

TMD chronic pain and masseter silent period in psychiatric patients on antidepressive therapy

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SUMMARY The aim of the study was to evaluate the long-term effects of antidepressive therapy on chronic pain and related disability, and masseter silent period in psychiatric depressive patients with temporomandibular disorders (TMD). The study included hospitalized psychiatric depressive patients on antidepressive therapy protocol (tetracyclic antidepressant-maprotiline and anxiolytic-diazepam) ($n = 30$) and non-psychiatric patients seeking prosthodontic treatment (control group, $n = 38$). TMD were diagnosed by Research Diagnostic Criteria for temporomandibular disorders proposed by Dworkin and LeResche. The surface electromyography was recorded from left and right masseter muscles and masseter inhibitory reflex (masseter silent period) was recorded after mechanical stimulation. The incidence of TMD appearance was very similar, of approximately 40% in both group of patients. The results of the study also indicated a

higher prevalence of joint related TMD, a lower prevalence of muscular subtype of TMD and a lower grade of chronic pain and related disability in the psychiatric group of patients on antidepressive therapy in comparison with findings in the control group. In the patients on antidepressive therapy with TMD masseter silent period was not prolonged, while in the control group of patients with TMD the prolongation of the silent period was observed. The study provided evidence that long-term, combined therapy (maprotiline and diazepam) in psychiatric depressive patients significantly modulated signs and symptoms of TMD in comparison with the control group.

KEYWORDS: temporomandibular disorders, chronic pain, psychiatric patients, antidepressive therapy, masseter silent period

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Introduction

Many clinical studies have revealed that the prevalence of temporomandibular disorders (TMD) is high in a normal and healthy non-patient population (1–3). These dysfunctions refer to a collection of medical and dental conditions with common characteristics of orofacial pain as well as masticatory muscles and temporomandibular joint (TMJ) dysfunctions (4, 5). Orofacial pain in TMD is a chronic pain of muscular or joint origin, and, as all chronic pain conditions, is associated with psychological, behavioural and social factors in addition to physical pathology (6). It is interesting to note that myogenic pain patients, as a class of TMD patients, exhibit higher levels of psychopathology than

joint disease patients (7, 8) and that pain and impairment of activity are associated with negative emotionality in muscle pain patients, but not in joint pain patients (9). Although tricyclic antidepressant drugs were primarily used for the management of depression, there are data concerning their usefulness in the management of a variety of chronic pain conditions, including pain in TMD (10, 11) because of their analgesic activity (12, 13). Nowadays, because of the severe side effects, tricyclic antidepressants are replaced by newer, tetracyclic antidepressants such as maprotiline, for the treatment of depression (14). In contrast to well-established analgesic effect of tricyclic antidepressants, there are only few reports concerning analgesic effect tetracyclic antidepressants, and only for chronic

pain such as chronic tension headache (15) and low back pain (16).

Anxiolytic agents such as benzodiazepines have been prescribed for TMD patients when high levels of emotional stress, acute and chronic, are thought to contribute to TMD (4, 17). On the other hand, benzodiazepines, acting as central myorelaxants, effectively reduce chronic myofascial orofacial pain (18) as well as chronic TMD-associated myofascial pain (19).

Besides the chronic pain, dysfunctional muscular activities, resulting in prolongation of muscle silent period, have been considered to aggravate TMD (20–22). Silent period has been suggested as a quantitative measure of pathophysiological processes, such as headache (23), trigeminal neuralgia (24) and TMD (25). However, investigations concerning association between prolonged muscle silent period and TMD give conflicting results. Namely, Hanson *et al.* (26) reported no significant difference in the length of the masseter silent period between subjects with internal derangement of the TMJ and healthy subjects. Zulquarnain *et al.* (27) have suggested that silent period in both masseter and anterior temporalis muscles is not much of value in mild mandibular dysfunction as in severe cases, since they observed that the length of this parameter in the mentioned muscles did not differ generally in patients with mild or moderate mandibular dysfunction symptoms from the asymptomatic subjects. On the other hand, there are studies confirming increased silent period in groups of TMD symptomatic patients. Mc Call and Hoffer (20) reported prolonged duration of the electromyographic (EMG) silent periods of the anterior temporal and masseter muscles in subjects with TMJ dysfunction compared with the asymptomatic subjects. Further, Liu *et al.* (22) reported that muscle pain, as well as joint pain, were positively associated with silent period in masseter muscles in patients with TMD. Also, Skiba and Laskin (21) showed that there was a direct relationship between the severity of symptoms and the degree of masticatory muscle silent period prolongation in myofascial pain dysfunction syndrome.

Concerning all these facts, the aim of the present study was to compare signs and symptoms of TMD in psychiatric patients on antidepressive therapy protocol (tetracyclic antidepressant and benzodiazepine) with respect to those in the control group of non-psychiatric patients with TMD.

Material and methods

Subjects

The group of psychiatric depressive patients consisted of 30 adults, 12 men and 18 women, age ranging from 26 to 48 years, selected from the patients at the Division of Psychiatry at Military Medical Academy in Belgrade, Serbia. Depression was diagnosed by ICD-10 codes F32–F33 (28). These patients have been treated with long-term antidepressive therapy for approximately 7 months between the beginning of the antidepressant therapy and examination. For the purpose of successful treatment antidepressive drug, maprotiline (Maprotilin)*, daily dose 75 mg and anxiolytic, diazepam (Bensedin)[†], daily dose 10 mg, were administered. The control group, non-psychiatric patients included 38 adults, 18 men and 20 women, age ranging from 20 to 43 years, selected from the patients seeking treatment in the Division of Prosthodontics at Military Medical Academy in Belgrade, Serbia. This treatment embraced aesthetic prosthodontic treatment excluding any occlusal adjustment.

Inclusion criteria for both groups were age between 20 and 50 years and absence of occlusal disturbances according to the Helkimo Index criteria (29). Exclusion criteria for both groups were systemic muscle and joint disorders, neurological (brainstem problems) and vascular diseases, neoplastic or congenital disorders and recent surgical treatment in orofacial region.

Experiments were undertaken with the understanding and written consent of each subject. The study was approved by the Ethical Committee of Military Medical Academy in Belgrade.

Clinical evaluation of TMD

Research Diagnostic Criteria for temporomandibular disorders (RDC/TMD) provides a standardized system for examination, diagnosis and classification of the most common subtypes of TMD, which includes the use of a dual-axis approach: Axis I and Axis II. In all subjects the Research Diagnostic Criteria for TMD (RDC/TMD) proposed by Dworkin and LeResche (30) were conducted as diagnostic criteria for the presence or absence of the TMD. Axis I involves the clinical TMD condition

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and Axis II includes several grades of chronic pain and related disability: the grade 0 – no TMD in the prior 6 months; the grade I – low disability–low intensity pain; the grade II – low disability–high intensity pain; the grade III – high disability–moderately limiting and the grade IV – high disability–severely limiting.

The RDC/TMD history questionnaire, examination forms and specifications for examinations used in the current study were identical as described in the mentioned study (30).

Electromyography registration

Electromyographic recordings were carried out on the following manner. Each subject was seated upright in the comfortable chair with head supported. Sites to be tested were chosen with manual palpation to determine the central part of the left and right masseter muscles midway between the upper and lower borders, and 1 cm posterior to anterior border. Ag-AgCl surface electrodes, 4 mm in diameter, were placed 15 mm apart along the muscle belly and a ground electrode was placed on the left upper arm. The skin over the recording position was cleaned with alcohol swabs. A conductive gel was placed between the electrode and the skin to reduce the impedance below 20 k Ω . The electrodes were fixed with adhesive tapes. After placing and connecting the electrodes, each subject was asked to rest and to clench to verify sufficient amplitude and the absence of artefact and line frequency interference. The silent period was induced by a light mechanical tapping the subjects chin with a tendon hammer (weighting 80 g, from the distance of 4 cm), during maximal voluntary teeth clenching. Seven trials were run, with inter-trial interval of 10 s. All traces were measured individually, and these seven measured values were averaged in each subject. Onset and offset latencies were determined corresponding to the beginning of the EMG suppression when EMG derivated below the line representing 80% of the mean background EMG activity, and return of EMG activity above those values. The corresponding duration of silent period was calculated as the time between onset and offset latencies (31). The EMG signal was amplified 2000–5000 using a Premiere[‡], filtered (bandpass filter, 0.05–2 kHz), digitized (A/D rate 4 kHz) and stored for off-line analysis.

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Statistical analysis

The data were subsequently exported to SPSS for statistical analysis. Chi-squared test, Fisher's exact probability test and Mann–Whitney *U*-test were applied for testing of the clinical parameters and Student's *t*-test for testing of the masseter silent period duration, respectively, between investigated groups. The significant level was set at $P < 0.05$. A Spearman's correlation coefficient was used to detect the association of masseter silent period and chronic pain and related disability in each group ($P < 0.01$). Two-way ANOVA was used for the analysis of masseter silent period with different groups (control and psychiatric depressive patients) and TMD (with or without TMD) as main factors. For further analysis of the results of two-way ANOVA it was used Scheffe test for *post hoc* comparison.

Results

In the control group of patients 42% showed signs and symptoms of TMD. About 31% of these patients had muscular subtype of TMD, while approximately 25% expressed signs and symptoms of joint subtype of TMD (disc displacements). The rest of the patients (44%) showed a combined form of TMD, muscle-joint disorders (Table 1). In this group none presented signs and symptoms of other joint conditions (arthralgia, osteoarthritis and osteoarthrosis). In the psychiatric group of patients 47% expressed signs and symptoms of TMD. Signs and symptoms of muscle disorders were presented in about 7% of these patients, joint disorders (disc displacements) in around 43% of the patients, and other joint conditions in about 21% of the patients. The rest of the patients (29%) showed a combined form of TMD, muscle-joint disorders (Table 1).

Statistical analysis (chi-squared test) of difference in TMD prevalence between investigated groups of patients showed that this difference was not statistically significant ($P = 0.332$). On the other hand, concerning certain subtypes of TMD, the psychiatric group of patients showed significantly higher prevalence of joint related TMD subtype comparing to the control group (Fisher's exact probability test, $P = 0.03$). Although the observed differences in prevalence of muscular and combined subtypes of TMD between the groups were high it was not statistically significant ($P = 0.12$), ($P = 0.20$) (Table 1).

Table 1. Frequency distribution of the subtypes of TMD (according to Axis I) in the control group and the psychiatric group of patients on antidepressive therapy protocol (PGP)

Patients	Without TMD, <i>n</i>	With TMD						Total with TMD, <i>n</i>	Total, <i>n</i>	
		MD	DD	OJC	Joint subtype total, <i>n</i>	MD + DD	MD + OJC			Combined subtype total, <i>n</i>
Control group	22	5	4	0	4	5	2	7	16	38
PGP	16	1	6	3	9	2	2	4	14	30
Statistical results, <i>P</i> -value		<i>P</i> = 0.12 [†]			<i>P</i> = 0.03[†]			<i>P</i> = 0.20 [†]		<i>P</i> = 0.332*

n, Number of patients; MD, muscle disorders = muscle subtype; DD, disc displacement; OJC, other joint conditions; Joint subtype, (DD) + (OJC); Combined subtype, (MD + DD) + (MD + OJC).

*Chi-squared test for TMD distribution between investigated groups (*P* > 0.05).

[†]Fisher's exact probability test for subtypes of TMD between investigated groups (*P* < 0.05), bold indicates statistical significance (*P* < 0.05).

Table 2. Chronic pain and related disability, masseter silent period duration and their correlation in the control group and the psychiatric group of patients on antidepressive therapy protocol (PGP)

	Control group				PGP			
	I	II	III	IV	Total	I	II	III IV Total
I Graded pain and related disability, <i>n</i>	5	4	5	2	16	9	5	- - 14
Statistical results(I), <i>P</i> -value								<i>P</i> = 0.015*
II Silent period (ms), mean ± s.d.	20.68±0.64	21.44±0.17	23.80±1.45	31.00±2.35	17.31±4.93	17.35±	1.74	
Statistical results, (I:II) <i>P</i> -value	0.934 (<i>P</i> < 0.001)[†]				-0.129 (<i>P</i> = 0.840) [†]			

Results for silent period are expressed as average values for left and right side (mean ± s.d.).

n, Number of patients.

*Mann-Whitney *U*-test, bold indicate statistical significance (*P* < 0.05).

[†]Spearman's rho (*P*-value), bold indicates significant positive correlation (*P* < 0.01).

Table 2 represents the data concerning chronic pain and related disability, and masseter silent period in the control group of patients and in the psychiatric patients on antidepressants with TMD. The measurement of chronic pain and related disability, using parameters of Axis II, showed that TMD patients in the control group expressed equally grade I, II and III of chronic pain. Only two patients were characterized by high disability and severely limiting (grade IV of chronic pain) (Table 2). In contrast to that, 64% of the psychiatric patients with TMD showed grade I of chronic pain, while the rest of the patients showed grade II of chronic pain and related disability (Table 2). The grade of chronic pain and related disability in psychiatric depressive patients was significantly lower (Mann-Whitney *U*-test, *P* = 0.015) with respect to the control group (Table 2).

The results in Table 2 also present average values of silent period measurements on the left and right

masseter muscle. In the control group of patients with TMD the duration of masseter silent period increased with the degree of chronic pain and related disability and a Spearman's correlation coefficient revealed a statistical significance of this relationship (*P* < 0.001) (Table 2). On the contrary, in the psychiatric patients on antidepressive therapy with TMD masseter silent period did not increase with raise in grade of chronic pain and related disability and there was no statistically significant correlation (*P* = 0.840) (Table 2).

The duration of masseter silent period in the left and right masseter muscle with respect to age and gender is shown in Table 3. The patients in the control group without TMD showed silent period duration in range of 14–17 ms for masseter muscles, while in control patients with TMD, silent period was prolonged and lasted from 20 to 32 ms. In the psychiatric patients on antidepressive therapy the duration of the masseter silent period was not significantly different (Student's

Table 3. Masseter silent period in the control group and psychiatric group of patients on antidepressive therapy protocol (PGP) with respect to age and gender

Patients Gender	<i>n</i>	age (mean ± s.d.)	Silent period (mean ± s.d.) (ms)				<i>P</i> -value between subgroups
			Left masseter	Right masseter	<i>P</i> -value left/right	average value left/right	
Control group of patients without TMD	22	33.07 ± 5.90	16.26 ± 1.96	16.16 ± 1.73	<i>P</i> = 0.332	16.21 ± 1.84	<i>P</i> = 0.084(1:4) <i>P</i> = 0.335 (1:3)
Female	14	31.64 ± 5.40	15.55 ± 2.29	15.48 ± 2.44			
Male	8	34.50 ± 6.98	16.96 ± 1.62	16.83 ± 1.01			
Control group of patients with TMD	16	31.35 ± 5.18	23.63 ± 2.79	23.67 ± 2.98	<i>P</i> = 0.846	23.65 ± 2.88	<i>P</i> < 0.001(2:1) <i>P</i> < 0.001(2:3) <i>P</i> < 0.001(2:4)
Female	6	31.33 ± 4.84	23.03 ± 1.67	23.33 ± 1.55			
Male	10	31.40 ± 5.52	24.24 ± 3.90	24.01 ± 4.42			
PGP without TMD	16	36.53 ± 3.55	15.29 ± 3.07	15.31 ± 3.62	<i>P</i> = 0.778	15.30 ± 3.34	<i>P</i> = 0.846(3:4)
Female	10	36.90 ± 4.25	15.09 ± 2.42	15.39 ± 2.20			
Male	6	36.16 ± 2.85	15.50 ± 3.73	15.23 ± 5.05			
PGP with TMD	14	36.36 ± 3.73	17.47 ± 2.32	17.45 ± 2.27	<i>P</i> = 0.851	17.46 ± 2.25	
Female	8	35.14 ± 4.04	16.87 ± 3.22	16.80 ± 3.10			
Male	6	37.57 ± 3.42	18.08 ± 1.42	18.10 ± 1.44			

Student *t*-test (*P*-value), bold indicates statistical significance (*P* < 0.05).
n, Number of patients.

t-test, *P* = 0.846) between patients with or without TMD, lasting from 13 to 18 ms. Statistical analysis showed significantly prolonged masseter silent period in the control group of patients with TMD (*P* < 0.001) with respect to masseter silent period duration in the control group of patients without TMD (*P* < 0.001) and psychiatric depressive patients with (*P* < 0.001) and without TMD (*P* < 0.001). In all examined groups there was no statistically significant difference in masseter silent period duration between left and right masseter muscle: control group of patients without TMD (*P* = 0.332), control group of patients with TMD (*P* = 0.846), psychiatric patients on antidepressive therapy without TMD (*P* = 0.778), psychiatric patients on antidepressive therapy with TMD (*P* = 0.851) (Table 3).

Two-way ANOVA showed significant influence of patients group (*P* < 0.001), TMD (*P* < 0.001) and significant interaction patients group – TMD (*P* < 0.001) on the masseter silent period duration (Table 4). Gender (*P* = 0.090) as well as age (*P* = 0.735), as covariates, were without significant influence on masseter silent period duration (Table 4). *Post hoc* Scheffe test showed that the significant difference between control groups, with and without TMD, with respect to duration of masseter silent period existed (*P* < 0.001). The mentioned test also revealed statistically significant difference in duration of masseter silent period between

Table 4. Two-way ANOVA effect of patient groups (control and psychiatric depressive patients) and TMD (with or without) on masseter silent period duration

Source of variation	Silent period		
	df	<i>F</i>	<i>P</i>
Patients groups	1	17.51	<i>P</i> < 0.001
TMD	1	42.18	<i>P</i> < 0.001
Patients groups x TMD	1	13.61	<i>P</i> < 0.001
Covariates:			
Age	1	0.116	<i>P</i> = 0.735
Gender	1	3.87	<i>P</i> = 0.090

Bold indicates statistical significance (*P* < 0.05).

control group with TMD and psychiatric patients on antidepressive therapy with TMD (*P* < 0.001) as well as between control group with TMD and psychiatric patients on antidepressive therapy without TMD (*P* < 0.001)(Table 5).

Discussion

The obtained results show that TMD occurred in 42% of patients in the control group, seeking prosthodontic treatment and never treated with antidepressants. However, higher frequency of TMD, 68% and 69% was reported by Pedroni *et al.* (3) and Schiffman *et al.*

Table 5. *Post hoc* Scheffe test for analysis of the significance of interaction between groups with and without TMD in control and PGP with respect to masseter silent period

Groups	Diagnosis	Silent period (ms)			
		1. 17.46	2. 15.30	3. 23.65	4. 16.21
1. PGP	With TMD		$P = 0.199$	$P < 0.001$	$P = 0.508$
2. PGP	Without TMD	$P = 0.199$		$P < 0.001$	$P = 0.870$
3. Control	With TMD	$P < 0.001$	$P < 0.001$		$P < 0.001$
4. Control	Without TMD	$P = 0.508$	$P = 0.870$	$P < 0.001$	

Bold indicates statistical significance ($P < 0.05$).

(32) respectively. The observed differences are probably due to the different population of patients investigated, as the mentioned studies included student and general population, respectively, while the patients in our control group were selected among patients seeking prosthodontic treatment. It is interesting that frequency of TMD, in our study, in the control group (42% of patients) is similar to that observed in psychiatric patients on antidepressive therapy (47% of patients). Concerning the fact that the subjects in both investigated groups exhibited an absence of occlusal disturbances according to the Helkimo Index criteria and the fact that patients from control group seek aesthetic prosthodontic treatment excluding any occlusal adjustment; it can be considered that our investigated groups could be similar in respect to occlusal conditions.

Concerning certain TMD subtypes, we found significantly higher prevalence of joint subtype disorders, including disc displacement and other joint conditions in the psychiatric group of patients than in the control group. However, myogenic subtype of TMD was observed only in one patient in the psychiatric group in contrast to five patients within the control group. The observed difference was not statistically significant, as the number of patients suffering from muscle related TMD in the psychiatric group was small compared with that of the control group. The small sample size in this study limits generalization of the findings, but these data could be supportive of diazepam-mediated myorelaxant effect. It could be supposed that this difference is the consequence of the diazepam-mediated myorelaxant effect, which camouflaged myogenic disorders, making the joint-related disorders more prominent (18, 19).

The present data obtained from Axis II concerning graded chronic pain and related disability in the control group of patients with TMD show that there was almost the same percentage (25%–31%) of patients with grade I, II, and III of chronic pain and related disability except IV grade which appears in 12.5%. These results are not in accordance with findings of Yap *et al.* (33) In their study, Yap *et al.* (33) presented that in the Asian population 78.5% of the patients showed grade I and II of chronic pain and related disability, while the grade III was present in 4–7% of the patients and grade IV was not observed. The observed distinction in the distribution of chronic pain and related disability between our study and the study of Yap *et al.* (33) could be related to the different age of patients, since the patients' age range in study of Yap *et al.* (33) from 12 to 64 years, while patients selected for our study aged from 20 to 43 years. It is well known that the signs and symptoms of TMD show the peak prevalence in young adults from 20 to 40 years of age (34). Additionally, different population of patients were investigated. Namely, population in the study of Yap *et al.* (33) was general, while the patients in our control group were selected among patients who were seeking prosthodontic treatment.

Concerning the grade of chronic pain and related disability in TMD psychiatric patients on antidepressive therapy, the obtained results showed that most of these patients (64.3%) expressed only the grade I of chronic pain and related disability, and the rest of the patients (35.7%) expressed the grade II of chronic pain and related disability. It could be supposed that such reduction of chronic pain score in this group of patients was because of the effect of tetracyclic antidepressant-maprotiline. There are data concerning that maprotiline, newer antidepressant, effectively reduce chronic pain in patients with chronic tension headache in dose range of 25–75 mg (15) and low back pain in dose of 150 mg daily (16). There is no evidence about analgesic effect of maprotiline in chronic TMD pain. Our study revealed for the first time that maprotiline in antidepressive doses, 75 mg daily, could be effective for such pain. On the other hand, it is known that classical tricyclic antidepressive drugs, such as amitriptyline, are successfully used for the treatment of chronic orofacial pain in doses smaller than their antidepressive doses (11, 35). It is noteworthy to emphasize that Plesh *et al.* (11) reported that long term, 1 year low-dose amitriptyline (10–30 mg) treatment in patients with chronic

TMD pain showed significant reduction for pain scores, but in comparison with 6 weeks treatment, pain and global treatment effectiveness were less improved at 1 year than at 6 weeks.

Besides, the observed high rate of the grade I of chronic pain and related disability in psychiatric patients on antidepressants could also be related to the fact that diazepam was included in their therapy, as results of other investigations suggested that medication which aid in muscle relaxation and sedation may be effective for treating the symptoms of masticatory pain of myogenic origin (36). Medications that do not provide relaxation of the muscles may not offer adequate pain relief under TMD conditions (36).

The present findings show that masseter silent period was prolonged in the control group of patients with different subtypes of TMD. Stimulation of the trigeminal nerve fibres elicits suppression of the voluntary contraction in the human masseter and temporalis muscles and this reflex has been called silent period or exteroceptive suppression (37). Although silent period in human jaw-closing muscles have been extensively studied, the physiological modulation of this reflex is still not fully understood. It was originally reported that patients with TMD and tension-type headaches had significantly changes silent period responses (38, 39). The underlining mechanism was speculated to be hyperactivity of the central nervous system and abnormal cortical or reticular activity that would enhance the excitability of the trigeminal motor neurons through the modulation of the multisynaptic reflexes (40). The fact that, the masseter silent period in the group of psychiatric patients with TMD was not prolonged, and, was similar to that in the subjects without TMD, could be related to the fact that increased muscle activity was reduced by myorelaxant effect of diazepam included in the antidepressive therapy protocol. Namely, the miorelaxant effect of diazepam is a result of inhibition of polysynaptic motor neurons in brain-stem through activation of GABA (γ -aminobutyric acid) receptor subtype (41).

Although we did not observe gender differences concerning the silent period, it is interesting to note that such differences were previously described. Kossioni and Karkazis (42) reported that the amplitude of jaw-jerk reflex was significantly higher in women than in men while Komiyama *et al.* (43) reported that women have a lower reflex threshold and pain threshold to cutaneous electrical stimulation than men.

Skiba and Laskin (21) analysing masticatory muscles silent period in patients with myofascial pain dysfunction syndrome, suggested that there was the direct relationship between prolongation of temporalis and masseter silent period and severity of symptoms in this syndrome. Liu *et al.* (22) conducting EMG examination of jaw muscles in patients with TMD, observed that muscle and joint pain were positively associated with duration of silent period of masseter muscle. Results in our study also revealed a significant correlation between the grade of chronic pain and related disability and the duration of masseter silent period in the control group of patients with TMD.

Conclusions

Our results provide initial data suggesting differences in TMD characteristics between non-psychiatric patients seeking prosthodontic treatment (control group) and hospitalized psychiatric patients on antidepressive therapy protocol (tetracyclic antidepressant-maprotiline and anxiolytic-diazepam) purposely checked for TMD. It could be concluded that the psychiatric patients on antidepressive therapy exhibited TMD with the frequency of appearance similar to that in the control group of non-psychiatric patients but with higher prevalence of joint related TMD, lower grade of chronic pain with related disability and without prolongation of masseter silent period with respect to control group.

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