Neurophysiologic evaluation of the temporomandibular joint and related masticatory muscles in rheumatoid arthritis patients

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ABSTRACT

الأهداف: دراسة تأثير التهاب المفاصل الرثوى على المفصل الفكي الصدغي و العضلات الماضغة باستعمال فحوص الفسلجة العصبية و المفراس الحلزوني.

الطريقة: اجريت هذه الداسة في مستشفى الجراحة المتخصص – بغداد – العراق، في الفترة مابين فبراير 2006م وحتى سبتمبر 2006م. تمت دراسة عينة تتكون من (42) مريض مصابا بالتهاب المفاصل الرثوي، وعينة اخرى من (30) شخص سويا متقاربين في العمر. تم فحص المرضى سريريا و خضع (37) منهم لفحص المفراس الحلزوني للمفصل الفكي الصدغي. كما تم قياس النشاط الكهربائي الفسيولوجي (تحليل طيف القدرة، وتخطيط العضلات الاعتيادي، بالإضافة إلى دراسة استجابة طرف العين الانعكاسي) لخمسة وعشرون منهم.

النتائج: بينت نتائج فحص المفراس الحلزوني وجود تآكل، وزوائد عظمية، وتسطح في لقمة العظم المفصلي، بالإضافة إلى صغر الفراغ المفصلي. تبين لنا في التخطيط الكهروعضلي الاعتيادي أن هنالك اختزال في طراز التداخل وأن الوحدات الحركية ذات أمد عالي ومدة واسعة و متعددة الأطوار. تحليل طيف القدرة بين أن هناك نقص مهم في متوسط تردد القدرة و متوسط الجذر التربيعي للفولتية (الجهد) في مجموعة المرضى. بينت دراسة استجابة طرف العين الانعكاسي أن هناك تأخر مهم في فترات الكمون عند تسجيل الفعالية الكهروعضلية لمكوني الاستجابة المبكر والمتأخر. كانت نتائج فحوص الفسلجة العصبية أكثر تأثراً في المرضى الذين بين فحص المفراس الحلزوني أن المفصل الفكي الصدغي لديهم اظهر علامات ايجابية من الذين كانت النتائج عندهم سلبية.

خاتمة: إن العضلات الماضغة للمفصل الفكي الصدغي في مرضى التهاب المفاصل الرثوي ضعيفة بسبب اعتلال الفرع الحركي للعصب الثلاثي التوائم، وهذا الضعف انعكس بصورة ملحوظة على فحوص الفسلجة العصبية خصوصا عند ظهور علامات تأثر المفصل الفكي الصدغي في فحص المفراس الحلزوني. إن فحص متوسط الجذر التربيعي للفولتية (الجهد) كان ايجابيا في جميع المرضى مما يجعله الفحص الأنسب لتشخيص اعتلال العضلات الماضغة لمفصل الفك الصدغي.

Objectives: To study the effect of rheumatoid arthritis (RA) on the temporomandibular joints (TMJ) and related muscles using CT scan and neurophysiologic tests.

Methods: Forty-two RA patients referred from the Maxillofacial Clinic at the Special Surgeries Hospital, Medical City, Baghdad, Iraq from February 2006 to September 2006 were included in this study. Thirty-seven of them underwent CT scan of the TMJ and 25 of these patients were neurophysiologically examined. The data were compared to 30 age-matched control subjects.

Results: Fifteen patients showed normal TMJ, whereas, abnormal TMJ on CT scan was present in 22 patients. Of these 22 patients, 6 showed decrease in the intra-articular space, 6 exhibited erosion of the condylar head, and 3 had flattening of the condylar head. The remaining 7 patients had all the abnormalities present. Electromyography (EMG) examination showed reduced interference pattern, poor recruitment of motor unit potentials, shift of the power spectra to the lower frequencies, low mean power frequency, and root mean square values, and prolonged blink reflex component latencies.

Conclusion: Rheumatoid arthritis patients with positive CT scan findings have poorer neurophysiologic data than those without CT scan detectable lesions. Trigeminal motor neuropathy is suggested to be the cause of the masticatory muscle weakness. Root mean square voltage as a parameter of the EMG power spectra is of great value in diagnosing such weakness.

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Rheumatoid arthritis (RA) is a chronic disease affecting the musculo-skeletal connective tissue of the body with a strong predilection for the joints. The temporomandibular joint (TMJ) is commonly affected by RA in adults and children, however, it is usually among the last joints to be affected.[1-3] Evaluation of masticatory muscle activity by conventional electromyography (EMG) is a valuable tool for diagnosing dysfunction of the masticatory apparatus. However, controversy exists with regard to the usefulness of EMG for patients with TMJ disorders.[4] The EMG can also be analyzed by frequency domain to generate the power spectral density and power spectra of the signal.[5] Frequency analysis of the EMG has been studied extensively in dental research, yet, studies concerned with masticatory muscles in RA are scarce. Trigeminal neuropathy and blink reflex (BR) have been studied in connective tissue diseases,[6-8] however, BR has not been studied extensively, specifically in RA.[6,9-12] The objective of our study was to investigate the effect of RA on the trigeminal system by CT scan and various electrophysiological parameters. In addition, we aimed to understand the possible causes of the masticatory muscle weakness in such patients.

Methods. Forty-five RA patients diagnosed according to the criteria of the American Rheumatism Association (ARA),[13] were referred from the Maxillofacial Clinic at the Special Surgeries Hospital, Medical City, Baghdad, Iraq. The study spanned over a period of 6 months, from February 2006 to September 2006. Each patient was informed regarding the purpose of this study, and that it would include questionnaires, clinical, and CT scan examinations, and EMG for masseter and anterior temporal muscles on both sides, and their consent was obtained. The study was approved by the local Ethical Committee of Baghdad University. Three RA patients were excluded as they had juvenile RA. The remaining 42 patients were 37 females and 5 males. The disease' duration varied from 1-38 years with a mean of 11.76±10.72 years. The study also included 30 control subjects (23 females and 7 males) with a mean age of 43.7±10.42 years. They were free from any neurological and systemic diseases. Thirty-seven out of the total of 42 were subjected to TMJ CT scan. Thirty of them were neurophysiologically examined, their age range was between 21-70 years (mean±SD = 46.03±13.82 years), whereas, only 25 RA patients out of the total of 42 were subjected to both CT scan, and EMG study. This is due to the reason that some patients skipped some of the tests, and others discontinued the study. Replies to a questionnaire covering oral, and TMJ symptoms were analyzed in conjunction with medical histories, and the results of clinical examination of the stomatognathic system. The patients were questioned for history of pain and aching on movement, or biting. A clinical examination was performed, including maximum voluntary mouth opening (inter-incisor distance), TMJ (clicking and crepitus), tenderness to digital palpation in the masticatory muscles, and maximum voluntary bite force. In addition, we looked for the presence of swelling and stiffness (if any), in the right or left TMJ and the duration of their presence, and finally any deviations or excursion of the mandible were also recorded. Blood samples were collected to measure the level of C-reactive protein (CRP) in mg/dl, the erythrocyte sedimentation rate (ESR) in mm/hour and rheumatoid factor (RF). Temporomandibular joint coronal slices were obtained by Somatom plus-4 CT scan (Siemens, Munich, Germany) . Dantec counter point 4-channel electromyography (Copenhagen, Denmark) was used for the electrophysiological tests, which include conventional EMG, power spectral analysis, and blink reflex. After instructing the subjects to clench, EMG of the masseter and anterior temporal muscles were studied using concentric needle electrodes Dantec 13L50. The ground electrode Dantec 13S93 was applied to the neck. The duration and amplitude of 20 motor unit potentials (MUPs) were taken from each muscle for the analysis. To study the power spectrum analysis, the electrical activity was automatically analyzed using an anti-aliasing filter, and fast Fourier transformation. From each scan-averaged spectrum, mean power frequency (MPF), and root mean square (RMS) voltage were analyzed from 20 records of each muscle. Blink reflex was obtained using self adhesive disposable electrodes Dantec C13L20 by the standard method[14] through applying an electrical stimuli of optimal intensity that elicited nearly stable responses with repeated trials using bipolar stimulating electrode Dantec 13L36. The electromyographic settings were 5 millisecond/division sweep speed, bandwidth of 8 Hertz-8 Kilohertz, and sensitivity of 500 microvolt/division. The recorded parameters were R1 component latency, ipsilateral R2 component latency (iR2) and contralateral R2 component latency (cR2). Blink reflex recording were repeated 3 times for each side, and the average latencies were taken for the study.

Statistical analysis. The results were expressed as mean±SD, and the statistical significance of the difference in the prevalence of abnormal values were tested by the student t-test. The percent of abnormal values in any test was calculated as mean±2.5 SD of the normal values for the control groups. Correlation between continuous variables was measured using Person's r-correlation coefficient. A probability limit of less than 0.05 was considered statistically significant. The Statistical Package for Social Sciences was utilized for the statistical analysis.

Results. *Clinical data.* Seventeen patients (40.4%) presented with pain in the region of TMJ, and feeling

of stiffness of the jaws on awaking, and they were the most common symptoms. The pain and tenderness on chewing were recorded in 8 (19%) patients, and difficulty in opening the mouth widely was noticed in 6 (14.2%) patients. Pain during maximum mouth opening and tenderness to digital palpation were correlated to difficulties with several activities such as yawning and opening the mouth wide. Temporomandibular sounds during condylar movement was present in 11 (26.1%) patients, and 19 patients (45%) gave history of swelling in the region of TMJ, especially during the acute phase of the illness which had subsided on taking their medication. The mean±SD disease activity scoring (DAS) for 28 joints was 3.24±1.35. Activities of daily living were influenced in all patients at different levels. The DAS values in patients with early RA (below 36 months [3.73±0.69], $p<0.05$), were significantly higher than in patients with advanced RA (3.29±0.79, $p<0.05$). A strong relationship between disease activity and functional disability was also observed (r=0.91; $p=0.0001$).

Laboratory testing. High concentration of serum acute-phase reactants, and high values of rheumatologic indices were correlated with the severity of TMJ involvement in RA.

CT scan data. Fifteen RA patients out of the 37 who were subjected to CT scan of the TMJ (40.6%) showed normal TMJ, whereas 22 patients (59.4%) showed abnormal TMJ. Fifteen patients with abnormal TMJ (68.2%) showed bilateral involvement of TMJ, and the remaining 7 (31.2%) showed unilateral involvement. Of the 22 patients with abnormal TMJ, 6 patients (27.3%) showed a decrease in the intra-articular space. Another 6 patients (27.3%) showed erosion of the condylar head, whereas 3 patients (13.6%) had flattening of the condylar head. The remaining 7 patients (31.8%) had all the mentioned abnormalities on the CT scan of their TMJ. There was a significantly positive correlation (r=0.87, $p<0.001$) between the duration of illness, and TMJ involvement.

Neurophysiologic data. On EMG examination, a reduced interference pattern, and poor recruitment of MUP were recorded in the RA patients, moreover, many MUPs were polyphasic, with wide duration and high amplitude. **Table 1** shows the mean amplitude and duration values of masseter, and anterior temporal muscles of both sides. The mean amplitude and duration values of the masseter were significantly higher and the anterior temporal muscles of both sides were wider in the RA patient group, in comparison to the control values. Regarding the data of power spectral analysis, it was found that the MPF and RMS voltage values of the control subjects were higher in the masseter, than in the anterior temporal muscle. However, this difference was only significant for the MPF ($p=0.0018$). In RA patients, the MPF and RMS voltage values were significantly reduced as compared to control values (**Table 1**).

The latencies of R1, iR2 and cR2 of both eyes were significantly prolonged in RA patients when compared to control values (**Table 2**). Analyzing the data of RA patients who were submitted to CT scan and EMG study, it was clear (**Table 3**) that the mean amplitude values of left masseter ($p=0.0144$), and anterior temporal muscles were significantly lower ($p=0.039$) in the patients with a positive CT scan (arthritic changes). Significantly wider MUP duration values of right masseter ($p=0.0411$) and anterior temporal muscles ($p=0.0089$) were also seen in this subgroup (**Table 3**). The MPF was decreased in patients who showed arthritic changes on CT scan when the recording was carried out in the right masseter, right and left anterior temporal muscles. Yet, this decrement was not significant in the left masseter. The RMS voltage of the right anterior temporal, right and left masseter muscles decreased significantly, while, RMS

Table 1 - Illustrates the mean values of MUP duration, MUP amplitude, MPF, and RMS voltage recorded from right and left masseter, and anterior temporal muscles of both sides from rheumatoid arthritis patients and control group.

Variable	Control group (n=30)		Rheumatoid arthritis patients (n=25)			
	Masseter	Anterior temporal	Masseter		Anterior temporal	
			Right	Left	Right	Left
Duration (msec)	8.25±1.65	8.57±1.75	9.02±2.83*	9.8±2.3†	9.7±2.9‖	9.51±2.9‖
Amplitude (µV)	318.13±91.20	319.17±90.21	416.14±175.61‡	447.98±245.16§	511.9±200.1§	473.44±236.26§
MPF (Hz)	327.67±163.77	212.72±93.58	151.13±42.7§	173.8±35.3§	162.8±49.05¶	163.32±52.65**
RMS voltage (mV)	6.47±2.14	5.36±2.08	0.109±0.72§	0.13±0.12§	0.085±0.057§	0.091±0.052§

MUP - motor unit potential, msec = millisecond, µV = microvolt, MPF = mean power frequency, Hz = hertz, RMS = root mean square, mV = milli volt, *$p=0.0031$, †$p=0.0445$, ‡$p=0.0005$, §$p=0.0001$, ‖$p=0.0052$, ¶$p=0.0009$, **$p=0.0027$ (Rheumatoid arthritis patients versus control subjects)
The RA patients included were those undergoing neurophysiologic testing, and CT scan

Table 2 - Illustrates the difference of BR components between the RA patients and the control group.

Component	Control group (n=30)	RA patients (n=25)
Right Eye		
R_1 latency (msec)	10.81±1.1	11.7±1.79*
iR_2 latency (msec)	33.2±4.49	37.1±8.1†
cR_2 latency (msec)	34.62±3.28	36.98±8.1‡
Left Eye		
R_1 latency (msec)	10.75±1.1	12.9±2.3‡
iR_2 latency (msec)	33.21±4.43	38.3±6.7§
cR_2 latency (msec)	33.95±3.29	37.97±6.5‖

BR = blink reflex, RA = rheumatoid arthritis, R - reflex, i = ipsilateral, c = contralateral, msec = millisecond, *p=0.0067, †p=0.0014, ‡p=0.0001, §p=0.0174, ‖p=0.0003
The RA patients included were those undergoing neurophysiologic testing, and CT scan

Table 3 - Illustrates the mean amplitude and duration values of masseter and anterior temporal muscles at both sides between RA patient with and without TMJ involvement.

Muscles examined	Patients with normal TMJ (n=11)		Patients with RA involvement of TMJ (n=14)	
	Duration	Amplitude	Duration	Amplitude
Left masseter	8.6±1.6	581.9±312.8	8.93±1.64	408.04±161.58*
Right masseter	8.5±1.3	509.4±171	9.44±2.28†	393.51±16574
Left anterior temporal	7.09±2.2	598.75±271.07	7.3±1.6	399±183.98‡
Right anterior temporal	7.34±3.08	511.9±200.12	9.22±1.5§	472.18±196.05

RA = rheumatoid arthritis, TMJ = temporomandibular joint, *p=0.0144, †p=0.0411, ‡p=0.039, §p=0.0089
The RA patients included were those undergoing neurophysiologic testing, and CT scan

Table 4 - Illustrates the mean MPF and RMS voltage values of the masseter and anterior temporal muscles at both sides in RA patients with arthritic changes and normal TMJ.

Parameter	Muscle tested	RA patients (n=25)	
		With arthritic changes (n=14)	Normal TMJ (n=11)
MPF (Hz)	Right masseter	157±49.6	157.3±31.5
	Right anterior temporal	144.98±6074	176.3±38.2
	Left masseter	182.11±35.4	178.9±33.6
	Left anterior temporal	139.6±52.8	189.33±47.62
RMS voltage (mV)	Right masseter	0.077±0.041*	0.139±0.085
	Right anterior temporal	0.074±0.042†	0.11±0.072
	Left masseter	0.099±0.049‡	0.186±0.166
	Left anterior temporal	0.087±0.05	0.116±0.052

RA = rheumatoid arthritis, TMJ = temporomandibular joint, MPF = mean power frequency, Hz = hertz, RMS = root mean square, mV= milli volt, *p=0.0133, †p=0.0474, ‡p=0.0003. The RA patients included were those undergoing neurophysiologic testing, and CT scan

voltage of the left anterior temporal muscle showed a decrement that was not significant (**Table 4**). Blink reflex components did not show any significant difference between RA patients with normal TMJ, and those with arthritic changes on CT scan, with the exception of R1 latency of left eye which was significantly prolonged (p=0.0285) in those who showed arthritic change on CT scan (**Table 5**).

Diagnostic yield of neurophysiological tests. An increase in the MUP duration or amplitude of masseter, and anterior temporal muscles above 2.5 SD of the normal values was noticed in 17 patients (56.6%) out of 30 RA patients who were subjected to neurophysiological tests. Seven patients showed prolonged BR components increasing the diagnostic yield to 80%. Abnormal values of the MPF were present in another 4 patients, increasing the diagnostic yield to 93.3%. On the other hand, the RMS voltage was abnormal in all RA patients.

Discussion. Temporomandibular abnormalities on CT scan were found in 59.4% of the RA patients, which was 17-45% more than it was reported by other

Table 5 - Illustrates the mean BR component latencies values of RA patients with normal TMJ and those with arthritic changes.

Blink Reflex Component	RA patients (n=25)	
	RA patients with normal TMJ (n=11)	RA patients with arthritis changes (n=14)
Right Eye		
R_1 latency (msec)	10.5 ± 0.9	10.7 ± 0.8
iR_2 latency (msec)	36.5 ± 6.14	36.6 ± 9.9
cR_2 latency (msec)	35.8 ± 6.2	36.8 ± 9.6
Left Eye		
R_1 latency (msec)	10.6 ± 1.7	11.1 ± 0.96*
iR_2 latency (msec)	36.9 ± 6.96	39.63 ± 6.2
cR_2 latency (msec)	38.1 ± 7.01	36.8 ± 5.8

BR = blink reflex, RA = rheumatoid arthritis, TMJ = temporomandibular joint, i = ipsilateral, c = contralateral, msec = millisecond,*p<0.0285. The RA patients included were those undergoing neurophysiologic testing, and CT scan

researchers,[3,15] and less than 88.4-88.6% as reported by Bayar et al[2] and Goupille et al.[16] This could be attributed to the difference in sample dimension, and to the use of high resolution computerized tomography in the studies conducted by these 2 researchers. From the results of this study, it was noticed that there were patients who lack correlation between disease duration and TMJ involvement. For example, one patient with a history of 12 years of the disease duration displayed no CT scan changes. On the other hand, in 4 patients, there were CT scan changes in the TMJ after a disease duration of less than 7 years. For those cases with positive symptoms, and clinical findings suggestive of TMJ involvement, however with negative results on CT evaluation, it is possible that joint inflammation is indeed present although it still had not affected the structure of the joint. These cases occurred in patients with disease duration of less than 10 years (5.5±3.11 years), with the exception of one patient who had started RA 12 years before.

As it was previously noticed, the disease duration of those patients with positive CT scan changes is long (15.64±13.64 years). Thus, a long standing disease is generally needed to cause destructive lesions of the TMJ, and related apparatus. Goupille et al,[16] discovered positive correlation between the intensity of destructive lesions of TMJ on CT scan, and the severity of RA. This also could explain the data of those patients with abnormal CT scan in comparison to those with normal TMJ. The significantly delayed latencies of the BR in RA patients was in agreement with the findings of Soubrier et al,[7] and Abdul-Kareem et al.[12] Bilateral delay of the R2 components with unilateral stimulation suggest affection of the afferent pathway (trigeminal nerve of BR).[17] This trigeminal sensory neuropathy could be ascribed: to the generalized neuropathy associated with RA and involving trigeminal (first hypothesis) or facial ganglia or the nerve trunks, or from autoimmunity associated with connective tissue diseases (second hypothesis).[11] Based on postmortem studies of peripheral nerve biopsies,[11] there is the possibility that brain stem involvement by vasculitic process could eventually lead to vasculitic neuropathy of the cranial nerves probably through affection of their interneuronal connection in the brain stem. A third hypothesis would be the presence of rheumatoid nodules within brain tissues that could press on the trigeminal or facial nerves pathway thereby prolonging their latencies.[11] This is due to the fact that the trigeminal nerve and nuclei are unique in the human body, with regard to their anatomical and physiological characteristics. They are also special regarding the lesions in which they are involved, both at the peripheral level due to the susceptibility of some terminal branches, and at the nuclei due to their large size, and the large amount of connections with other centers.[18] The latter 2 possibilities needs to be further clarified and elucidated.

The reduced EMG interference pattern recorded in our patients could be due to trigeminal motor neuropathy, and coexistent weak denervated masticatory muscles. This is for the reason that the trigeminal nerve conducts both sensory and motor impulses,[19] and EMG of the masseter muscles provided an intuition on the function of the trigeminal motor roots.[20] The EMG interference pattern offers information regarding the number and size of the MUAPs, recruited at different levels of voluntary muscle activation.[21] In chronic or old peripheral neuropathies, the MUAPs exhibit high amplitude, and a wide duration as a result of continuous processes of denervation, reinnervation, and collateral sprouting. This affected the results of EMG power spectra recorded from the diseased masticatory musculature as there was a clear shift toward lower frequencies. The explanation for this shift is, in the first place, the power spectrum of the EMG signal is inversely related to the average durations of the MUAP.[22] Secondly, the MUs are activated according to their size, and the recruitment order is from small to large MUs.[23] Finally, the duration of MUAP seems to relate to the size of a MU,[24] thus the recruitment of larger MUs have an increased average duration of the MUAPs. Muscle force increase is produced by a higher motor neuron firing rate, and by recruitment of new motor units.[25] Hence, as speculated earlier, loss of many MUs from the masticatory musculature of the RA patients makes their muscles weaker with reduced capacity for clenching.[4] Eventually, few MUs are recruited in weaker musculature resulting in an ultimate decrease in their force and strength. Moreover, literature on RA has noted that the clinical weakness is so common

that it could be caused by muscle involvement by rheumatoid inflammation, diminution of muscle bulk due to atrophy,[26] and reflex weakness due to pain.[27] The aforementioned factors, in concert, cause weakness of the masticatory musculature in RA patients, and finally result in the decrease of MPF and RMS voltage values, which are the best representative of muscle strength.[28]

The MPF and RMS were lower in RA patients than in healthy controls, while the amplitude of the recruited MUPs was higher. The RMS was also lower among the patients with arthritic changes according to CT scan, and those with more bone destruction. The amplitude was, however, lower in patients with CT scan changes than those without, which might be explained by the fact that CT scan does not provide information on the current level of TMJ inflammation, and subsequently this level may be higher in the group without CT changes.

Further study should investigate the possibility that brain stem involvement by vasculitic process could eventually lead to vasculitic neuropathy of the cranial nerves, probably through affection of their interneuronal connection in the brain stem. In addition, to search for the possibility of the presence of rheumatoid nodules within brain tissues that could press on the trigeminal or facial nerves pathway , thereby prolonging latencies.

In conclusion, RA patients with abnormal TMJ on CT scan showed worsened neurophysiological data than those without. Masticatory muscle weakness could be attributed to trigeminal motor neuropathy, rather than the inflamed painful arthritic TMJ. As a whole, neurophysiologic parameters were better in diagnosing masticatory musculature affection than each one of them separately. The RMS is the best neurophysiologic parameters in diagnosing the masticatory musculature affection in RA.

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