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RESEARCH

Evaluation of sternocleidomastoid muscle cutaneous silent period in patients with cervical dystonia

Servikal distonili hastalarda sternokleidomastoid kasta kutanöz sessiz periyot değerlendirilmesi

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Abstract

Purpose: The aim of this study to examine the in changes inhibitory interneurons of the brainstem, which are likely to be involved in pathophysiology of cervical dystonia, using the cutaneous silent period(SP) method which is an inhibitor reflex.

Materials and Methods: SP latency and interval values were obtained from bilateral sternocleidomastoid (SCM) muscles of individuals in 20 patients with cervical dystonia and 25 healthy volunteers using superficial electrodes. SP parameters obtained were compared within and between groups. Furthermore, intra-individual correlation analysis was performed for SP parameters from bilateral SCMs of the individuals in both groups.

Results: The mean age of the patients participating in the study was 49.3 ± 14.7, and the control group was 48.0 ± 13.7. There was no statistically significant difference between the two groups in terms of age and gender. No significant difference was found in the comparison of SP parameters both within and between groups. There was a strong intra-individual correlation between SP latencies of bilateral SCMs of both patient and control groups. It was determined that the strongly positive intra-individual correlation between SP intervals also continued in the control group, whereas at patient group disappeared.

Conclusion: The loss of correlation between bilateral SCM SP durations in the dystonia group indicates that the abnormality in interneuron connections, which is thought to be present in dystonia, may actually be a loss of order. This "disorganization" may explain the discrepancies in the results of cutaneous SP studies in dystonia patients.

Keywords: Cervical dystonia; interneurons; electromyography

Öz

Amaç: Bu çalışmada servikal distoni patofizyolojisinde rol oynaması muhtemel beyin sapı inhibitör internöronlarındaki değişikliklerin inhibitör bir refleks olan kutanöz sessiz periyot(SP) yöntemi ile incelenmesi amaçlandı.

Gereç ve Yöntem: Servikal distonisi olan 20 hasta ve 25 sağlıklı gönüllünün bilateral sternokleidomastoid (SCM) kaslarından yüzeyel elektrotlar kullanılarak SP latans ve interval değerleri elde edildi. Elde edilen SP parametreleri gruplar içinde ve gruplar arasında karşılaştırıldı. Ayrıca her iki grupta karşılıklı SCM'lerinin SP parametrelerinin grup içi korelasyon analizi yapılmıştır.

Bulgular: Çalışmaya katılan hastaların yaş ortalaması 49,3 ± 14,7, kontrol grubunun 48,0 ± 13,7 olup iki grup arasında yaş ve cinsiyet açısından istatistiksel olarak anlamlı bir fark saptanmadı. Grup içi ve gruplar arası SP parametreleri arasında anlamlı fark saptanmadı. Hem hasta hem de kontrol gruplarında bilateral SCM'lerin SP latansları arasında güçlü bir birey içi korelasyon vardı. Birey içi SP intervalleri arasındaki güçlü pozitif korelasyonun kontrol grubunda da devam ettiği, hasta grupta ise kaybolduğu gözlendi.

Sonuç: Distoni hastalarında yapılmış olan kutanöz SP çalışmalarının sonuçları oldukça farklıdır. Distoni grubunda bilateral SCM SP süreleri arasındaki korelasyon kaybı, distonide var olduğu düşünülen aranöronlar arası bağlantılardaki anormalliğin aslında bir düzen kaybı olabileceğini göstermektedir. Bu "düzensizlik", distoni hastalarında kutanöz SP çalışmalarının sonuçlarındaki tutarsızlıkları açıklayabilir.

Anahtar kelimeler: Servikal distoni; aranöronlar; elektromiyografi

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INTRODUCTION

Dystonia is a movement disorder characterized by increased muscle contractions leading to abnormal posture and repetitive movements. Classification of dystonia is made according to etiology, age of onset and affected body part. Cervical dystonia (CD) is the most common type of adult-onset dystonia which involves neck and shoulder muscles, is repetitive, and causes clonic and tonic movements, leading to abnormal posture of the head[1]. Dystonia, like almost all other disorders, is a product of the interaction of genetic and environmental factors. One possible hypothesis is that there is a genetic loss of inhibitory interneurons in dystonia, and this deficiency is a substrate on which other factors can act to produce dystonia. Today, the studies to explain the underlying physiological base of dystonia have focused on three major mechanisms: loss of inhibition, impaired sensory-motor integration and abnormal plasticity[2].

There are various electrophysiological studies regarding pathophysiology of dystonia and one of these is cutaneous silent period (CuSP). CuSP is an inhibitory reflex characterized by a short pause in voluntary muscle activity following a potent stimulation of a cutaneous nerve[3]. Of this inhibitory reflex; the afferent arch is thought to be comprised of A-delta fibers, whereas the efferent arch is thought to be comprised of alpha-motor neurons[4]. It is thought to represent the inhibitory part of the sophisticated defense reflex of CuSP. It cooperates with excitatory withdrawal reflexes used to withdraw an extremity from a painful stimulus. CUSP Apart from dystonia, it has also been studied in various movement disorders such as parkinson, tremor, restless legs, paroxysmal kinesigenic dyskinesia and spinocerebellar ataxia[5-9]. Clinical interest in the CuSP stems from its potential usefulness for evaluating segments and components of sensory nerves that are not well assessed by standard electrodiagnostic methods[10].

Since there has been a noninvasive technique to evaluate interneurons of the brainstem and spinal cord according to the muscle of interest, CuSP has been studied in dystonia and various movement disorders until today. Since it is an inhibitory reflex, it may provide insight about the loss of inhibition, which is among the mechanisms defined in pathophysiology of dystonia. However, to the best of our knowledge, there are only two studies on CD which was carried out to examine CuSP, particularly in cervical muscles[11,12]. These studies, however, yielded different results. In this study, it was aimed to examine the in changes inhibitory interneurons of the brainstem, which are likely to be involved in pathophysiology of CD, using the CuSP method performed on sternocleidomastoid (SCM) muscle and contributing to the understanding of the pathophysiology of dystonia.

MATERIALS AND METHODS

Sample

This is a prospective case-control study. A total of 20 patients, 15 female and 5 male, diagnosed with primary cervical dystonia in Necmettin Erbakan University Meram Medical Faculty Movement Disorders Outpatient Clinic, and healthy 25 volunteers without polyneuropathy, radiculopathy, or neurodegenerative disease were included and then prospectively evaluated. Acquired dystonia usually arises from specific underlying conditions, such as perinatal brain injury or exposure to dopamine receptor-blocking drugs (eg, acute dystonic reactions, tardive dystonia). The presence of neurologic abnormalities other than dystonia may provide the clue to the cause of acquired dystonia[13]. Therefore all patients underwent etiological evaluations based upon medical history, laboratory work-up and craniocervical magnetic resonance imaging, and 5 patients who were found to have secondary dystonia were excluded from the study. In electrophysiological evaluation of the SCM muscle, those with unilateral spontaneous discharges were included in the study, whereas 9 patients who were found to have marked dystonia in both SCM muscles were excluded from the study. Thus, the study was started with 34 CD patients, 14 of which were excluded from the study and continued with 20 patients.

Patients who received botulinum toxin injections were performed electromyographic (EMG) studies at least 4 months after the last injection, it was confirmed that none of the patients had received drugs used for treatment of dystonia, including baclofen, benzodiazepines, anticholinergics, etc, within 2 weeks before the procedure. The control group was comprised of age- and gender-matched healthy volunteers. The procedure was applied to all patients by the same neurologist in the Meram medical faculty electrophysıology laboratory. The medical information was recorded anonymously by giving numbers to participants to protect their

personal data. The study was approved with Necmettin Erbakan University Meram Medical Faculty ethical committee decision dated and no: 2019/1655. All patients and volunteers were given detailed information about the electromyographic procedure and their written consents were obtained. The study followed the Declaration of Helsinki.

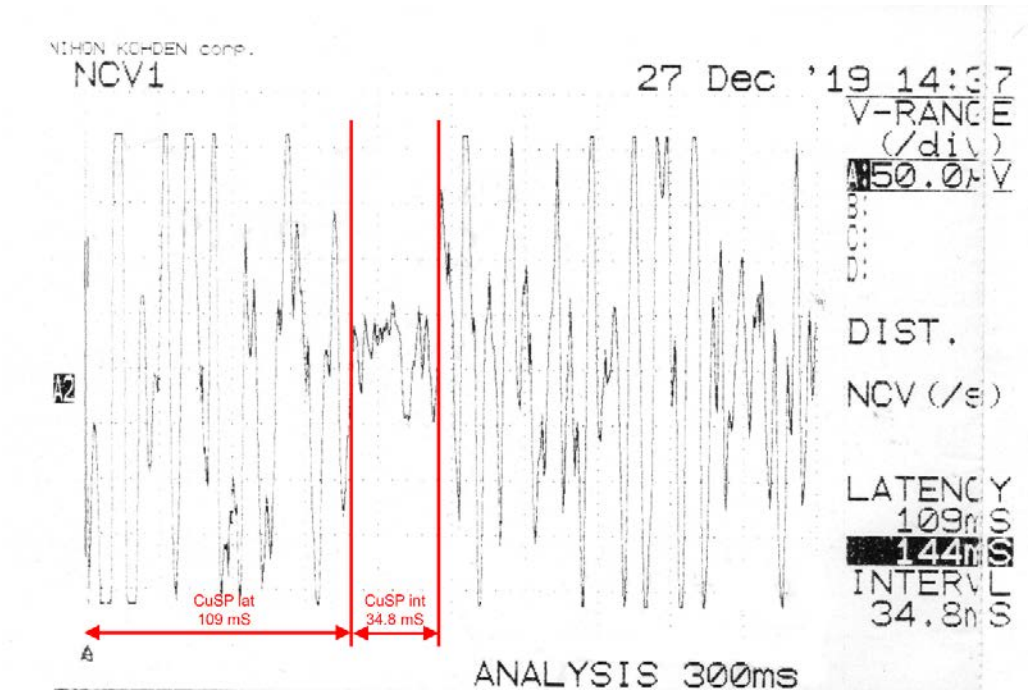
Nerve conduction and muscle evaluation

The electrophysiological records were obtained by using a Nihon Kohden (Model: MEB 7102K, SN: 1997-00457, Tokyo, Japan) brand device. Based on medical history and physical examination findings, the eligible patients' bilateral sternocleidomastoid, anterior scalene, trapezius, splenius capitis and cervical paravertebral muscles were evaluated using a concentric needle electrode.

Cutaneous silent period study

In the patient group, SCMs on the opposite side of where the chin was facing towards were determined to be dystonic sides. The side selections were confirmed by observing spontaneous motor unit potential activity with a needle EMG performed. The SCM muscle with no or minimal spontaneous motor unit potential activity was determined to be the normal side.

In order to obtain CuSP, the EMG device was tuned to the mode for motor unit evaluation, with its filters were manually calibrated for a velocity of 2 Hz-10 kHz, sensitivity of 500 μV and sweep duration of 300 msec. For recording, the disc probe was initially placed on the SCM muscle on the dystonic side or the predominant side in accordance with belly-tendon principle. The stimulation electrode was placed within the area innervated by supraorbital branch of the trigeminal nerve on the ipsilateral side of the recorded SCM muscle; the silent period (SP) was then obtained by electrical stimulation of the SCM muscle for 0.5 msec during 60% to 80% of the maximal contraction at a 10-fold greater sensory threshold potential (usually 30, 40 or 50 mA) (Figure 1). The onset and end of CuSP can be defined by quantitative criteria such as the unit of time when the EMG falls below 50% or 80% of the pre-stimulus baseline activity4. In our study, as SP, we accepted the area where the basic activity before the warning fell below 50%.



CuSP lat: Cutaneous silent period latency, CuSP int: Cutaneous silent period interval

Figure 1. CuSP sample obtained from dystonic SCM muscle in a patient with cervical dystonia.

At least 3 responses were attempted to obtain with this method. Onset and end latencies, as well as interval values of the silent period were determined in msec by visual inspection. In statistical analyses, medians of these measurements were used. SP latency indicated onset of SP, whereas the interval indicated duration of SP. All these procedures were performed by using standard electrodiagnostic equipment.

Statistical analysis

For our study, SPSS 15.0 (version 15.0; SPSS Inc, Chicago, IL) statistics package program was used. For intergroup comparisons, the comparisons were made between dystonic SCM measurements of the patients and SCM measurements of the controls. For intra-group comparisons, however, SP measurements of the right and left SCMs of the controls were compared between each other, and SP measurements of the dystonic SMC and contralateral SCM of the patients were compared between each other. A Kolmogorov-Smirnov test was performed for evaluation of distribution normality. A student t-test was performed for comparison between parametric groups. For intra-group paired comparisons, a paired sample t-test, a parametric test was performed. To investigate the linear correlation between the parameters, a Pearson correlation test was performed. To examine the correlation between the parameters with strong correlation, a Linear Progression Analysis was performed. A $p<0.05$ was considered statistically significant.

RESULTS

For the study, a dystonic patient group comprised of 20 participants (15 female, 5 male) with a mean age of 49.3 ± 14.7 years and a control group comprised of 25 participants (17 female, 8 male) with a mean age of 48.0 ± 13.7 years were included. No statistically significant difference was determined between the patient group and the control group in terms of age and gender ($p>0.05$). Twenty patients had a mean of 7.3 ± 6.8 yearly symptoms of CD. Of these patients; 6 (30%) were among those who had never received botulinum toxin injections, whereas other 14 patients had been receiving botulinum toxin injections for 3.1±4.5 years. Mean number of botulinum toxin injections the patients received was 7.8±10.2.

No significant difference was determined between SP latency and duration of dystonic SCM of the patient group and those of SCM of the control group (p: 0.762 for latency, and p: 0.318 for duration) (Table 1). Intra-group comparisons of SP measurements of bilateral SCMs of the patient and control groups, with no significant difference between these groups in terms of SP latency and duration values, were performed. No significant difference was determined between the dystonic SCM and the contralateral one in the patient group (p: 0.289 for latency, and p: 0.674 for duration) (Table 2). Similarly, both SCM measurements in the control group did not significantly differ (p: 0.554 for latency, and p: 0.900 for duration) (Table 3).

Table 1. Comparison of silent period parameters of the patient and control groups

	Patient's dystonic SCM (n=20)	Control's SCM (n=25)	p
SP Latency	38.08±19.16	36.52±13.89	.762
SP İnterval	39.05±14.56	35.24±9.37	.318

SP: silent period; SCM: sternocleidomasteideus

Table 2. Intra-group comparison of silent period parameters in the patient group

	Patient's dystonic SCM (n=20)	Patient's normal SCM (n=20)	p
SP Latency	38.08±19.16	35.55±20.84	.289
SP İnterval	39.05±14.56	37.60±11.49	.674

SP: silent period; SCM: sternocleidomasteideus

Table 3. Intra-group comparison of silent period parameters in the control group

	Control's right SCM (n=25)	Control's left SCM (n=25)	p
SP Latency	36.52±13.89	35.82±10.46	.554
SP İnterval	35.24±9.37	35.39±8.75	.900

SP: silent period; SCM: sternocleidomasteideus

Intra-group SP latencies of bilateral SCMs in both patient and the control groups were found to have a strongly positive correlation with each other (r: 0.869 and $p<0.0001$ for the patient group; r: 0.926

p<0.0001 for the control group). In regard to correlation for duration it was determined that the strongly positive correlation between SP duration of bilateral SCMs was ongoing in the control group (r: 0.776, p<0.0001), whereas the correlation between SP duration of bilateral SCMs in the patient group disappeared (r: 0.341, p: 0.141) (Table 4-5).

Table 4. Correlation between silent period variables of the control group (Pearson correlation test)

		Right SCM interval	Left SCM latency	Left SCM interval
Right SCM latency	r	-.030	.926**	.020
	p	.888	.000	.925
	N	25	25	25
Right SCM interval	r		-.007	.776**
	p		.974	.000
	N		25	25
Left SCM latency	r			-.063
	p			.766
	N			25

SCM: sternocleidomasteideus

Table 5. Correlation between silent period variables of the patient group (Pearson correlation test)

		Dystonic SCM interval	Dystonic SCM latency	Dystonic SCM interval
Dystonic SCM latency	r	.061	.869**	-.157
	p	.798	.000	.510
	N	20	20	20
Dystonic SCM interval	r		.170	.341
	p		.473	.141
	N		20	20
Dystonic SCM latency	r			-.145
	p			.541
	N			20

SCM: sternocleidomasteideus

A linear regression analysis was performed to examine the association between the parameters with strong intra-group correlations in both groups. Since distance is important in the measurement of conduction latency in electrophysiology studies, the SP measurements of reciprocal SCMs of the same individual in both the patient and control groups were included in the regression analysis as the dependent variable. In regard to SP latencies, a significant linear correlation was found between bilateral SCM measurements in the control group (p<0.0001, t: 11.764, B: 0.697, R square: 0.857). The same correlation was also observed between SP latency measurements of the dystonic and healthy SCM of the patient group (p<0.0001, t: 7.457, B: 0.799, R square: 0.755). In regard to SP durations, again, a significant linear correlation was determined between bilateral SCM measurements of the control group (p<0.0001, t: 5.903, B: 0.725 R square: 0.602), whereas no such correlation was determined between the dystonic side and the healthy side in the patient group (p: 0.141, t: 1.538, B: 0.269 R square: 0.116).

DISCUSSION

In this study, SP characteristics of dystonic SCM of patients with cervical dystonia were compared with those of the healthy side and those of the control group for evaluation of changes in inhibitory intermediate circuits of the brain stem, which is likely to be involved in pathophysiology of cervical dystonia. In consistency with the literature, the number of females was higher than males in the patient group(F/M:3/1)[14]. SP responses from bilateral SCMs of individuals in both groups were obtained with the aforementioned method, SP latencies and durations were compared between the dystonic group and the control group, which demonstrated no significant difference between the groups. Later, SP measurements of the dystonic SCM and normal SCM were compared in the patient group and, again, no significant difference was observed.

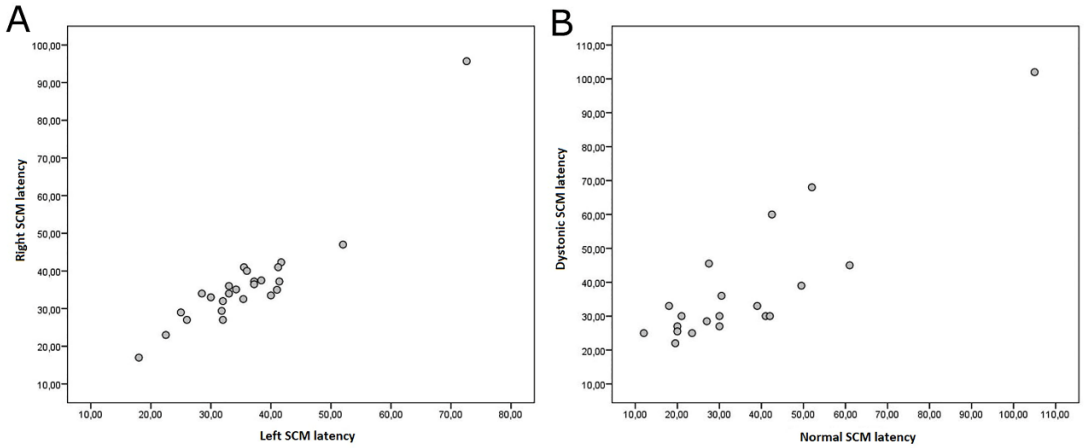
In the literature, SP studies in dystonic patients have been conducted as two major ways: CuSP and cortical silent period (CoSP). When the CoSP studies achieved with transcranial magnetic stimulation, the CoSP duration was found to be shortened in the affected neck muscles[15,16] and unaffected hand muscles in CD patients[17-19]. When Odorfer et al., discussed their findings with literature, they reported that CoSP durations were found to be shortened in all patients with writer's spasm, facial and cervical dystonia[17]. The CoSP duration has also been found to be shortened in various forms of idiopathic focal dystonia other than cervical dystonia[20-27]. Despite all these, there also are studies reporting that CoSP remained unchanged in dystonic patients[28-29].

In conclusion, in dystonic patients, the CoSP duration is mostly found to be shortened as to indicate loss of cortical inhibition. In CuSP studies, however, the obtained results are controversial. In the literature, there are studies not only reporting shortened or unobtained CuSP in dystonic

patients[12,30], but also reporting prolonged CuSP duration in dystonic patients[31,32]. As in our study, some studies determined no difference between the dystonic patients and the control group in terms of CuSP parameters. For instance, in a study by Nakashima et al., in which they examined SP characteristics they obtained from SCM of patients with spasmodic torticollis, there was no difference between the patient and control groups in terms of SP latency and duration11. Similarly, Tutuncu et al., carried out a CuSP study from APB muscle in 10 patients with cervical dystonia and 13 with generalized dystonia, and found no significant difference between the patients and the control group in terms of CuSP latency and duration[33].

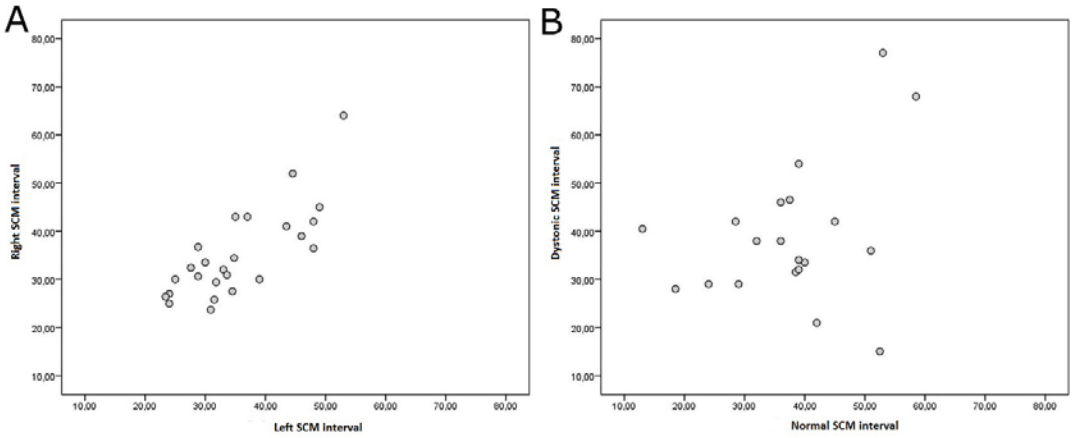
There also are studies in which CoSP and CuSP were studied together. Bocek et al. examined CoSP, CuSP and H-reflex in upper extremity muscles before and after bilateral pallidal stimulation in patients with generalized and cervical dystonia. Following the deep brain stimulation, there was no change in CuSP and H-reflex, whereas the CoSP duration, which was significantly shortened compared to the controls, became similar to that of the control group after the stimulation. In that study, it was emphasized that deep brain stimulation had no effect on CuSP but a potential therapeutic effect on CoSP[34].

As shown by the aforementioned information, results of the CuSP studies are not as consistent as those of CoSP studies. CuSP duration has been found to be prolonged, shortened or unchanged in some of previous studies. Also in our study, no statistically significant difference in CuSP parameters was observed in intra- and inter-group comparisons. However, in contrast to the literature, intra-group correlations of bilateral CuSP parameters of both patients and the controls were examined. A strong correlation was determined in SP latencies of the corresponding SCMs in both patient group (r: 0.869 $p<0.0001$) and the control group (r: 0.926 $p<0.0001$). While the same strong correlation was maintained also or SP durations in the control group (r: 0.776, $p<0.0001$), the correlation between SP durations of dystonic SCM and normal SCM disappeared in the patient group (r: 0.341, p:0.141) (Figure 2-3).



SCM: Sternecleidomasteideum

Figure 2. Scatter diagram showing the correlation between the SP latencies of the control group bilateral SCMs (A) and the scatter diagram showing the correlation between the SP latencies of the patient group bilateral SCMs (B).



SCM: Sternecleidomasteideum

Figure 3. Scatter diagram showing the correlation between SP times of control group bilateral SCMs (A) and scatter diagram showing the correlation between SP times of patient group bilateral SCMs (B).

Since stimulated and recorded distances in a healthy individual will be related to height and neck height of the individual as a fundamental principle of electrophysiology, if SP latency of right SCM is prolonged, it will be prolonged also on the left, if it is shortened on the right, it will be shortened also on the left. A similar situation is apparent for SP durations of the control group as well. Same harmony continued for SP latency in dystonic muscles, whereas this harmony was found to disappear in SP durations. In other words, random responses are obtained in SP durations of dystonic SCM, being longer or shorter than the healthy side in some patients. Despite of this disharmony, no inter-group difference develops due to bi-directional distribution. We are in thought of that the major reason for the chaos in the literature is this situation. Loss of the positive correlation between SP durations of bilateral SCMs in the patient group may be explained by irregular and chaotic response produced by dystonic SCM against exteroceptive stimuli.

Maintenance of the harmony in SP latencies in dystonic muscle may possibly result from intactness of the afferent and efferent pathways of this reflex. This irregularity in SP duration of the dystonic muscle may possibly be due to abnormal functioning of inhibitory interneurons that regulate the connection between Cranial Nerve V, which mediates exteroceptive inhibition, and motor neurons of the spinal accessory nerve and upper cervical nerves. When we review the literature, it is observed that latencies do not generally differ from the control groups in SP or other trigeminocervical reflex studies performed in dystonic patients, and that duration, amplitude or other characteristics of reflex responses obtained may differ from the control group[12,32,35]. Our study and aforementioned studies did not indicate the reflex pathways, but interneuron circuits that interconnect these pathways and regulate the response as the cause of abnormalities of brainstem reflexes.

Studies comparing pre- and post-treatment CuSP parameters in patients with various movement disorders may shed light on the association of these discussed abnormalities of interneurons in the brainstem and spinal cord with the cortex and basal ganglia. In studies conducted with patients diagnosed with Parkinson's disease, restless legs syndrome and essential tremor, significant changes in CuSP durations were observed primarily before and after treatment with centrally acting agents[36-39]. The centrally acting pharmacological agents used probably influence primarily the basal ganglia and cortex in these patients, normalizing abnormal SP characteristics at the level of spinal cord.

Our study has some limitations. One of them is that we could not do cortical silent period study, so changes in cortical level were not analyzed. Another is that our sample size is small (power analysis value 0.17). It can be said that the most important reason

for this situation is that cervical dystonia is a disease with a low frequency (5.7/100.000). However, our study has more patients than previous studies on the subject[11,12].

In the light of all these data, a potential underlying mechanism for the pathology under control of dystonic muscles may be that the dysfunction occurring in cortex and basal ganglia may lead to abnormalities in interneuron connections at the level of brainstem and spinal cord via descending pathways. As a result of this abnormality, the interneuron connections at the level of brainstem and spinal cord become disorganized, making the responses at these levels against exteroceptive stimuli unpredictable and chaotic. As a result of this, the positive intra-group correlation in SP durations which was observed in the control group disappeared in the dystonia group. This "disorganization" may explain the controversial results of the studies on dystonic patients which examined the difference in CuSP parameters between the patient and control groups.

In conclusion, these findings suggested that the abnormality of interneuron connections in the brainstem and spinal cord in dystonic patients may be a loss of organization in principle. We are in thought of that our study may shed light on SP and other electrophysiological studies regarding dystonia.

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