was the asymptomatic side in all patients, except for one.

It was detected that N-SSRs reached normal values, demonstrating a relative hyper-function in the first 3 days after the headache bilaterally. The fact that the activity of the trigeminovascular system, which is active during headaches, decreased after the headache occurred may have led to the increase in symptomatic activity after the headache occurred, which was found to be hypoactive during the headache. It is possible that the increase in the symptomatic output prevents the trigeminovascular activation, resulting in the elimination of the headache and in recovery. Till date, the period just after the headache has stopped has not been examined separately, as in this study. It was considered that the conflicting results of the activity of the sympathetic system in migraines may result from the fact that the periods after attacks and during the interval between attacks were not separated, and this may be an important reason for the observation of hyper-function in some of the studies.

After separation from the superior cervical ganglion, postganglionic and sympathetic fibers are separated into two groups together with carotis communis bifurcation. Certain fibers proceed with carotis artery, whereas others proceed with external carotis artery. Although the fibers proceeding together with the internal carotis artery innervate the medial part of the forehead and the eye, those proceeding with the external carotis artery are responsible for the innervation of the lateral and upper parts of the forehead and the other parts of the face (31). There are many studies examining the pupillary function in migraine and asserting that there is sympathetic hypo-function (4,7,8,28,30,32). Drummond asserted that the sympathetic dysfunction observed in migraine results from the compression of the sympathetic plexus around the internal carotis artery in the internal carotid channel (8). In this study, because the neck region where SSRs were examined are innervated not by the internal carotis artery but by the external carotis artery or its branches (postauricular and occipital branches), it was considered that only the fibers proceeding with the internal carotis artery were not kept.

The differences in the SSRs of those whose headaches were on the right side and those whose headaches were on the left side were examined. When only the responses of the patients with bilateral responses were compared for the period during the attack, it was detected that those having headaches on their left sides had significantly smaller responses. In the periods after attacks and during intervals between attacks, there was a significant amplitude difference between those having headaches on their right sides and those having headaches on their left sides. Avnon et al. (33) argued that the autonomic control of the brain is asymmetric, with

the left hemisphere mostly undertaking the parasympathetic function and the right hemisphere mostly undertaking the sympathetic function. The present study demonstrated that a more evident sympathetic hypo-function occurred during the headache in those having headaches on their left sides and made us consider that right- and left-sided migraines must be investigated separately.

Again, in a study conducted in our clinic, the SSRs from the frontal regions (F-SSR) of patients with migraine were recorded (34). According to this study, the symptomatic and asymptomatic sides behave differently in the frontal region; however, similar to N-SSRs, hypo-activity is observed during an attack in F-SSRs as well. In the period after the headache, the sudomotor hyper-function is more dominant on the symptomatic side. Our findings demonstrate that the N- and F-SSRs both had similar and different features that complete each other. As a result, the sympathetic sudomotor activity of the neck region is bilaterally hypo-active during a migraine headache. In the period after the attack, the neck region becomes bilaterally more active compared with that in the interval period. In the interval period, the neck is again bilaterally hypo-active.

It has been considered that many cortical and subcortical regions are affected and the periods with and without headaches must be clearly separated in the clinical studies of this disease.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Lewis DW. Toward the definition of childhood migraine, Curr Opin Pediatr 2004; 16:628-636. [CrossRef]
2. Silberstein S. Migraine. Lancet 2004; 31:381-391. [CrossRef]
3. Meyer JS, Hata T, Imai A, Zetuksky WJ. Migren and intcranial swelling. Lancet 1985; 7:1308-1309.
4. Gotoh F, Komatsumoto S, Araki N, Gomi S. Noradrenergic nervous activity in migraine. Arch Neurol 1984; 41:951-955. [CrossRef]
5. Peroutka SJ. Migraine: A chronic sympathetic nervous system disorder. Headache 2004; 44:53-64. [CrossRef]
6. Boiardi A, Munari L, Milanesi I, Paggetca C, Lamperti E, Bussone G. Impaired cardiovascular reflexes in cluster headche and migraine patients. Headache 1988; 28:417-422. [CrossRef]
7. Battistella PA, Ruffili R, Zacchello F. Pupillary adrenergic sensitivity and idiopathic headache in pediatric patients. Headache 1989; 29:163-166. [CrossRef]
8. Drummond PD. Disturbances in ocular sympathetic function and facial blood flow in unilateral migraine headache. J Neurol Neurosurg Psychiatry 1990; 53:121-125. [CrossRef]
9. De Marinis M. Pupillary abnormalities due to sympathetic dysfunction in different forms of idiopathic headache. Clin Auton Res 1994; 4:331-338. [CrossRef]
10. Mylius V, Braune HJ, Schepelmann K. Dysfunction of the pupillary light reflex following migraine headache. Clin Auton Res 2003; 13:16-21.[CrossRef]
11. Micieli G, Tassorelli C, Magri M, Sandrini G, Cavallini A, Nappi G. Vegetative imbalance in migraine. A dynamic TV pupillometric evaluation. Funct Neurol 1989; 4:105-111.
12. Cortelli P, Pierangeli G, Parchi P, Contin M, Baruzzi A, Lugaresi E. Autonomic nervous system function in migraine without aura. Headache 1991; 31:457-462. [CrossRef]
13. Yakıncı C, Mungen B, Er H, Durmaz Y, Karabiber H. Autonomic nervous system function in childhood migraine. Pediatr Int 1999; 41:529-533. [CrossRef]
14. Appel S, Kuritzky A, Zahavi I, Zigelman M, Akselrod S. Evidence for instability of the autonomic nervous system in patients with migraine. Headache 1992; 32:10-17. [CrossRef]
15. Zigelman M, Kuritzky A, Appel S, Davidovitch S, Zahavi I, Hering R, Akselrod S. Propranolol in the prophylaxis of migraine-evaluation by spectral analysis of beat-tobeat heart rate fluctuations. Headache 1992; 32:169-174. [CrossRef]
16. Elie B, Guiheneuc P. Sympathetic skin response: normal results in different experimental conditions. Electroencephalogr Clin Neurophysiol 1990; 76:258-267. [CrossRef]
17. Nordin M. Sympathetic discharges in the human supraorbital nevre and their relation to sudo- and vasomotor responses. J Physiol 1990; 423:241-255. [CrossRef]
18. Chen HJ, Lu K, Liang CL. Transthoracic endoscopic T-2,3 sympathectomy for facial hyperhidrosis. Auton Neurosci 2001; 93:91-94. [CrossRef]
19. Yıldız SK, Türkoğlu ŞA, Yıldız N, Öztürk A, Töre F. Sympathetic skin responses of the face and neck evoked by electrical stimulation. Auton Neurosci 2007; 134:85-91. [CrossRef]
20. Bonanno PC, Palaia D, Rosenberg M, Casson P. Prophylaxis against Frey's syndrome in parotid surgery. Ann Plast Surg 2000; 4:498-501. [CrossRef]
21. Matsutani K, Tsuruoka M, Shinya A, Furuya R, Kawawa T. Stimulation of the locus coeruleus supresses trigeminal sensorimotor function in the rat. Brain Res Bull 2000; 53:827-832. [CrossRef]
22. Sasa M, Fujimoto S, Igarashi S, Munekiyo K, Takaori S. Microiontophoretic studies on noradrenergic inhibition from locus coeruleus of the spinal trigeminal nucleus neurons. J Pharmacol Exp Ther 1979; 210:311-315.
23. Gruzelier JH, Nicolaou T, Connolly JF, Peatfield RC, Davies PT, Clifford-Rose F. Laterality of pain in migraine distinguished by interictal rates of habituation of electrodermal responses to visual and auditory stimuli. J Neurol Neurosurg Psychiatry 1987; 50:416-422. [CrossRef]
24. Reich SG, Grill SE. Gustatory sareating: Frey syndrome. Neurolgy 2005; 65:E24. [CrossRef]
25. Braune HJ, Korchounov AM, Schipper HI. Autonomic dysfunction in Parkinson's disease assessed by sympathetic skin response: a prospective clinical and neurophysiological trial on 50 patients. Acta Neurol Scand 1987; 95:293-297. [CrossRef]
26. Fusina S, Conte S, Bertolasi L, Fincati E, Nardelli E, Bongiovanni LG. Sympathetic skin response asymmetry in early stage idiopathic Parkinson's disease. Clin Neurophysiol 1999; 110:358-366. [CrossRef]
27. Zakrzewska-Pniewska B, Jamrozik Z. Are electrophysiological autonomic tests useful in the assessment of dysautonomia in Parkinson's disease? Parkinsonism Relat Disord 2002; 9:179-183. [CrossRef]
28. Drummond PD. Effects of body heating and mental arithmetic on facial sweating and blood flow in unilateral migraine headache. Psychophysiology 1991; 28:172-176. [CrossRef]
29. Chronicle EP, Mulleners WM. Visual system dysfunction in migraine: a review of clinical and psychophysical findings. Cephalalgia 1996; 16:525-535. [CrossRef]
30. Drummond PD. Pupil diameter in migraine and tension headache. J Neurol Neurosurg Psychiatry 1987; 50:228-230. [CrossRef]
31. Morris JGL, Lee J, Lim CL. Facial sweating in Horner's syndrome. Brain 1984; 107:751-758. [CrossRef]
32. Fanciullacci M. Iris adrenergic impairment in idiopathic headache. Headache 1979; 19:8-13. [CrossRef]
33. Avnon Y, Nitzan M, Sprecher E, Rogowski Z, Yarnitsky D. Autonomic asymmetry in migraine: augmented parasympathetic activation in left unilateral migraineurs. Brain 2004; 127:2099-2108. [CrossRef]
34. Yıldız SK, Yıldız N, Korkmaz B, Altunrende B, Gezici AR, Alkoy S. Sympathetic skin responses from frontal region in migraine headache: A pilot study. Cephalalgia 2008; 28:696-704. [CrossRef]