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EMG Activity of Chewing Muscles in Dysfunctional Disorders of Temporomandibular Joints

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Electromyograms were recorded from the main chewing muscles (*mm. masseter* and *temporales*) in 184 patients suffering from dysfunction of the temporomandibular joints and 25 healthy subjects (controls). In the patient group, the mean duration of the silence phase in cyclic EMG activity of the above muscles related to normal chewing (293.8 msec, a value averaged for all examined muscles and all examined patients) was by about 30% smaller than the analogous value in the control group. At the same time, the mean duration of the activity phase did not differ significantly from that in the control. The ratio of durations of the activity vs. silence phases in the patient group was 1.352, as compared with 0.946 in control subjects. The mean duration of the chewing cycle (also totally averaged for the patients) was 691.1 sec vs. 814.4 msec in the control; the respective mean values of the chewing frequency in the above groups were 1.447 and 1.228 sec^{-1}. The results obtained confirm the statement that parameters of functioning of the brainstem neuronal central pattern generator (CPG), a neuronal network responsible for generation of cyclic motor commands for chewing muscles, can be significantly modified under the influence of a changed afferent inflow, especially in the case where the latter contains a considerable nociceptive component related to the dysfunctional state of the temporomandibular joints.

Keywords: chewing, brainstem central pattern generator, cyclic EMG activity, *mm. masseter*, *mm. temporales*.

INTRODUCTION

Chewing is one of the crucial vitally important motor phenomena in most mammals, including humans. Significant disorders of this process result in dramatic deterioration of the quality of life. Dysfunction of the temporomandibular joints (TMJs) is a rather frequent reason for such disorders. Effective diagnostics and treatment of this pathology is an urgent problem in modern stomatology. Such pathology is widespread, and indices of the respective morbidity are at present constantly rising. Dysfunctions of the TMJs are characterized by variable clinical manifestations, and its diagnosis frequently meets considerable difficulties [1].

According to the published data, significant pathology of the TMJs occupies the third position after cases of caries and diseases of the periodontium. Its more or less expressed manifestations can be met in about 20% of children and adolescents and in nearly 80% of adults [2]. This pathology is characterized by a long symptom-free course and absence of organic changes within early stages of the disease (at both clinical and X-ray examinations) [2, 3].

Opinions on the pathogenesis of dysfunctional sates of the TMJs are controversial. It was believed that this pathology is initiated due to primary changes in the neuromuscular component of the dental and maxillary/mandibular apparatus [4]. At the same time, it seems that special attention should be paid to a nociceptive component in the pathogenesis of this disorder. Pain is the main complaint in the respective cases; its intensity may vary significantly but is reported by 86.7% of patients, on average [5, 6].

Examination of EMG correlates of TMJ dysfunctions has begun [7–9], but the accessible data are rather limited. Modifications of the chewing rhythm related to this pathology has attracted limited attention; as is known, this rhythm is, to a great extent, determined by the activity of the

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brainstem neuronal central pattern generator (CPG) of chewing. The activity of the latter determines the main rhythm and intensity of the chewing process; this activity is addressed to the brainstem motor nuclei of the muscles involved in the chewing process, and efferent activities of the respective motoneurons evoke cyclic contractions of these muscles.

Electromyographic examination of the respective contingent of patients may provide clinicians with significant assistance in diagnostics and with detailed information on the state of the maxillary/mandibular apparatus and efficiency of treatment. This is why we examined changes in EMG activity of the main chewing muscles in a group of patients suffering from dysfunction of the TMJs.

METHODS

Examined groups. In total, we examined 184 patients treated in the Department of Prosthetic Dentistry of the Bogomolets National Medical University (Kyiv). Among them, there were 124 women and 60 men (age from 16 to 65 years) with a clinically estimated diagnosis of dysfunction of the TMJs; in most patients, this diagnosis was confirmed by the data of X-ray examination. In all patients, the pathology was bilateral, but it was possible in nearly all cases to identify the joint impaired to a greater extent (according to the patient's report and clinical manifestations). The total group was subdivided into three subgroups according to values of the modified Helkimo index (h) widely accepted in dental clinics. Eighty-six subjects demonstrated a low level of dysfunctional modifications (h = 1–5), in 52 subjects, the pathology pattern corresponded to a medium level (h = 6–15), while a high level of dysfunction (h = 16–25) was observed in the third subgroup (n = 46). The respective patients were classified as belonging to clinical subgroups 1, 2, and 3, respectively.

For comparison, a control group was formed. It included 25 subjects (14 women and 11 men) of comparable age range, with intact dental rows, a normal state of the dental cavity, and no dysfunctional changes in the TMJs according to the data of clinical (h = 0) and X-ray examinations.

EMG examination of the chewing muscles was carried out using an 8-channel computerized electroneuromyographic complex, BioEMG-III (BioRESEARCH Assoc., USA). This complex, having the respective technical characteristics and software, provides extensive possibilities for recording a variety of electroneurographic and electromyographic phenomena in humans; in particular, its frequency range corresponds to 0.5 Hz to 20 kHz.

Recording of EMGs was performed using a generally accepted technique of EMG examinations, in a soundproof room (temperature 20–23°C). The examined subject was in a semihorizontal position in a comfortable armchair. First, motor points of the muscles involved in the chewing process were identified by palpation under conditions of maximum teeth compression. After degreasing of the skin above the respective points, standard bipolar BioFLEX electrodes (BioResearch, USA) were fixed on the above points using an adhesive tape; a grounding electrode was on the wrist of the examined subject.

A standard testing procedure included recording of EMGs in the state of relaxation of the chewing and other face muscles, during maximum voluntary contraction of the chewing muscles, in the course of the normal voluntary chewing, and during unilateral chewing [10]. In the examination of voluntary chewing, we used a standard stimulating food object, hazelnut kernels.

Statistical analysis of the obtained numerical data was carried out using standard statistical approaches with the help of Microsoft Office Excel 2010 (Microsoft Corp., USA) and a program WinPEPI 11.45 module COMPARE2 (J. H. Abramson).

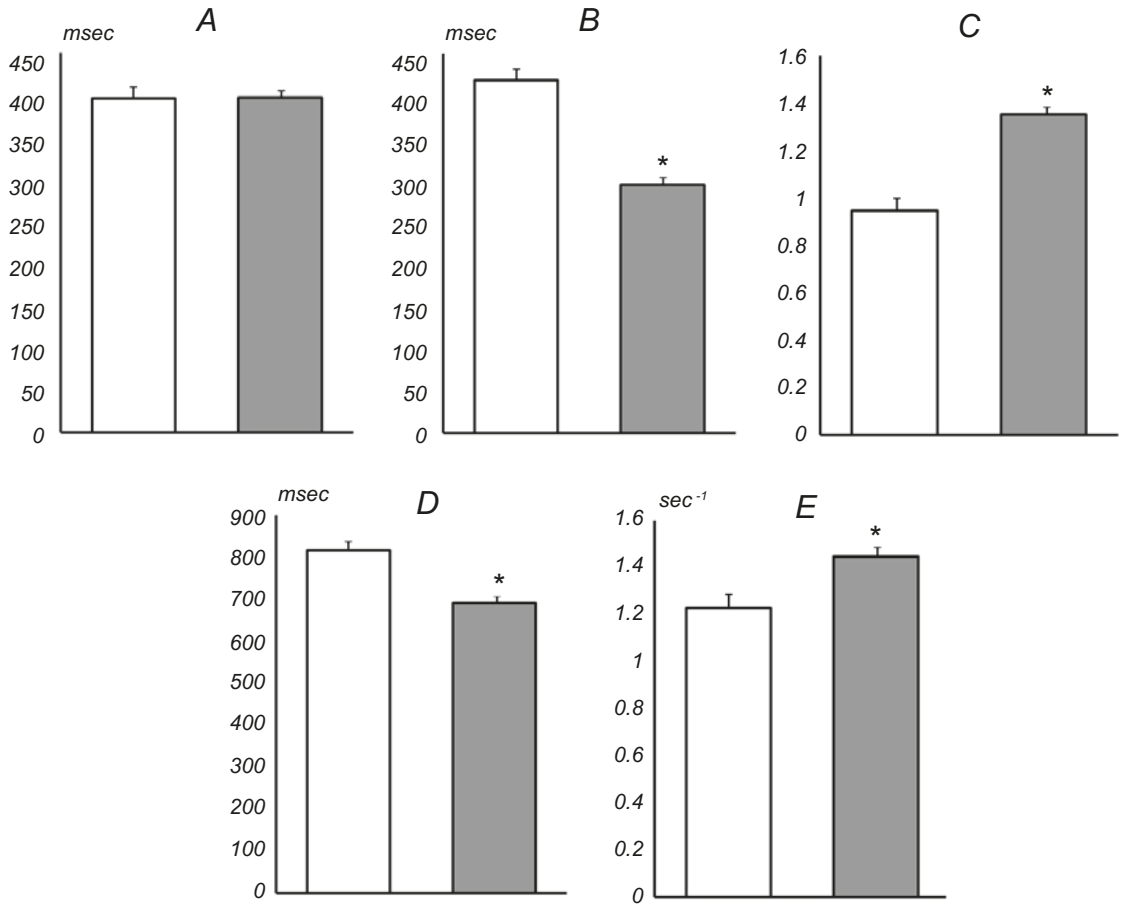
RESULTS

Chewing-related EMG activity in subjects of the control group. As in our previous study, most attention was paid to the activity of the main muscles involved in the chewing act, right and left *mm. masseter* and *temporales* (*Md*, *Ms*, *Td*, and *Ts*, respectively). At normal voluntary chewing, mean durations of the activity phases in the four above muscles varied within the entire control group between 392 to 400 msec. The respective variations of the silence phase were 415 to 422 msec. The averaged values of the above indices for the control group were 396.1 ± 8.4 and 418.4 ± ± 7.3 msec, respectively (Fig. 1 A, B). After averaging the durations of all the above indices for all muscles and for all examined control subjects, it was found that the mean duration of the entire chewing cycle in the control group was 814.5 ±

± 8.2 msec (Fig. 1 C), which gives an average frequency of normal chewing in this group equal to 1.228 ± 0.007 sec^{-1} (Fig. 1 E). The above-mentioned parameters of the chewing process in the control group examined in this study did not significantly differ from the corresponding indices in the control group of our previous study [10] ($P > 0.05$).

It should be taken into account that in the latter study [10] we calculated the ratio between durations of the silence vs. activity phases of the chewing cycle ($k_{s/a}$). In the present work, however, we calculated an inverse value, i.e., the ratio between durations of the activity vs. silence phases ($k_{a/s}$) as an index more routinely used in the analogous studies. In the present study, the mean value of this coefficient, after the above-mentioned total averaging of the respective data, was found to be equal to 0.947 ± 0.008 (Fig. 1 C). Thus, durations of the activity and silence phases at normal chewing performed by control subjects are nearly equal to each other (the activity phase is, on average, slightly shorter).

Chewing-related EMG activity in the group of patients suffering from dysfunction of the TMJs (**group dTMJ**). In this group, EMG activity in all patients performing standard chewing was also characterized by a clear cyclic pattern with easily distinguishable activity and silence phases. The activity phase, similarly to that in the control group, was also spindle-like, but with some specificities mentioned below. It should be mentioned that, in a noticeable part of the examined patients, some spontaneous oscillations were observed within the silence phase. These oscillations were of low



F i g. 1. Diagrams of values of the duration of the active phase in the chewing cycle (msec, panel A), duration of the silence phase (msec, panel B), ratio between the durations of the above phases ($k_{a/s}$, panel C), duration of the single chewing cycle (msec, panel D), and frequency of chewing (sec^{-1}, panel E) according to EMGs recorded from the main chewing muscles. The respective values were averaged for all four tested muscles (right and left *mm. masseter* and *temporales*) and for all subjects of the examined groups, the control group (open columns, $n = 25$), and that of patients suffering from dysfunction of the temporomandibular joints (filled columns, $n = 184$). Asterisks indicate cases of significant difference between the examined groups ($P < 0.05$).

amplitude (usually below 0.5 μV) but, nonetheless, readily distinguishable in 15 (17.4%) of the subjects of clinical subgroup 1 (with relatively weak manifestations of dysfunction of the TMJs. In patients of subgroups 2 and 3, the above spontaneous EMG oscillations were observed more frequently, in 26 and 32 subjects (50.1% and 69.6%), respectively.

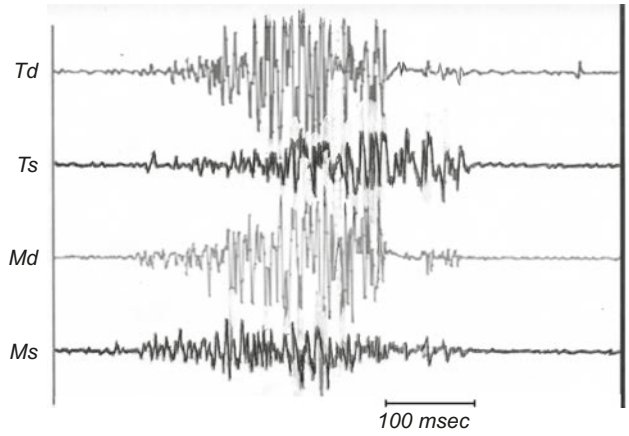
The temporal characteristics of chewing-related cyclic EMG activity in the dTMJ group demonstrated certain significant differences from those in the control group. First, the mean durations of the silence phase in the four examined main chewing muscles in dTMJ patients were significantly smaller than the respective values in the control. For the four above muscles (Md, Ms, Td, and Ts) the mean values of this phase were 299 ± 6, 294 ± 5, 291 ± ± 3, and 291 ± 6 msec, respectively. Averaging of these values gave the mean value of the silence phase duration of 293.8 ± 5.1 msec, which is only about 70% of the corresponding index in the control group (396.1 ± 8.1 msec; Fig. 1 B). In clinical subgroups 1–3, the durations of the silence phase showed no significant dependence on the intensity of manifestations of TMJ dysfunction.

At the same time, the durations of the activity phase in the dTMJ group demonstrated only slight differences from those in the control. The corresponding values in the patient group were 396 ± 9, 392 ± 4, 402 ± 3, and 402 ± 5 msec with a mean value for all muscles of 398.3 ± 5.2 msec (Fig. 1 A). For comparison, in the control group the analogous values varied between 413 and 422 msec, with an averaged total value of 418.5 ± 5.4 msec. There was a slight trend toward an increase in the duration of the activity phase in clinical subgroups 1–3 of the patients, but the respective differences were statistically insignificant ($P > 0.05$).

For the four above-mentioned muscles, the mean durations of the chewing cycle (summed mean durations of the activity and silence phases) were 694, 686, 693, and 692 msec. These values gave an averaged total value for the above-mentioned parameter of chewing of 691.2 ± 5.2 msec, which is only 84.8% of the respective value in the control group ($P < 0.05$; Fig. 1 D). Naturally, the respective differences were also found between the inverse parameters, i.e., mean frequencies of the chewing cycle. In the dTMJ group, this index was equal to 1.447 ± 0.008 sec^{-1}, in comparison with 1.228 sec^{-1} in the control (a 17.8% difference, $P < 0.05$; Fig. 1 E). In other words, patients suffering from dysfunction of the TMJ demonstrate a significantly shorter average duration of the chewing cycle than in the norm and, thus, perform chewing with a higher frequency. These specific features were associated with much greater values of the ratio between durations of the activity and silence phases ($k_{a/s}$) than those in the control group. As was mentioned below, the averaged value of this coefficient in the latter group was 0.946. In four examined chewing muscles of the patients with TMJ dysfunction, these coefficients varied from 1.321 to 1.381, and the averaged value of this parameter was 1.352 ± ± 0.007 (143% in comparison with the control; Fig. 1 E). Thus, relative durations of the activity phase of chewing cycles in relation to the silence phase were significantly longer ($P < 0.05$) than those in the norm.

Amplitude characteristics of EMG oscillations in the chewing cycles were not specially analyzed in this our study because we concentrated on the time/phase relations in these cycles. Nonetheless, we would like to mention two clear specificities of the amplitude EEG characteristics. First, noticeable deviations from a spindle-like pattern of the activity phase were observed in dTMJ patients much more frequently than in the control group. In the latter group, relatively even amplitudes of oscillations within a rather long central portion of the respective discharges were typical (see [10]). In patients, however, relatively high-amplitude oscillations were in many cases concentrated within a central part of the activity phase, while oscillations within the beginning and terminal parts of the above phase were rather low-amplitude (Fig. 2). In other words, the activity phases lost, to a certain extent, their regular spindle-like form in many cases.

Another specific feature of chewing-related EMG discharges in the patient group was the following. As we have mentioned above, despite the usually bilateral mode of lesions of the TMJs, one of these joints was damaged in most cases to a greater extent (according to the reports of the patients on the intensity of pain sensations). In the overwhelming majority of such cases, the amplitudes of EMG oscillations within the activity phase were noticeably lower precisely at the side of stronger pain sensations (Fig. 2).



F i g. 2. A representative example of the active phase in cyclic activity of the main chewing muscles in the performance of normal chewing by a subject of subgroup 2 of the patient group with dysfunction of the temporomandibular joints. *Td*, *Ts*, *Md*, and *Ms* are the right (d) and left (s) *mm. temporales* and *masseter*, respectively. Black traces are records from the left muscles (*Ts* and *Ms*, pain sensations in this patient were stronger at the left side); gray traces are records from the right muscles with weaker pain sensations in the joint. Amplification is the same in all channels.

DISCUSSION

At present, it is generally accepted that cyclic chewing movements, a highly important motor phenomenon in the course of food consumption, are primarily controlled by a rhythmic process produced by a brainstem central pattern generator (CPG) whose activity is switched on and off by descending influences from higher nerve centers. From such aspect, chewing demonstrates certain similarity to other cyclic motor phenomena (various types of locomotion, scratching, etc.). The chewing CPG is based on a network of interconnected brainstem interneurons, which generates alternating spike trains finally sent to motor nuclei of the chewing muscles. Among those, *mm. masseter* and *temporales* play the main roles, while some other muscles, playing an accessory role, are also partly involved in the process of chewing. It is obvious that basic cyclic CPG activity undergoes certain modulatory influences coming, first of all, from the upper cerebral centers (probably, mostly from the neocortex) because chewing is a voluntarily regulated (initiated and terminated) rhythmic motor act. On the other hand, CPG activity is inevitably subjected to modulatory influences coming from the peripheral receptor apparatus. The components of the latter are proprioceptors of the chewing muscles, mechanoreceptors of the oral cavity, joint receptors, and, naturally, nociceptors localized in the above structures. The question of to what extent the activity of peripheral receptors can modify the CPG rhythm is, from certain aspects, disputable.

In our previous study [8, 10] we found that positioning of fixed dentures in the oral cavity, which inevitably leads to some distortion of neuromuscular relations in the dental and maxillary/mandibular apparatus and, consequently, to certain modifications of the afferent flow from mechanoreceptors of the oral cavity (in the broad sense of this term), induces noticeable changes in the time/phase parameters of the chewing rhythm. Such interventions lead to mild but considerable shortening of the active phase of the above cycle, while the silence phase undergoes rather small (or negligible at all) changes. The averaged frequency of chewing in the studied group of patients with dentures also slightly but noticeably increased. Such modulation remains obvious within a rather long time (several months after positioning of the dentures).

In the present study, it became obvious that dysfunctional states of the TMJs also induce significant changes in the time/phase characteristics of EMG cyclic activity related to "standard" chewing. It should be emphasized that these modifications are considerably greater than those induced by implantation of fixed dentures in the oral cavity. As was mentioned above, the mean duration of the silence phase in the chewing cycle (after total averaging of the data for all four examined muscles and for all examined patients with dysfunction of the TMJs) was about 30% smaller than that in the control group ($P < 0.05$). The respective shifts led to a dramatic increase in the ratio between durations of the active and silence phases of the cycle (an about 43% increment, as compared to the control; $P < 0.05$). This pattern differed principally from that observed under "fixed dentures" conditions. In the latter case, the activity phase in the chewing cycle became noticeably shorter, while the silence phase was subjected to minimum (or even no) changes [10].

The total duration of the single chewing cycle was about 15% smaller than the analogous value in the control ($P < 0.05$). Correspondingly, the mean frequency of chewing in subjects with dysfunction of the TMJs was significantly greater (by about

18%, $P < 0.05$) than in subjects without such pathology. In other words, subjects suffering from problems with TMGs try to perform chewing with a higher frequency and with shorter intervals between the active phases.

Thus, the chewing cycle undergoes rather profound modifications under conditions of the above dysfunction. The latter conditions differ from those related to fixed dentures by the presence of a new significant pathogenetic factor, a considerably increased inflow of nociceptive influences, and this is the main probable reason for the above stronger modifications. Naturally, the results of this study confirm the statement that activity of the brainstem neuronal CPG of chewing (a relatively "autonomous" neuronal brain system capable of generating its "own" cyclic activity) may undergo significant modulation by influences from the peripheral receptor apparatus.

In this study, we have not concentrated on the amplitude characteristics of chewing-related EMG activity. Nonetheless, we should mention two phenomena related to this aspect. First, dysfunction of the TMJs induces in many cases noticeable rearrangements of the active phase in the chewing cycle (distortions of a regular spindle-like pattern of the respective EMG bursts). Maximum EMG oscillations are frequently grouped within a central portion of the above phase, while the beginning and final portions of this phase were of low amplitude. Second, there was a clear tendency for a lower amplitude of chewing-related EMG discharges in pairs of the main chewing muscles at the side of stronger pain sensations in the injured TMJ, and also a tendency for the development of unilateral chewing. The latter tendency clearly correlated with the severity of TMJ dysfunction, reaching about 100% in the third clinical subgroup. It is obvious that this phenomenon is of a defensive nature (prevention of strong pain sensations). The question of where the respective inhibitory process is localized (whether it is organized at the central, i.e., CPG, level, or inhibition develops at the level of the motor nuclei of the chewing muscle) probably needs special investigation in the further studies.

The study was conducted in accordance with the existing international ethical principles (Helsinki declaration) and approved by the Ethical Commission of the Bogomolets National Medical University. Written consent was obtained from all persons involved in the study.

The authors of this paper, T. M. Kostiuk, A. A.Kaniura, I. A. Shinchukovskiy, A. V. Tsyzh, and N. I. Medvinska, declare the absence of any conflicts regarding commercial or financial relationships with organizations or individuals who may be involved in the study, as well as conflicts between the co-authors.

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