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Preparatory Brain Activity and Anticipatory Postural Control in Cervical Myofascial Trigger Point

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ABSTRACT

Introduction: Neck pain is a highly prevalent disorder in developed countries. A myofascial trigger point (MTrP) is distinguished under the name of the fundamental excessive menstruation for it and certain reason for musculoskeletal dysfunction. MTrP refers to a sensitive spot in a taut band whose stretch and compression can induce pain. Modifications in the motor cortex, sensory input, irritability, and integration are the adaptive mechanisms to pain. Accordingly, this study aimed to investigate the preparatory brain activity and anticipatory postural control in chronic neck pain.

Materials and Methods: The study participants included 25 women with an active MTrP in the upper trapezius muscle and 25 healthy women in the control group. We recorded the brain activities from Cz, Pz, and Fz regions and muscle activities of both sides of the upper trapezius, anterior deltoid, cervical and lumbar paraspinal, sternocleidomastoid, and medial head of the gastrocnemius. The participants had to flex their arms in reply to the second sound stimulus, followed by the first sound. Then, their reaction time and brain activity were evaluated.

Results: Significant differences between the two groups were detected in brain activities' measurements and the onset of muscle activation ($P<0.001$). The exception was the onset of gastrocnemius muscle activation ($P>0.05$).

Conclusion: The current study suggests that active MTrP induces latency and lengthens the muscle reaction time; thus, the production of suitable motion after the stimulus will be affected. Brain alteration after pain damages movement changes and postural control mechanism.

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1. Introduction

Neck pain is one of the most complaints reported in countries with high technology. According to the numerical analysis, 30% to 70% of the general population with neck pain are experiencing incremental and perpetual symptoms, such as an increased limitation of neck range of motion. A huge amount of money is spent by health organizations on neck pain [1, 2]. Many factors can potentially induce neck pain whose underlying cause is myofascial trigger point (MTrP), which, in turn, can lead to musculoskeletal disorders. MTrP is a hypersensitive spot and taut band formed in skeletal muscles [3]. Two kinds of MTrPs are active MTrPs (AMTrPs) and latent MTrPs (LMTrPs). AMTrPs cause disability and pain in the upper extremity [4].

Many possible factors can cause AMTrPs [4, 5]. One of the probable mechanisms for the improvement of AMTrPs is movement patterns that have changed. Altered movement patterns impose harmful stress on the tissue and cause chronic pain in skeletal muscles [6-8]. Patients with MTrPs, during voluntary tasks, utilize the altered postural positions, which helps change their movement patterns [4]. These alterations in how to recruit the postural muscles are related to changing motor function within the cerebral cortex [9, 10]. Hence, the alteration mentioned above with regard to AMTrPs reveals a long-term adjustment in the central neural control of posture. This alteration of posture may increase the chronic and recurrent pain symptoms in the neck and shoulder [4]. Yassin found that AMTrPs in the upper trapezius muscle increase the reaction time of the upper trapezius, sternocleidomastoid, lumbar, and cervical paraspinal muscles on both sides [5]. The changed muscle reaction time, also seen in patients with previous chronic back pain, is similar to patients with AMTrPs [11, 12].

Furthermore, the higher pain-related anxiety is directly associated with the perpetuation of postural alterations pursuant to the pain [13, 14]. In the standing position, an internal perturbation occurs in the body when the limbs and trunk move [15]. It is necessary to activate the postural trunk muscles and lower limbs to control the standing posture before voluntary movement to fulfill this compensation [16, 17]. This postural control is recognized as the anticipatory postural adjustments (APAs). Activation of APAs is prior to the voluntary movement and reduces the impacts of oncoming disturbance on posture and balance. Centrally preprogrammed motor commands manage the anticipatory postural muscle activities of the trunk and lower extremities [18, 19]. APAs are tuned by biomechanical particulars, e.g., movement velocity and preliminary standing position. APAs are influenced by several high-level cognitive processes such as anticipation [19-21], attention [19, 21, 22], and anxiety [13, 14]. Particular areas of the central nervous system (CNS) such as the prefrontal cortex, primary motor area, premotor area, supplementary motor area, and basal ganglia are involved in producing APAs [10, 23, 24]. In addition, previous studies suggest the significant task of these areas in motor programming and planning [24-26]. Gurfinkel and El'ner [16] reported that the supplementary motor area closely pertains to the APAs and relates to upper extremity movements. The previous activation of postural muscles in a patient suffering from the lesion of the supplementary motor area was neglected by them [16, 17]. Wadsworth et al. [27] explored the sequence and timing of rotator muscles of the scapula in shoulder abduction. They suggested that upper trapezius activity is a prime mover and stabilizer in shoulder abduction. The neurophysiologic mechanisms associated with acute pain-related alteration of postural muscles utility are still unknown.

A direct review is probably required to evaluate cortical signals in reply to pain to unerring study the brain in patients with MTrPs. Using Event-Related Potentials (ERP) of the CNS can help us to assess the activity of the CNS, which is happening before the voluntary movement. The contingent negative variation (CNV) as an ERP signal can be represented by a slow negative shift of electroencephalogram (EEG) amplitude in the preparatory period between warning to response stimuli [11, 28]. Walter et al. initially announced the use of CNV as a way to measure the primary activity of the brain [24]. CNV is a slow negative shift in EEG amplitude computed by EEG recorded average from beckon to response stimuli [29]. CNV also indicates that cerebrocortical activity is associated with a foresight to the warning stimulus, pre-movement, and motor provision reply to stimulus. CNV originates from the motor, cognitive, and sensory components of the primary, premotor, supplementary motor, and parietal and temporal cortices. CNV elements are the peak of late CNV amplitude, mean of CNV amplitude, and post-imperative negative variation (PINV) [29, 30]. Therefore, the CNV is a potential way to find out the alterations in the neural control of postures associated with the patients with MTrPs.

In this study, the chosen participants were patients with AMTrPs in the upper trapezius muscle. The onset times of some postural and cervical muscles and CNV potentials were studied in patients who suffered from AMTrPs in the upper trapezius muscle. Also, a comparison of in-

spections was made with a control group. Therefore, it was postulated that patients with AMTrPs show alteration in the onset time of muscle activation and alteration in cerebrocortical activation during upper extremity movement.

2. Materials and Methods

Study participants

Fifty participants aged 18 to 45 years old joined this study voluntarily. Twenty-five women were samples with one reachable AMTrPs in the upper trapezius muscle, and 25 healthy women formed the control group.

All phases of the experiments were approved by the Institutional Ethics Committee of the Tehran University of Medical Sciences and were explained to the study participants. The approval number was 92/D/130/297. Before taking part in this study, the participants presented their assent by signing the consent form. Then, the participants were assessed based on the following inclusion criteria: having neither severe postural disorders nor suffering from a seizure, depression, migraine, and other mentality dysfunction, lacking a history of operation during the last six months on the shoulder and neck, not undergoing treatment of AMTrPs within the last month, lacking any specific signs of dizziness and vertigo in motion or special position, reporting no complaints of painful joints, osteoarthritis, and cervical radiculopathy in the upper extremity, lacking temporomandibular joint dysfunction, and not consuming caffeinated food or drinks like coffee on the day of the experiment. Also, the participants of the controlled group should neither have AMTrPs in the upper trapezius nor other muscles of their heads and necks [15].

A proficient examiner did all the phases of the examination. The exclusion criteria were as follows: be in the period of the menstrual cycle, not to fulfill proper recording of Electromyography (EMG) and CNV, not feeling pain in the shoulder and neck, and using sleeping and sedative drugs within 24 hours before the examination, consuming caffeinated food or drink like coffee on the day of the experiment, and not completing the study due to pain and overwhelming tiredness.

To perform the inspection of MTrPs in the upper trapezius muscle, diagnostic measurement has been utilized, such as: 1) Existence of a tangible taut band whose snapping or palpation would activate the local twitch response, 2) Applying a 25 N/cm^2 pressure would trigger at least one hypersensitive tender point in the taut band found previously, and 3) Spontaneous presence of the typically referred pain pattern and patient recognition of the referred pain as familiar [31, 32].

If all of the above-mention indicators existed, those MTrPs would be considered AMTrPs [32].

Experimental protocol

The participant was requested to stand erectly with arms beside of body on a force platform for 10 s and repeat it 5 times with 30 s inter-trial intervals. Again, the participant was requested to stand on the force platform; unlike the previous position, her arms were in 60-degree flexion [20]. Shifting of the center of foot pressure (CFPy) was observed. If CFPy were about ±1 cm in an anterior-posterior direction, fundamental experiment phases would be started [20]. The goal of

the performance of this step was to monitor the body's sway. Two warning sounds were used as stimulus (S1) and responsive Stimulus (S2) with 2 s intervals as a preliminary period. All features of the two stimuli were the same such as intensity (50 dB), duration (100 ms), and frequency (2 kHz). This circuit was started by 3 s silence while the participant was in a standing position, and then the first stimulus started warning, and after 2 s silence, S2 was stimulated. Shoulder flexion to 90 degrees with the highest possible velocity was demanded from the participant [19]. The experimental setup is illustrated in Figure 1.

EEG recording

A 64-channel EEG system recorded the activity of the cortex (Brain Quick System 98, Micromed, Mogliano Veneto, Italy) (A/D convertor: 32 bit, gain: 20 mV/Div, and band-pass filter: 0.05-60 Hz) [19, 20]. We used silver-silver chloride cup electrodes 8 mm in diameter. Electrodes Fz, Cz, and Pz were placed on the scalp per the International 10–20 system. The standard reference electrode is located on the mastoid process in a monopolar position. In addition, a ground electrode was attached to Fpz. To control the artifacts triggered by blinking, electrooculograms (EOG) were recorded with electrodes placed above and below one of the eyes and a pair of electrodes on both sides of the eyes [21]. All electrode impedances were held less than 5 kΩ. The sampling rate of signals was 256 Hz. The recorded data were evaluated from 500 ms before S1 to 1500 ms after S2 [19, 20].

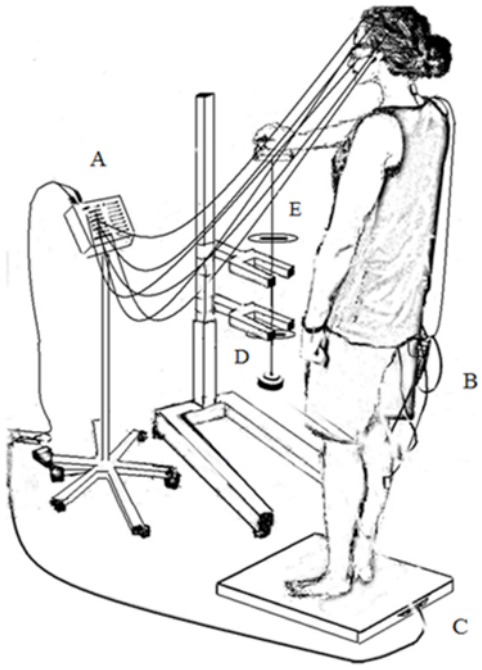

Figure 1. Experimental setup

A= Electroencephalograph tools, B= Electromyography tools, C= Force platform, D= Onset trigger or 60° sensor, E= Offset trigger or 90° sensor

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EMG recording

In this study, an 8-channel EMG system (P3×8 Biometric Ltd, Gwent, UK) with CMRR (common-mode rejection ratio) >96 dB at 60 Hz, input impedance >1012 Ω, gain: 1000 b, and pass filter of 20-450 Hz, and sensitivity of 100 μV/div was used. Signals were acquired at a sampling frequency of 1000 Hz. Surface electrodes were set at fixed positions on shaved and cleansed skin. The integral dry reusable electrodes (SX230, Biometrics Ltd, Gwent, UK) with 10 mm diameter, bipolar structure, and 20 mm inter-electrode distance were used. The electrodes were in the same direction of fibers located on the belly of each muscle based on SENIAM guidelines [19, 20, 33]. The ground electrode was connected to the individual's wrist. The electrode placements are listed in Table 1.

Data analysis and interpretation

Electroencephalography

CNV signals were recorded from 2 s before S1 to 2 s after S2 and were analyzed by CNV Analyzer Software. For data analysis, the time points of 500 ms before S1 and 1500 ms after S2 were chosen. If disturbing factors were identified during this interval, the received signal would not be picked out for construal. Artifacts include eye movement in the horizontal direction and blinking [19]. The peak of CNV amplitude 200 ms before S2 was described as the peak of late CNV amplitude. Definition of the mean CNV amplitude was the average value of CNV amplitudes, 500 ms before S1. The regression signal to the baseline is called the Post-Imperative Negative Variation (PINV). To compute PINV, the average value

Table 1. The electrode placement

Muscle	Location
Anterior part of the deltoid	One finger width distal and anterior to the acromion
Paraspinal cervical	Level of the C4 vertebrae
Paraspinal lumbar	Level of the iliac crest
Upper trapezius	Midway between acromioclavicular joint and C7 vertebrae
Sternocleidomastoid	One-third of the distance from sternal notch to mastoid processes
The medial head of the gastrocnemius	The prominent medial bulge of the muscle

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Table 2. Mean±SD Values of Anthropometric Parameters

Variables	Mean±SD		P
	Control	MTrP	
Age (y)	23.30±1.6	24.5±2.7	0.9
Height (cm)	164.76±6.45	163.35±5.4	0.9
Weight (kg)	56.53±6.2	57.5±5.47	0.8

Abbreviation: MTrP, myofascial trigger point.

*Significant at P < 0.05.

of CNV amplitudes ranges between 500 to 1500 ms after S2 was utilized [34].

Electromyography

To specify the initiation of activity while the shoulder was in an abducted position, the anterior deltoid was supposed to be the prime mover muscle. An algorithm was designed in the MATLAB software (v 7.6, 2008) to prepare a valid and reliable tool to specify EMG onset, particularly for fast movements, and to assign the onset of muscle activity [35]. Initially, the raw data were high-pass selected with a zero-phase shift, and a stoppage of frequency of 30 Hz was characterized to omit artifacts movement. Then, the Root Mean Square (RMS) values were selected with a moving window of 100 ms. The starting time was defined as the first EMG signal upgraded to a value higher than average plus 3 times the Standard Deviation (SD) (Mean±SD), stayed up for 100 ms, as per the procedure suggested by the Moraes et al. and Cools [36].

Statistical analysis

Based on the Kolmogorov-Smirnov test, all study data were normality distributed; hence, the independent t test determined the difference between the two groups. Also, the levels of association between the parameters for both groups were explored using the Pearson product-moment correlation coefficients. As per Cohen's values, the correlation coefficients (r) beyond 0.5 were shown good to excellent levels of relationship (low: r = 0.10 – 0.29, medium: r = 0.30 – 0.49, and high: r = 0.50 – 1.0). All statistical calculations were conducted using SPSS version 17.0 (SPSS Inc, Chicago, USA), and the statistical significance threshold was set at 0.05 for all tests.

Results

Anthropometric parameters

The Mean±SD values of age, height, and weight of participants in both groups are shown in Table 2. There was no statistically significant difference between groups in

Table 3. Results of contingent negative variation (CNV)

Variables	Mean±SD		P (t Test)*
	Control	AMTrPs	
PINV. Fz	111.71±-60.97	150.21±141.86	0.008
PINV. Cz	104.81±-48.59	186.87±131.01	0.019
PINV. Pz	104.32±-44.06	164.05±115.54	0.029
The peak of Late CNV. Fz	57.10±9.80	166.59±123.26	0.001
Peak of Late CNV.Cz	46.57±28.46	189.60±154.22	0.001
Peak of Late CNV. Pz	40.32±22.46	179.45±141.60	0.001
Mean of CNV. Fz	18.82±15.57	140.01±122.57	0.001
Mean of CNV. Cz	30.68±20.79	160.45±146.94	0.001
Mean of CNV. Pz	25.58±16.73	155.20±141.61	0.001

Abbreviations: PINV, post-imperative negative variation; AMTrP, myofascial trigger point.

* P<0.05

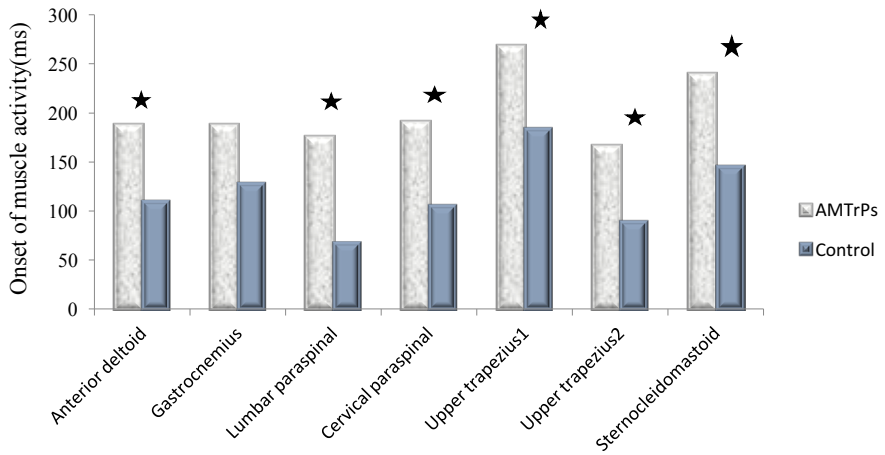

Figure 2. Comparing the onset of muscle activity

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*P<0.05.

Table 4. Linear relationships between contingent negative variation (CNV) and electromyography

Output 1	Output 2	r (P)	
		MTrPs Pearson	Control Pearson
PINV Cz	Onset time of AD	0.640(0.044)	0.115(0.693)
	Onset time of UT with MTrPs	0.675(0.033)	-0.238(0.413)
	Onset time of SCM	0.645(0.042)	-0.121(0.681)
Peak of Late CNV Fz	Onset time of AD	-0.594(0.046)	0.575(0.043)
	Onset time of LP	0.290(0.314)	0.853(0.001)
	Onset time of UT	-0.582(0.050)	0.825(0.002)
	Onset time of UT with MTrPs	0.081(0.784)	0.749(0.006)
	Onset time of SCM	0.176(0.546)	0.722(0.001)
Peak of Late CNV Cz	Onset time of AD	-0.072(0.807)	0.499(0.035)
	Onset time of LP	0.186(0.534)	0.852(0.001)
	Onset time of UT	-0.040(0.891)	0.854(0.001)
	Onset time of UT with MTrPs	-0.035(0.905)	0.774(0.004)
	Onset time of SCM	0.054(0.853)	0.739(0.007)
Peak of Late CNV Pz	Onset time of GcM	-0.827(0.006)	0.183(0.531)
	Onset time of LP	-0.711(0.024)	0.820(0.002)
	Onset time of UT	0.090(0.761)	0.771(0.005)
	Onset time of UT with MTrPs	0.092(0.754)	0.646(0.022)
	Onset time of SCM	0.196(0.502)	0.701(0.012)
Mean of CNV Fz	Onset time of AD	-0.612(0.040)	0.199(0.494)
Mean of CNV Pz	Onset time of GcM	-0.693(0.028)	0.257(0.376)
	Onset time of LP	-0.660(0.038)	-0.288(0.318)

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Abbreviations: PINV, post-imperative negative variation; AMTrP, myofascial trigger point; AD, anterior deltoid; LP, lumbar paraspinal; SCM, sternocleidomastoid; GcM, gastrocnemius; UT, upper trapezius. Correlation is significant at the 0.05 level (2-tailed). r is defined as the Pearson product-moment correlation coefficient.

age, height, and weight before the study. Both groups were homogeneous.

The Mean±SD values of pain and pain pressure threshold were 65±25.14 mm and 6.33±3.99 N/cm^2, respectively, in the AMTrPs group.

CNV parameters

The results of the CNV comparison between the two groups are presented in Table 3. There were significant differences between these two groups in accordance with the PINV, the peak of late CNV, and the mean of CNV in the Fz, Cz, and Pz areas (P<0.001).

EMG parameters

The onset of muscle activity

There was a significant difference between the AMTrPs and the control group in the initiation of activity of anterior deltoid (AD), cervical paraspinal (CP), lumbar paraspinal (LP), sternocleidomastoid (SCM), and upper trapezius (UT) muscles with AMTrPs (P<0.05). The exception is the medial head of the gastrocnemius (GcM) muscle. The latency in the onset of muscle activity occurs more in all muscles of the AMTrPs group than in the control group. Figure 2 compares two groups of the control and the AMTrPs regarding the initiation of activation of muscles.

Correlation between the parameters

The correlations between the outputs of CNV and EMG obtained from the Pearson correlation are listed in Table 3. It is essential to mention that only correlations with significantly less than 0.05 in each group are presented in Table 4.

4. Discussion

The outcome of this study shows that these two groups are different with respect to the onset of muscle activity. The increase in the onset of muscle activity is associated with the increased amplitude of CNV. In the MTrPs group, the extended time of movement raised the peak amplitude of late CNV and the mean amplitude of CNV in the AMTrPs group compared to the control group. A similar study reports the increased peak amplitude of late CNV in patients with chronic low back pain (LBP) [37-41].

Sadeghi et al. [37] investigated the effect of LBP on the CNV parameters. Their study specifies that fast arm movement into flexion direction increases the latency of CNV amplitude in the AMTrPs group compared to the control group. Many studies prove that prefrontal cortex activation in the postural control is more in LBP patients, proposing that they have more cognitive spinal control than the control group.

Latency of CNV is significantly associated with the prefrontal cortex [19]. Late CNV is attributed to motor preparation, anticipatory attention, and cognitive processes [30, 42]. Hence, concerning the previous research, severe engagement of the prefrontal cortex and attention may explain the increased peak of the amplitude of late CNV [28, 43] in the AMTrPs group. Impaired neural circuits that generate CNVs, such as supplementary motor area, premotor area, and basal ganglia, could be considered the reason for the increased peak amplitude of late CNV. The prominent task of these areas in the initiation of the voluntary movements as well as motor programming and planning of sequential movements has been well demonstrated [25, 26]. The general movement strategy is ascertained in motor planning by retaining and running the motor programs. The motor programs select the appropriate subordinate routines for the requested movement. They appoint a particular group of muscles to prepare subordinate routines and adjust the proper timing of the onset of muscle activity [44]. In other words, the motor programs determine the particulars of a movement, consisting of the temporal sequence of muscle activity, the duration of the activity, and the force created by each muscle [30].

Thus, the coordinated movements depend on the correct timing in the running and switching of motor programs [26]. Accordingly, a higher engagement of the prefrontal cortex alters mean CNV amplitude, the peak of late CNV, and the onset of muscle activation in patients with AMTrPs. In this study, a delayed onset activity of LP, AD, GC, CP, UT-free MTrPs, and UT muscles was observed in the AMTrPs group. The increased onset of muscle activity is consistent with alteration in the arousal level of CNS and the growth in CNS parameters. Jacobs et al. [10] stated that the reason for increased motor response and movement time in patients with a high level of excitement might be due to encountering much more sensory system inputs. Impaired information processing should clarify the cause of the unusual response to peripheral stimuli. Patients with AMTrPs have impaired limbic systems, particularly damaged movement planning [45]. Reduced neuromuscular control in the AMTrPs group might be associated with impairment, as explained above. Increased tone of cervical muscles may affect neuromuscular control in the AMTrPs group [18, 46].

Another explanation for impaired neuromuscular control in patients with AMTrPs is increased sympathetic response. Higher inputs of different cutaneous afferents are the reason for sympathetic response engaged. Eventually, gamma fusimotor in the muscle spindle and cervical proprioception were affected by the increased cutaneous afferent inputs [37, 44, 47, 48]. The necessary prerequisite to having effective motor planning consists of organization and integration of sensory inputs from the muscles as well as interpretation of data [47]. It is noteworthy that sensor processing is a challenging task in patients with AMTrPs, leading to inefficient motor planning for movement initiation and ineffective postural preparation.

Increased PINV showing the preparation of the neuromuscular system for admission to new conditions was another CNV factor assessed in this experiment. In this study, PINV reflects a growing trend in the AMTrPs group. This consequence proves the preparation of the neuromuscular system for admission to new situations; thus, in the AMTrPs group, a reduction in the ability to manage and forecast a specific perturbation is considered. In addition, after each stimulus, the time required for CNS to admit a new stimulus is increased. The trend of hyperactivity in CNS results in lowering self-regulation. An abnormal pattern of muscles or distinct co-activation might be the reason for this condition. This action could undoubtedly impact motor planning and movement programming. Although patients with AMTrPs appropriately react to the stimulus, their reaction time is longer. Accordingly, patients with AMTrPs in their muscles suggest increased PINV amplitude and the onset of muscle activity.

The outcome of correlation analysis in the AMTrPs group states that earlier onset of muscle activities is associated with the higher peak of late CNV and mean CNV amplitudes. This result might lead us to a severe lesion of the prefrontal cortex, supplementary motor area, premotor area, and basal ganglia in the patients with AMTrPs. The involvement of CNS affects the muscle programming and different muscle reaction, which finally end in the increased activation of the muscle. Also, increasing CNV latency and deterioration of postural preparation could be related to the weak performance of the somatosensory cortex in patients with AMTrPs. The dysfunction of the somatosensory cortex results in the incorrect activation of specific muscles. Hence, the changed APAs in the patient with AMTrPs show a long-term adjustment in the central neural control of posture. This alteration of posture may increase the chronic and recurrent pain symptoms in the neck and shoulder. These alterations may cause reduced movement control and shoulder and cervical muscle impairments.

A high positive correlation was considered between the late CNV peak and CNV latency in Fz, Cz, and Pz and initiation of AD, LP, UT, and UT with AMTrPs and SCM in the control group. The mean value of CNV was positively correlated with the onset time of AD. The earlier onset of the time of muscles may be associated with the lower CNV amplitudes. Consequently, the outcomes of correlation analysis can explain the associations of EEG and EMG parameters in AMTrPs and control groups.

The limitations of this research are as follows: An insufficient number of joint shoulder muscles examined and the lack of comprehensive evaluation regarding the counter effect and deficits of muscles inserted into scapula or pertaining to shoulder elevation.

5. Conclusion

The current study suggests that active MTrP induces latency and lengthens the muscle reaction time; thus, the production of suitable motion after the stimulus will be affected. Brain alteration after pain damages movement changes and postural control mechanism.

Ethical Considerations

Compliance with ethical guidelines

The research was approved by Local Ethics Committees of Tehran University of Medical Sciences. The approval number was 92/D/130/297.

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Authors' contributions

All authors were equally contributed in preparing this article.

Conflict of interest

The authors declared no conflict of interest.

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