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FIBROMYALGIA AND TEMPOROMANDIBULAR DISORDER: AN ELECTROMYOGRAPHIC ANALYSIS

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SUMMARY
Temporomandibular Disorder (TMD) development in fibromyalgia syndrome (FMS) is still superficially understood and altered neuromuscular control in FMS may play a triggering role for the TMD. This work involved an analysis of facial pain and median frequency of myoletric signal of masticatory muscles in patients with FMS (n=27) and TMD (n=28). We found that possible different neuromuscular controls in masticatory muscles are correlated with perception of facial pain in patients with FMS.

INTRODUCTION
Coexistence of Fibromyalgia Syndrome (FMS) and myofascial pain associated with Temporomandibular disorder (TMD) has been reported in the literature[1]. However the development of TMD in FMS patients can be particularly different. This is because previously studies reported spinal cord hyperexcitability[2] and a different pattern of muscle activation in FMS. So here we analyzed the electromyographic signal (median frequency) of masticatory muscles and the facial pain of patients with FMS and TMD.

METHODS
It was examined 27 female FMS patients (53.2(5.61) years old) and 28 female TMD patients (45(9.53) years old). The subjects were evaluated based on the Diagnostic Criteria for Temporomandibular Dysfunction Research (RDC/TMD) and surface electromyography (SEMG). The intensity of facial pain was assessed by the Visual Analogue Scale (VAS).
The SEMG signal was recorded simultaneously by the four electrodes attached to the skin on the region of the right and left temporalis (TR and TL) and masseter muscles (MR and ML), following the recommendations of The International Society of Electrophysiology and Kinesiology (ISEK). Briefly, simple active differential surface electrodes were used, composed of two parallel bars of pure silver, 1mm thick and 10mm long, with a distance of 10mm between electrodes, a 20-fold increase (gain), an input impedance of 1015 Ohms and a common-mode rejection ratio (CMRR) of 92dB (Datahominis Tecnologia Ltda). The electrodes were connected to a MyosystemBr1_P84 (portable model) signal acquisition module. The SEMG signals were amplified 100-fold at a frequency of 2 kHz and band-pass filtered (20-1000Hz - Butterworth). The reference electrode was placed to the region of the ulnar styloid process and greased with gel, while the active differential electrodes were placed on the muscle bellies. Prior to attaching the electrodes, the skin was cleansed with 70% alcohol. During the collection of the signals, the patient sat on a chair, leaning against the back of the chair, Frankfurt plane parallel with the floor, eyes open, feet planted on the floor and arms resting on the thighs. Three 15-second recordings were collected at maximum intercuspation (isometry), while clenching "Parafilm M" between the premolars and molars to ensure the reliability and effectiveness of the recording. Data acquisition was controlled by a specific software program with 16-bit resolution, the Myosystem-Br1 software application, based on the median frequency (MNF) of the myoelectric signal calculations. To observe the behavior of the MNF over time in isometric contraction, SEMG signal windows were defined using a specific software program, disregarding the first and last window of the signal, and analyzing the second, fifth and ninth windows.
In the SEMG analysis under maximum isometric contraction, one can see physiological muscle fatigue, which occurs when the median frequency shifts towards lower frequencies.
The data were subjected to an analysis of variance (ANOVA), followed by Tukey's post hoc test and to complement the analysis of variance and test the effect of windows and covariables on the median frequencies, the Tukey-Kramer multiple comparison test of means was applied, maintaining the 5% level of significance. For the comparison means of the slope coefficient of the linear regression line of the electromyography signal spectrogram of the masticatory muscles was performed unpaired Student's t-test or Mann-Whitney test, maintaining the 5% level of significance.

RESULTS AND DISCUSSION
We found a significant decrease in the MNF values over time in isometric contraction of masticatory muscles at both studied groups, i.e., characterizing muscle fatigue, but the decreased coefficient (measured by SLOPE) is not different between of then (Slope Median of the linear regression line of the EMG signal of Left Masseter muscle was -1.97 (2.5) for FMS and -1.65 (2.4) for TMD (p value = 0.63); Right Masseter was -2.44 (2.7) for FMS and -2.44 (2.7) for TMD

(p value= 0,48), Left anterior Temporalis muscle was -0.9 (3.46) for FMS and -1.81 (2.4) for TMD (p value = 0,29) and Right anterior Temporalis was -2.3 (4.7) for FMS and -1.48(2.01) for TMD in isometric contraction (p value = 0.23) (at maximum intercuspation).
A significant correlation was found between the higher values of median frequency of the myoelectric signal and the increase in facial pain in FMS. We found equations with positive slope for MNF and pain in FMS and the opposite occurred in TMD, i.e., negative slope for MNF and pain (Figure 1).

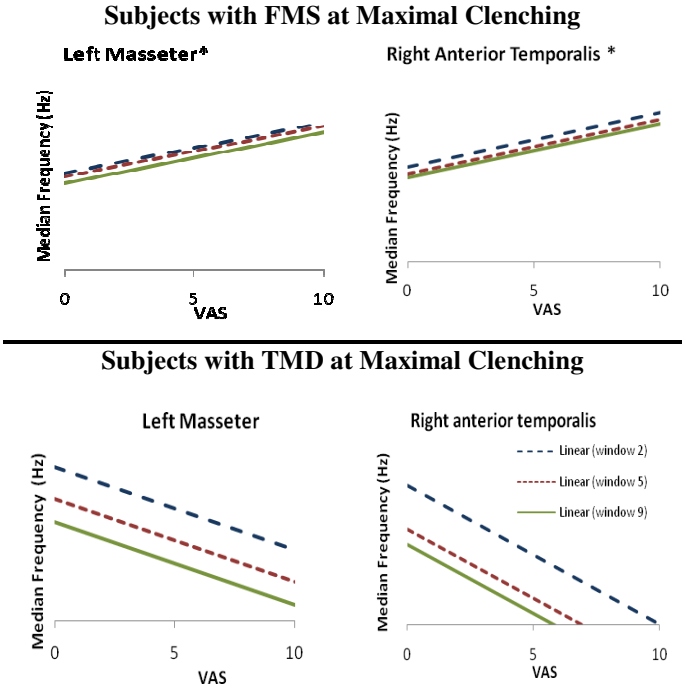

Figure 1: Equations from the analysis of variance with repeated measures to test the effects of VAS(cm) covariable (facial pain) on the Median Frequency (Hz) in the left masseter isometric (y = 7.1898 x + b) and right anterior temporal (y = 8.2928 x + b) with *(p <0.05) in the electromyographic signal windowing in patients with FMS (n = 27). And equations from the analysis of variance with repeated measures to test the effects of VAS(cm) covariable (facial pain) on the Median Frequency (Hz) in the left masseter isometric (y = -0.014x + b) and right anterior temporalis (y = -25.41x + b) in the electromyographic signal windowing in patients with TMD (n = 28) with p>0.05.

The level of neural drive (number of motor unit action potentials) in the presence of facial pain was different in FMS patients than TMD patients.

Integrated Pain Adaptation Model (Murray and Peck, 2007)[3] proposes that changes in muscle activity limit movement and thereby protect the sensorimotor system from further injury. With pain, a new, optimized motor unit recruitment strategy arises, leading pain minimization in order to maintain homeostasis. Therefore in FMS, contraction may have occurred discharging the motor units at higher frequencies (tetanic contraction) in order to activate the required contraction, which is even more fatiguing, generating a cycle of muscle fatigue and pain.
So there must be some abnormality in the afferent feedback making inefficient muscle control in FMS patients and leading to no protection of the stomatognathic system, being able to act as a predisposing or perpetuating factor of facial pain in these patients.

CONCLUSIONS

Here we raise the hypothesis that the FMS can play a triggering role of TMD, since FMS patients experience facial pain associated with a different SEMG response. It seems that pain not inhibits muscle contraction.

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