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Sensory processing and central pain modulation in patients with chronic shoulder pain : a case-control study

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INTRODUCTION

Shoulder pain is the third most common musculoskeletal disorder with strongly diverging life time prevalence numbers ranging up to 67%.¹ Only half of all shoulder patients recover during the first 6 months, and 40% of them still report problems after one year.² Furthermore shoulder pain problems are characterized by a strong episodic nature with high recurrence rates.³ Chronicity and recurrence in shoulder pain is therefore a problem, not only for the individual, but also for the socio-economical system facing substantial costs for care and sick leave.⁴

Central sensitization (CS) is defined as “an amplification of neural signaling within the central nervous system (CNS) that elicits pain hypersensitivity”.⁵ This mechanism encompasses distorted sensory processing in the CNS⁶, malfunctioning of descending pain-inhibitory mechanisms⁷, enhanced activity of pain-facilitatory mechanisms⁸ and long-term potentiation of the neural synapses in the anterior cingulate cortex⁹, and can amplify pain experiences by increasing its degree, duration, and spatial extent.⁵ Temporal Summation (TS) is a phenomenon in which an individual experiences progressive increase in pain intensity during the repetition of identical nociceptive stimuli. It is assumed to represent summation of C-fiber mediated second pain and hence a pain-facilitatory mechanism.^{10,11} To assess malfunctioning of descending pain-inhibitory mechanisms, the ‘conditioned pain modulation’ (CPM) paradigm has been recommended.¹² The awareness is growing that clinicians should integrate the concept of CS during clinical reasoning and patient management.^{13–15}

Despite conflicting evidence, several authors have concluded that a possible explanation for chronification of musculoskeletal shoulder pain can be found in the concept of CS.^{16–20} This notion is supported by the findings from a systematic literature review that weighted the available evidence and concluded that CS is a feature of

chronic shoulder pain.²¹ Still, the number of studies that examined aspects of CS in chronic shoulder pain is limited, some had methodological flaws (e.g. possible selection bias), and so far a comprehensive quantitative sensory testing (QST) protocol has not been used for examining features of CS in patients with chronic shoulder pain. In addition, pain perception is known to be influenced by several demographic characteristics (e.g. gender, race and age)^{22–25} and is prone to cognitive²⁶, emotional²⁷ and other psychosocial factors²⁸. Furthermore, a growing amount of evidence for the influence of sports participation and regular physical exercise on pain perception exists.^{29–31} Previous work did not address these factors thoroughly, warranting further research in this area. Finally, few studies examined the TS and CPM paradigms concomitantly in one study in patients with chronic shoulder pain.

Therefore, the overall purpose of this study was to gain knowledge about sensory processing and central pain modulation in patients suffering from chronic shoulder pain. Our primary study aim was to compare a group of patients with chronic unilateral shoulder pain and pain-free healthy controls using a comprehensive QST protocol, including measures of TS and endogenous analgesia. QST thereby provides a means to identify altered static sensory and nociceptive processing using thermal and mechanical trigger-response relationships.³² We hypothesized that patients suffering from chronic (more than 3 months in duration) musculoskeletal shoulder pain, compared to pain-free healthy controls, are characterized by altered sensory and pain processing. Since pain perception is known to be influenced by intrinsic and extrinsic factors (cfr. supra), we explored the associations of pain related outcome measures with a selection of relevant factors (e.g. psychological factors and sports participation) within both groups as a secondary study aim.

METHODS

Study Design

A single blinded (blinded evaluator) case-control study design was used. Figure 2 provides an overview of this study design. The ethics committee of the Antwerp University Hospital approved the study (ref. nr. B300201316593).

Participants

Patients suffering chronic unilateral shoulder pain were recruited from two orthopedic settings and physiotherapy departments in Antwerp (Belgium). The patient group consisted of adult subjects suffering self-reported unilateral shoulder pain. Patients were included if they reported having unilateral shoulder pain (figure 1, pain in right or left marked area of the body chart) for at least 3 months with a current pain intensity of at least 4/10 on a numeric pain rating scale (NPRS) (scaling 0 (= no pain at all) to 10 (= worst pain imaginable)). Reported shoulder pain problems could be idiopathic, traumatic or non-traumatic. Participants reporting any rheumatic, neurologic or cardiovascular problem were excluded. When using medication with a known influence on the central nervous system (e.g. anti-epileptic, anti-depressant), analgesics and/or NSAID's, the participant needed to be stable in medication intake for at least 4 weeks prior to his/her participation to be included in this study. The control group consisted of pain-free and healthy subjects without a history of shoulder or neck pain requiring medical treatment. They were relatives, friends or acquaintances of researchers, students, and university personnel. In both groups we have included participants characterized by a diverse profile of sports participation and physical activity status. Both groups were matched for age, gender, hand dominance and sports participation. This way the study accounted for the possible influence by several

demographic characteristics.^{22–25} The assessor was blinded for the participants' condition. All participants received an information leaflet and provided us with a written informed consent. The rights of all subjects were protected.

Psychophysical testing

In this study we used a modified QST protocol, as being clarified hereafter, to assess static sensory and nociceptive stimulus-response relationships. Furthermore and after completion of the modified QST protocol we used CPM and TS, both being dynamic measures of pain modulation.

Quantitative sensory testing (QST)

Hereby we selected three mechanical triggers/sensations known to show modified stimulus-response relationships in chronic musculoskeletal pain problems such as fibromyalgia syndrome, whiplash associated disorders and osteoarthritis.^{14,33} To ensure local, distant and widespread stimulation, all sensory input were applied on five body regions being bilaterally at the middle of the upper trapezius muscle belly (fig. 1: A & B) and at the dorsal side of the middle-finger between the PIP and DIP joint (fig. 1: C & D), and unilaterally at the muscle belly of the right leg calf muscle (fig. 1: E). The modified QST protocol was performed in the order listed hereafter:

Pressure pain thresholds (PPTs) were measured using a calibrated digital algometer (Wagner Instruments, FDIX; Wagner Instruments, Greenwich, CT, USA) by gradually increasing mechanical pressure perpendicularly to the test side at a computer-controlled rate of 1 kg/s until the moment the sensation first becomes painful. Participants were instructed to say “stop” at this moment. The amount of force applied at this moment is defined as the PPT. Pressure algometry was found to be a reliable method to determine pressure pain thresholds.³⁴

Information on *mechanical detection (MD)* was gained using the Neuropen (Owen-Mumford Ltd, Oxford, UK) which consists of a 10-g monofilament to assess large myelinated fibre sensory perception (hereafter referred to as dull detection) on one end, and a pinprick mechanism (Neurotip™) to test small C-fibre sensory perception (hereafter referred to as sharp detection) on the other end. The Neurotip™ delivers a standardized 40-g force using a calibrated spring mechanism. This instrument has shown to be simple and sensitive.³⁵ Participants were stimulated three times (with each tool) at the above outlined body parts, and were asked to inform the researchers on detection ('How many times did you feel the stimulus?') and pain provocation ('Was the stimulus painful?'). Positive scores on these detection tests were defined as 'not feeling a stimulus' and 'the stimulus being painful'.

Vibration detection thresholds (VDT) were measured using a vibrometer (TSA II, Medoc Inc., Israel) consisting of a computer-controlled device ending on a round plastic disc (1 cm²), which was placed perpendicularly to the test site. The frequency of vibration was gradually increased until the patient felt sensation. The patient was asked to say 'stop' when the vibration sensation was first felt. The frequency of vibration applied at this moment is defined as the VDT.

The above mentioned 'static' measures of thresholds provide a limited view on the pain-processing system. After completion of the modified QST protocol two additional and 'dynamic' tests (TS and CPM) were used to further explore the pain-processing system in patients with chronic shoulder pain and healthy controls.

Temporal summation (TS)

A 10-fold repeated mechanical stimulation using a calibrated digital algometer (Wagner Instruments, FDIX; Wagner Instruments, Greenwich, CT, USA) at 110% of pressure

pain threshold intensity with an inter-stimulus interval (ISI) of 1 second was applied to elicit temporal summation at all the above-mentioned regions. TS elicitation did commence at least 2 minutes after the final PPT is measured to make sure no contamination of tests can occur. Subjects were asked to verbally rate their pain level of the first, fifth and 10th pulse on a numeric pain rating scale (NPRS) (scaling 0 (= no pain at all) to 10 (= worst pain imaginable)).

Conditioned Pain Modulation (CPM)

To evaluate the endogenous analgesic system, we used the CPM-model (previously known as diffuse noxious inhibitory control (DNIC)). The effect of CPM on pressure pain thresholds was studied using a painful conditioning stimulus while applying pressure algometry on the previously mentioned anatomical reference points except the finger of the dominant hand. As a conditioning stimulus, we used a thermal and computer-controlled stimulus of 0°C provided by a thermode (TSA II, Medoc Inc., Israel) on the participants thenar eminence of the dominant hand. CPM was expressed as the relative difference in pressure pain thresholds measured before and during application of the conditioning stimulus.

Questionnaires

A structured interview was conducted with each participant. Information regarding general demographic data (age, gender and hand dominance), medical status, medical history and medication intake was obtained during this interview. Information on sports participation was also captured during this interview. All participants needed to indicate whether or not they participate in sports activities. If so, they were asked to inform us on their average amount of time they performed these activities each week. This information was used to check for eligibility and during data analysis.

Several standardized questionnaires were applied to gain information on the psychosocial context each participant acts. The pain vigilance and awareness questionnaire (PVAQ) and the pain catastrophizing scale (PCS) were reported and analyzed here.

The PVAQ was developed as a broad measure of attention to pain and can be applied to various pain populations.³⁶ The PVAQ consists of 16 items and respondents are asked to consider their behavior over the last 2 weeks and to indicate how frequently, on a six-point scale from zero (never) to five (always), each item is a true description of their behavior.³⁶ Pain catastrophizing is known to be a predictor of pain outcomes.³⁷ The PCS asks participants to reflect on past painful experiences and to indicate on a 5-point scale from 0 (not at all) to 4 (all the time), the degree to which they have experienced each of 13 thoughts or feelings when experiencing pain. The PCS is divided into 3 components (rumination, magnification and helplessness) and a total score is computed as a summation of the 3 components.³⁸

Sample Size Determination

Based on previously published data³⁹ and on the aim to detect clinical significant differences of 20% on PPT levels between both groups, sample size calculation generated the need for at least 25 participants in each group. Within this calculation an α -level of 0.05 and a power of 0.80 was used.

Statistical analysis

All data were analyzed using IBM SPSS Statistics v.22.0 for Windows (IBM Corporation, Somers, NY). Normality of the variables was tested visually using Q-Q plots and with the Kolmogorov-Smirnov test. Appropriate descriptive statistics were calculated. When necessary log transforming the data made the distributions more normal. Comparisons between chronic shoulder pain patients and healthy controls

were performed using independent sample t testing except when indicated differently. Spearman's rho and Pearson's correlation analyses were used to examine the relationships between selected intrinsic characteristics and measured variables. Absolute values for r below .40 are considered as weak, those between .40 and .60 as moderate and those above .60 as strong correlation. For each statistical test, the significance level was set at $P \leq 0.05$.

RESULTS

Participants

Twenty-five patients suffering chronic shoulder pain and 25 healthy controls were recruited for the study. Participants' characteristics are presented in table 1. Both groups were matched for age, gender, hand dominance and the amount of sports participation. Table 1 also briefly presents results of the questionnaires. In the patient group, we observed that in about one-third of the individuals, the affected shoulder was the non-dominant shoulder. To build a matched control group for the affected shoulders in the healthy pain-free group, we randomly selected one-third of the non-dominant shoulders and two-third of the dominant shoulders. These shoulders were labeled as ipsilateral shoulders in the healthy *and* pain-free control group. It resulted in a distinct denomination of the psychophysical tests on the ipsilateral and contralateral upper trapezius muscle belly.

Psychophysical testing

All participants participated and completed the psychophysical test procedure. The results of the adapted QST-protocol are presented in table 2. None of the selected static measures showed statistically significant differences between participants with chronic shoulder pain and healthy controls.

The results of the dynamic psychophysical tests are presented in table 3. Both the patient and the control group showed an increase in pain rating during the test for TS. However, no statistical significant difference exists between both groups ($p > 0.05$). The healthy control group showed only negative CPM results, indicating an increase in pressure pain sensitivity during a conditioning stimulus, whereas the patient group

showed positive CPM results at both the affected and unaffected shoulder (see Table 3). Again, these differences reached no statistical significance.

Correlation analysis

Correlation coefficients between 2 variables were calculated for both groups, patients and controls. Only significant correlations were reported in table 4. Strong positive correlations between age and vibration detection thresholds are apparent in the patient group. Moderate positive correlations exist between the amount of sports participation and pressure pain thresholds in patients, whereas these PPTs show weak-to-moderate negative correlations with pain catastrophizing. Typical examples of the latter correlations are shown in figure 3.

DISCUSSION

The purpose of this study was to gain knowledge about sensory processing and central pain modulation in patients with chronic musculoskeletal shoulder pain. Inadequacy of these mechanisms could possibly indicate a role for CS in chronification and recurrence in musculoskeletal shoulder pain as concluded earlier by several authors.^{16–19,21} In this study however we did not find significant differences between patients suffering from chronic shoulder pain and healthy control subjects based on static and dynamic psychophysical measures. Noteworthy are some moderate associations of these measures with selected intrinsic and extrinsic factors, e.g. a moderate positive correlation between sports participation and pressure pain thresholds at different body regions.

Included patients were selected based on their self-reported clinical history indicating the presence of unilateral musculoskeletal shoulder pain for at least three months, with a pain rating of at least 4/10 on a NPRS. Our selection procedure, irrespective of a possible medical diagnosis, was different from other studies using specific pathologies and/or diagnoses and/or treatment options (e.g. arthroscopic surgery) as inclusion criteria.^{16,17,20} This supposition of the patient being exposed to negatively burdened medical information (e.g. medical diagnosis, surgical treatment planned) may facilitate the occurrence of placebo driven hyperalgesic responses which are known to be associated with negative expectations, anxiety and catastrophizing.^{40,41} These placebo effects are known to possibly play a role in the transition from acute pain into chronic pain.⁴²

In this study we have combined an adapted static QST-protocol and dynamic measures of pain modulatory mechanisms on five distinct body areas, including the affected shoulder region in the patient group. According to our knowledge, the

combination of static and dynamic measures in patients with shoulder pain has previously been used in two studies only.^{20,43} As in this study, Valencia et al. compared patients with chronic shoulder pain with a control group. They found evidence for the presence of CS in the patient group based on differences in suprathreshold heat pain responses.²⁰ Similar to our findings they could not observe group related differences in CPM.²⁰ Their application of thermal stimuli and their specific patient selection (i.e. all scheduled for surgery) could account for the differences with our results.²⁰ In their study using pressure and thermal stimuli as static measures, and thermal TS as a dynamic measure in patients with preoperative unilateral shoulder pain, Coronado et al. found evidence for the presence of augmented sensitivity for experimental pain at the involved side.⁴³ As it was not their primary intention to examine nor conclude for the presence of CS in this study, they did not include a healthy control group.⁴³ Furthermore they have not defined chronicity as an inclusion criterion despite the possibility that varying stages of the same pathological process may result in differences in experimental pain sensitivity.⁴³

Our results on pressure algometry in patients with chronic shoulder pain reject the hypothesis of the presence of CS within this group. These results support the findings of Albuquerque-Sendín et al. in patients with unilateral shoulder impingement syndrome.⁴⁴ However, based on pressure algometry a number of studies provided evidence supporting the presence of central sensitization in chronic shoulder pain.^{17–19} This studies' patient group shows large dispersions on the amount of sports participation known to possibly influence pain perception.^{29,45} Growing evidence does indeed indicate long term effects of physical training on pain related outcome measures such as improved pain tolerance and higher CPM in endurance athletes.^{29–31} The effects of regular exercise on PPTs are less uniform.²⁹ Within this patient group it is

apparent that PPTs show moderately positive correlations with the amount of sports participation, indicating that those who participate more in sports are less sensitive to experimentally induced pressure pain. Influences of sports participation on pain perception, although not previously studied in shoulder pain patients, could therefore be an explanation for our results being in conflict with previous studies. Further research within this field is necessary.

To the best of our knowledge, vibration detection or vibration perception has not been studied before in chronic musculoskeletal shoulder pain patients. This measure is often used to detect peripheral nerve disorders and is selective for large and myelinated A α and A β sensory fibres.⁴⁶ No significant differences were found between patients and healthy controls indicating the absence of peripheral neural dysfunction of the specified neural fibres. Additionally we could not find any differences in dull A-fibre mediated and sharp C-fibre mediated detection. This reflects the absence of differences between shoulder patients and healthy controls with respect to hypoesthesia, hyperalgesia and allodynia.

As mentioned earlier we have added measures of TS and CPM as two dynamic psychophysical tests to further explore the pain modulatory processes in patients with chronic shoulder pain. Augmented TS is known as a sign of CS caused by an increase of C-fibre-mediated responses in the multi-receptive dorsal horn neurons.^{8,11,47} No differences were observed between patients and healthy pain-free controls. Using the pain-inhibits-pain model by the paradigm of CPM, we experimentally tested the malfunctioning of descending pain-inhibitory mechanisms.¹² No statistically significant differences were noted when comparing shoulder pain patients with the control group. These results are in accordance with the findings by Valencia et al.²⁰

Based on our findings we cannot conclude that CS is a characteristic feature in chronic musculoskeletal shoulder pain, like it is in chronic whiplash associated disorders, chronic fatigue syndrome, fibromyalgia syndrome and irritable bowel syndrome.^{13,14} These results however do not reject the possibility for the presence of central sensitization in a subgroup of patients with chronic shoulder pain as seen in subgroups presenting with other musculoskeletal pain problems such as chronic low back pain, osteoarthritis and tendinopathy.^{14,48–50} Indicative for this possibility is the high variability in the obtained values for CPM and PPT at local and remote body areas. Additionally we have found moderately negative correlations of pain catastrophizing and pain sensitivity measured using PPTs, indicating that those who catastrophize more appear more sensitive for experimental pressure pain. Future research including subgroup analysis seems important to explore and define these and other influential factors. Despite the presence of conflicting evidence it seems imperative for clinicians to consider the concept of central sensitization during clinical reasoning and patient management in those suffering chronic shoulder pain.^{13,15,51}

Even though this study was the first to apply a comprehensive QST protocol and accounted for several methodological flaws of previous studies (e.g. selection bias) in this area, our results should be interpreted in the light of a limitation in our protocol. Due to practical reasons and to avoid contamination of tests we chose to adapt the protocol for quantitative sensory testing.³² On the other hand, the study has several strengths. The evaluator was blinded for the participants' condition. Furthermore, heterogeneity and diversity concerning age, gender, sports participation and other psychosocial factors facilitated the aim to avoid sample selection bias based on these factors.

PERSPECTIVE

Chronic shoulder pain is highly prevalent in the general population. As in other regional musculoskeletal problems a broad biopsychosocial approach is needed to thoroughly assess and manage it. This clinical necessity is reflected by a growing body of knowledge within this context. Our study contributes by further exploring the interaction between chronic shoulder pain and pain neuroscience. It is the first study using an adapted comprehensive quantitative sensory testing protocol for exploring sensory processing and central pain modulation in patients with chronic shoulder pain. Our findings, based on static and dynamic psychophysical measures, indicate adequate functioning of sensory processing and central pain modulation in chronic shoulder pain patients. Based on these findings we can conclude that central sensitization is no characteristic feature in people suffering from chronic musculoskeletal shoulder pain. Given the high prevalence of shoulder injuries in sports, our results could be of importance in sports related shoulder pain. We should however take note of the specific biopsychosocial context of sportsmen and –women facing intrinsic and extrinsic factors possibly contributing when injured.^{45,52} Further research within this context is needed.

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FIGURE 1. Body chart.

FIGURE 2. Flow chart of the study design.

TABLE 1. Participants' characteristics

	Patient group (n=25)	Control group (n=25)	p- value
Age (years)	31.4 (10.97)	26.12 (8.29)	0.07
Gender (M/F)	12/13	12/13	/
Hand dominance (R/L)	22/3	22/3	/
Weekly sports participation (hours/week)	3.72 (4.82)	3.60 (2.87)	0.92
NPRS (0-10)	5.52 (1.94)	/	/
Duration of complaint (months)	34.3 (40.4)	/	/
PVAQ	38.44 (8.83)	33.24 (6.97)	0.03
PCS	16.8 (10.29)	9.56 (6.85)	0.01

Abbreviations: n, number; M, male; F, Female; R, Right; L, Left; NPRS, Numeric Pain Rating Scale; PVAQ, Pain Vigilance and Awareness Questionnaire; PCS, Pain Catastrophizing Scale

TABLE 2. Results from the static measures (adapted quantitative sensory testing-protocol)

	Patient group (n=25)	Control group (n=25)	p-value
PPTs (Nm) – mean (SD)			
<i>PPT ipsilateral upper trapezius</i>	45.7 (20.1)	40.24 (24.0)	.217*
<i>PPT contralateral upper trapezius</i>	42.7 (16.3)	39.18 (24.8)	.214*
<i>PPT right middle finger</i>	94.7 (33.1)	90.24 (27.9)	.613
<i>PPT left middle finger</i>	98.1 (31.7)	87.65 (28.3)	.224
<i>PPT right calf muscle</i>	62.6 (28.8)	58.37 (26.6)	.593
VDT (Hz) – mean (SD)			
<i>VDT ipsilateral upper trapezius</i>	2.69 (1.63)	2.18 (0.88)	.368*
<i>VDT contralateral upper trapezius</i>	2.58 (1.94)	2.19 (0.93)	.391
<i>VDT right middle finger</i>	0.86 (0.47)	0.81 (0.49)	.580*
<i>VDT left middle finger</i>	0.76 (0.41)	0.59 (0.39)	.050*
<i>VDT right calf muscle</i>	5.29 (4.43)	3.81 (3.48)	.325*
DD (freq)			
<i>Absent Sensation (freq)</i>	3/125	2/125	
<i>Pain Sensation (freq)</i>	0/125	7/125	
SD (freq)			
<i>Absent Sensation (freq)</i>	1/125	0/125	
<i>Pain Sensation (freq)</i>	31/125	27/125	

Abbreviations: n, number; PPTs, Pressure Pain Thresholds; Nm, Newton Meter; SD, Standard Deviation; VDT, Vibration Detection Threshold; DD, Dull Detection; SD, Sharp Detection; freq, frequency

* Indicates statistics performed on log transformed data

TABLE 3. Results from the dynamic measures

	Patient group (n=25)	Control group (n=25)	p- value
TS (NPRS) – median (IQR)			
<i>TS ipsilateral upper trapezius</i>	2 (3)	2 (4)	0.93*
<i>TS contralateral upper trapezius</i>	1 (2)	2 (3)	0.50*
<i>TS right middle finger</i>	1 (3)	1 (3)	0.83*
<i>TS left middle finger</i>	2 (3)	2 (2)	0.91*
<i>TS right calf muscle</i>	1 (2)	2 (3)	0.23*
CPM (%) – mean (SD)			
<i>CPM ipsilateral upper trapezius</i>	8.39 (40.33)	-0.78 (33.42)	0.39
<i>CPM contralateral upper trapezius</i>	3.86 (28.33)	-7.47 (23.16)	0.13
<i>CPM middle finger</i>	-15.06 (19.67)	-16.14 (21.89)	0.85
<i>CPM right calf muscle</i>	-6.45 (37.45)	-2.22 (28.41)	0.65

Abbreviations: n, number; TS, Temporal Summation; NPRS, Numeric Pain Rating Scale (0-10); IQR, InterQuartileRange; CPM, Conditioned Pain Modulation; SD, Standard Deviation

* Indicates the use of Mann-Whitney U test

TABLE 4. Results from the correlation analyses

Intrinsic parameter	Psychophysical variable	Patient group (n=25)	Control group (n=25)
Sports Participation	PPT ipsilateral upper trapezius	0.488	NS
	PPT left middle finger	0.494	NS
	PPT calf muscle	0.584	NS
NPRS	PPT right middle finger	-0.397	-
	PPT left middle finger	-0.469	-
	PPT calf muscle	-0.405	-
	CPM middle finger	0.405	-
PVAQ	PPT right middle finger	-0.429	NS
	PPT calf muscle	-0.587	NS
	TS right middle finger	-0.431	NS
PCS	PPT ipsilateral upper trapezius	-0.478	NS
	PPT contralateral upper trapezius	-0.399	NS
	PPT right middle finger	-0.597	NS
	PPT left middle finger	-0.524	NS
	PPT calf muscle	-0.396	NS
	CPM ipsilateral upper trapezius	NS	-0.400

Abbreviations: n, number; NS, Non-Significant ($P \geq 0.05$); PPT, Pressure Pain Threshold; NPRS, Numeric Pain Rating Scale; CPM, Conditioned Pain Modulation; PVAQ, Pain Vigilance and Awareness Questionnaire; TS, Temporal Summation; PCS, Pain Catastrophizing Scale