A Study on the Changes in Motor Unit Action Potential, EMG Power Spectrum, and Pressure Pain Threshold of Masticatory Muscles during Sustained Fatiguing Contraction

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CONTENTS

Ⅰ. INTRODUCTION

Masticatory muscle disorder is frequently concurrent with chronic orofacial pain. It is classified into myofascial pain disorder, protective muscle splinting, myospasm, myositis, and muscle contracture. Muscle disorder can occur with or without joint disorder. Schiffman *et al.* reported epidemiologic study that 33% of the general population was involved with temporomandibular disorder and 41 % had masticatory muscle disorder[1)]. Muscle pain is the most frequent symptom in patients with masticatory muscle disorder.

Muscular fatigue originated from parafunction, postural muscle tension, and hyperactivity of the masticatory muscles. Muscular fatigue is known as a major cause of muscle pain in the orofacial region[2)]. Local muscular fatigue may be defined as a transient decrease in the capacity to perform work due to prior physical activity. Therefore muscle tenderness during palpation has been reported as the most common diagnostic tool of masticatory muscle disorder. The mechanism of muscle tenderness is clearly unknown. It is suspected that type III or IV nociceptors are sensitized to pressure stimuli or activated by local factors giving rise to an increase in nociceptive afferent fiber activity[3)]. The most common method to evaluate tenderness in the masticatory muscles is manual palpation but it is difficult to measure various levels of muscle tenderness. A patient's response is dependent on the strength of palpation and it is therefore difficult to objectively measure this response. As a tool of measurement, the pressure algometer is one of the reliable methods used to quantify changes in muscular pain threshold. This instrument is

therefore used in many studies as a means of evaluating muscle tenderness and of measuring the pressure pain threshold (PPT) in the orofacial region. It has been shown to be sensitive and reliable[4,5].

Another sensitive research tool for measuring muscular functions and conditions is the Electromyography (EMG). Moyers first introduced it into dental research[6]. Three types of electrodes: surface, needle and fine wire have been used for EMG examinations. The surface electrode is used to evaluate the overall activity of whole muscles. The needle electrode and fine wire electrode are used to evaluate the functions of deep muscles and the properties of motor units.

A motor unit is composed of the motor neuron, axon, and muscle fibers. It is an individually activated functional basic unit of the motor system, without any relation to an activating state of other motor units. Stålberg reported that the feature of motor unit action potential (MUAP) was determined by the structure of a motor unit, *i.e.* the number, size, distribution of muscle fibers in a motor unit and geometric form of end-plate zone[7]. Stålberg also reported that the structure of a motor unit could be changed by loss of muscle fibers and re-innervation[7]. The intensity of muscle contraction is determined by the number of recruited motor units, firing frequency of an individual motor unit, and the degree of resistance to fatigue during muscle contraction. The size, distribution and function of a motor unit can be changed by various physiologic factors and by diseases. Therefore, it can be possible to detect physiologic and pathologic changes of a motor unit by observing the changes of MUAP with EMG. There are a number of parameters for analyzing MUAP such as duration, amplitude, area, and phase. From the analysis of these parameters we can estimate the physiologic and pathologic changes in muscles.

Analysis of muscular fatigability induced by sustained muscle contraction can be also performed with power spectral analysis. The conversion of time-domained myoelectric signals into frequency-domained signals can be done with the aid of fast Fourier transformation[8]. Many authors reported that power spectrum of myoelectric signal was shifted to lower frequency range as muscular fatigue induced by sustained masticatory muscle contraction[8,9,10].

The relation between EMG activity and PPT has been investigated by several researchers. Fricton *et al.* reported that the motor unit electrical activity in trigger points was found to be significantly higher than that of the normal muscle[11]. Jensen reported that muscle tenderness and EMG-amplitude levels in tension-type headache patients were significantly increased over those of a control group[12]. He suggested one of the primary sources of pain in tension-type headache might be a local sensitization of nociceptors in the pericranial muscles. Sandrini *et al.* reported EMG and pressure pain threshold of patient with chronic tension-type headache were lower than those of normal population and patients with migraine[13].

The purpose of this study is to investigate the relationships between the level of pressure pain threshold and the parameters of MUAP in human masseter and the anterior temporal muscle. This study purposes to as well examine the changes in morphologic parameters of motor unit action potential, pressure pain threshold, and EMG power spectrum during experimentally induced acute muscular fatigue.

Ⅱ. MATERIALS AND METHODS

1. Subjects

Thirty-six subjects, 26 males (26.5±2.1 years old) and 10 females (23.8±2.7 years old), without past history and present symptoms of pain and discomfort in temporo-mandibular joints and masticatory muscles, with normal bilateral posterior occlusion were included in this study. Because there were no significant differences of myoelectric signal and PPT between right and left side of masticatory muscles in pilot study, MUAP, EMG power spectrum, and PPT were measured in the right

anterior temporal and masseter muscles before and after sustained isometric contraction.

For the estimation of the maximum voluntary contraction level (MVC), the subjects were asked to clench for 3 seconds three times with maximum forces in maximal intercuspal position. The highest force among the three trials was defined as the maximum voluntary contraction level. After 5 minutes rest, each subject performed a sustained clenching at 70 % MVC with visual feedback until endurance time when they could not maintain the level.

Simultaneous examination of MUAP and EMG power spectrum was difficult in anterior temporal muscle because of its small size, however it was possible in a masseter muscle. In the case of anterior temporal muscle, MUAP and power spectrum were analyzed on different days. PPTs were also measured with each analysis, respectively. Each muscle was analyzed at different days.

2. Pressure pain threshold

PPT measurement was performed with the Electric Algometer Type II (Somedic Production AB, Stockholm, Sweden). The algometer has 1 cm^2 circular probe. The subjects were seated and Frankfort planes were parallel to the floor. The force was applied perpendicularly to the anterior temporal and masseter muscle, and the increasing rate of force is maintained at 40 kPa/sec.

Each application was repeated twice and the mean values were obtained. The interval between the first and second pressure algometer application was at least five minutes. In order to avoid experimental bias neither the examiner nor the subjects could watch the digital display.

3. Motor unit action potential

The EMG equipment, Nicolet Viking IV electro-diagnostic system (Nicolet Biomedical Inc., U.S.A.) was used for recording myoelectric signals. This system can perform MUAP analysis automatically with a concentric needle electrode. The concentric needle electrode (Nicolet Biomedical Inc., U.S.A.) was inserted in the right masseter muscle, in the area of 1 cm superior and anterior to the angle of the mandible about 1 cm in depth. And the pre-gelled, silver/silver chloride surface electrodes (Nicolet Biomedical Inc., U.S.A.) were aligned in the direction of the muscle fibers as the same position which PPTs were measured. The ground electrode was installed on the skin of the neck of the subject just below the earlobe. Then the examiner let the subject generate a few motor units with minimum effort. As this effort measurably appeared on the displayed screen, the examiner carefully moved the needle electrode until it was inside the motor unit's territory as shown by the on-screen display.

Once the needle electrode was inserted into the motor unit territory a few MUAPs were recorded. Then the needle electrode was carefully repositioned to the other site in the tested muscle in order to record different MUAPs until the twenty different MUAPs were obtained in a tested muscle for the statistical analysis. MUAPs were recorded at a sensitivity of 100-200 μV per division. The low pass and high pass filter of amplifier set at 10 kHz and 2 Hz respectively for undistorted recording of MUAPs. Analysis was performed using a computer-assisted program and the MUAP features were quantitated. In recording MUAPs, the motor unit action potentials selected for assessment must

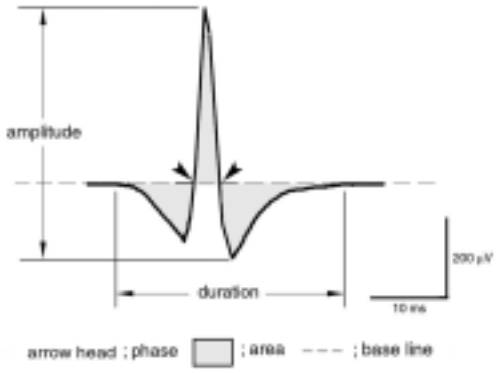

Fig. 1. Representative motor unit action potential waveform obtained from masseter muscle.

have a rise time less than 0.5 ms and an area value in range of mean ± 2 standard deviation among different twenty MUAPs. Fig. 1 shows a typical MUAP waveform with important parameters.

4. EMG power spectrum

Power spectral analysis of the EMG signal was performed by Nicolet Viking IV electrodiagnostic system. It determined the individual frequency components and their power contained within the EMG signal. Time-domained myoelectric signals could be transformed into frequency-domained signals. During the sustained isometric contraction, integrated EMG activity and the firing frequency from both anterior temporal and masseter muscles were recorded.

5. Statistical analysis

For each subject the mean values and standard error of means of each MUAP parameter of twenty MUAPs were calculated. All the statistical analyses were performed by SPSS/PC$^+$ program. Paired *t*-test was used to evaluate the difference between anterior temporal and masseter muscles and the difference of median frequency and pressure pain threshold between before and after sustained isometric contraction. Student *t*-test was performed to examine the gender difference. The slope of median frequencies was calculated by means of linear regression analysis to evaluate decline pattern of median frequencies during sustained isometric contraction of masticatory muscles.

The correlation between the PPTs and MUAP parameters and median frequency was investigated by Pearson correlation coefficients. Whenever statistical tests were performed they were two-tailed and at the 5 % significance level.

Ⅲ. RESULTS

During sustained fatiguing contraction the means and standard errors of pressure pain threshold (PPT) of both anterior temporal and masseter muscles are shown in Table 1 and Fig. 2.

Before sustained isometric contraction, PPTs in both anterior temporal and masseter muscle were significantly higher than those measured after sustained isometric contraction ($P < 0.001$). In the

Table 1. Pressure pain threshold (PPT) values before and after sustained isometric contraction. Data presented as mean ± standard error of means. Asterisks indicate statistically significant difference between before and after sustained isometric contraction within the same muscle (***: p<0.001). The 'a' indicates a statistically significant difference between anterior temporal and masseter muscle in either before or after sustained isometric contraction (aa: p<0.01). The 'b' indicates a statistically significant gender difference of PPT values (b: p<0.05).

			PPT-before(kPa)	PPT-after(kPa)	ΔPPT(kPa)
Male	(n=26)	Anterior Temporal	230.2±12.7	197.9±9.9	32.3±5.4***
		Masseter	197.4±10.5aa	170.8±10.0aa	26.6±4.2***
Female	(n=10)	Anterior Temporal	188.2±12.9	158.8±12.4b	29.4±3.5***
		Masseter	174.2±13.1	150.6±10.6	23.7±3.9***
Total	(n=36)	Anterior Temporal	218.5±10.3	187.0±8.4	31.5±4.0***
		Masseter	190.9±8.5aa	165.2±7.9aa	25.8±3.2***

PPT-before : PPT values before sustained isometric contraction
PPT-after : PPT values immediately after sustained isometric contraction
ΔPPT : the difference between PPT-before and PPT-after

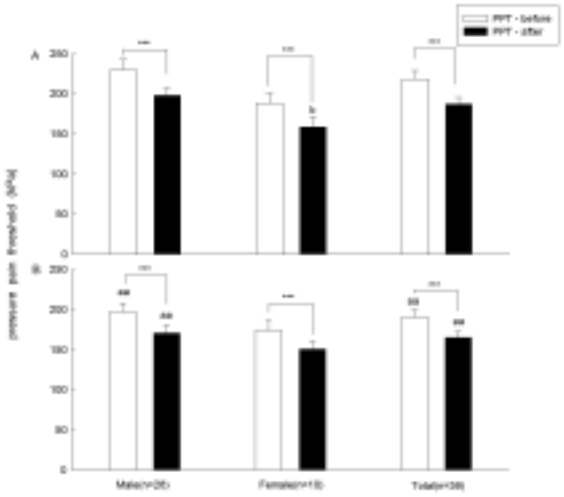

Fig. 2. Pressure pain threshold (PPT) values before and after sustained isometric contraction in both anterior temporal muscle (A) and masseter muscle (B). Asterisks indicate a statistically significant difference of PPT values between before and after sustained isometric contraction (***: p<0.001). The 'a' indicates a statistically significant PPT difference between anterior temporal and masseter muscle (aa: p<0.01). The 'b' indicates a statistically significant gender difference of PPT (b: p<0.05).

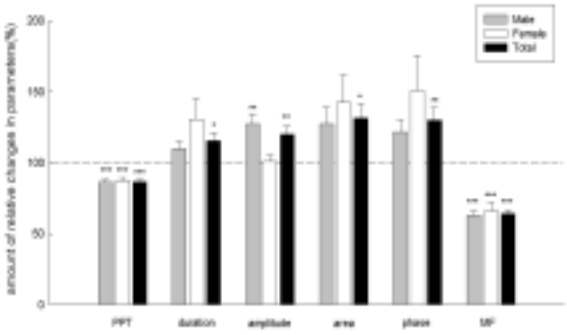

Fig. 3. Relative changes of anterior temporal muscle parameters during sustained isometric contraction. Asterisks indicate statistically significant changes of parameters during sustained isometric contraction (*: p<0.05, **: p<0.01, ***: p<0.001).

male group, all of PPTs in anterior temporal muscle were significantly higher than in masseter muscles ($p < 0.01$), however, all PPTs did not show any significant differences between two muscles in the female group. For all subjects PPTs in anterior temporal muscles were significantly higher than those in masseter muscles ($p < 0.01$), and only statistically significant gender difference were found in PPTs, which measured after contraction in anterior temporal muscles (Fig. 2).

In anterior temporal muscle of the male group, 3 MUAP parameters, except amplitude, which measured after fatiguing contraction were significantly higher than those before fatiguing contraction. Statistical significances were as follows; duration, phase ($p < 0.001$), and area ($p < 0.01$). In the female group, all MUAP parameters did not show any significant differences between before and after fatiguing contraction. For all subjects, 3 MUAP parameters, except amplitude, which measured after fatiguing contraction were significantly higher than those before fatiguing contraction. Statistical significances were as follows: phase ($p < 0.001$), duration, and area ($p < 0.05$) (Table 2, Fig. 3).

In a masseter muscle of the male group, MUAP amplitude ($p < 0.01$) which measured after fatiguing contraction was significantly higher than those before fatiguing contraction. In the female group, all MUAP parameters did not show any significant differences between before and after fatiguing contraction. For all subjects, all MUAP parameters, which measured after fatiguing contraction were significantly higher than those before fatiguing contraction. Statistical significances were as follows; duration, area ($p < 0.05$), amplitude, phase ($p < 0.01$) (Table 3, Fig. 4).

The median frequencies (MFs) were found to be compressed significantly to lower frequency range as muscle contraction was sustained in all subjects ($p < 0.001$). The MFs did not show any significant differences between male and female groups in both muscles. There were no significant differences between male and female groups/between anterior temporal and masseter muscles in the amount of change in MFs and slope of median frequency shift.

Table 2. Motor unit action potential (MUAP) parameters in anterior temporal muscle before and after sustained isometric contraction. Data presented as mean±standard error of means. Asterisks indicate statistically significant differences of MUAP parameters between before and after sustained isometric contraction (*: p<0.05, **: p<0.01, ***: p<0.001).

			Duration(ms)	Amplitude(μV)	Area(μVms)	Phase
Male	(n=26)	before	22.55±2.13	447.3±62.9	877.9±131.9	4.3±0.4
		after	28.25±2.81	492.8±72.7	1259.9±196.6	5.7±0.6
		Δ	-5.70±1.55***	-45.5±47.2	-382.0±125.0**	-1.4±0.4***
Female	(n=10)	before	26.94±3.53	405.6±82.4	990.6±240.1	4.6±0.6
		after	25.11±3.31	438.9±87.2	890.7±179.1	5.0±0.8
		Δ	1.83±2.61	-33.4±47.2	99.9±116.2	-0.5±0.6
Total	(n=36)	before	23.77±1.83	435.7±50.3	909.2±114.7	4.4±0.3
		after	27.38±2.22	477.8±57.3	1157.4±151.7	5.5±0.5
		Δ	-3.61±1.43*	-42.1±36.2	-248.2±101.7*	-1.1±0.3***

before : before sustained contraction
after : after sustained contraction
Δ : the difference of MUAP parameter values between before and after sustained contraction

Table 3. Motor unit action potential (MUAP) parameters in masseter muscle before and after sustained isometric contraction. Data presented as mean ± standard error of means. Asterisks indicate statistically significant differences of MUAP parameters between before and after sustained isometric contraction (*: p<0.05, **: p<0.01)

			Duration(ms)	Amplitude(μV)	Area(μVms)	Phase
Male	(n=26)	before	29.36±1.74	432.8±33.0	1198.0±133.4	5.4±0.4
		after	31.55±2.07	537.8±47.7	1381.9±155.7	6.3±0.6
		Δ	-2.20±1.53	-105.0±31.6**	-183.9±131.6	-9.7±0.5
Female	(n=10)	before	30.29±1.85	548.9±64.6	1292.7±197.3	5.9±0.6
		after	38.05±2.88	556.2±65.3	1623.9±172.2	8.5±1.2
		Δ	-7.76±3.58	-7.3±21.9	-331.2±206.2	-2.6±1.2
Total	(n=36)	before	29.62±1.35	465.1±30.6	1224.3±109.6	5.5±0.3
		after	33.36±1.74	542.9±38.5	1449.1±122.3	6.9±0.6
		Δ	-3.74±1.52*	-77.8±24.6**	-224.8±110.0*	-1.4±0.5**

before : before sustained contraction
after : after sustained contraction
Δ : difference of MUAP parameters between before and after sustained contraction

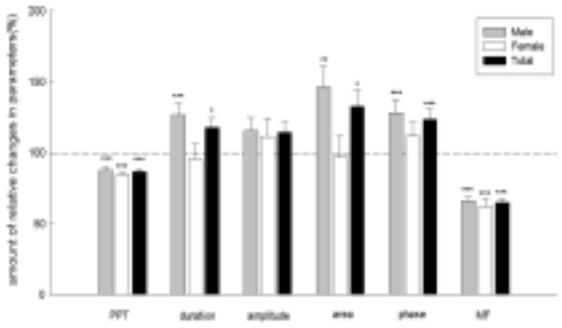

Fig. 4. Relative changes of masseter muscle parameters during sustained isometric contraction. Asterisks indicate statistically significant changes of parameters during sustained isometric contraction (*: p<0.05, **: p<0.01, ***: p<0.001).

The MFs in anterior temporal muscles of the male group were significantly higher than those in masseter muscles in both beginning ($p < 0.001$) and endpoint ($p < 0.05$) of sustained isometric contraction. But the MFs in the female group did not show significant differences between anterior temporal and masseter muscles. For all subjects, the MFs were significantly higher in anterior temporal muscles than those in masseter muscles in both the beginning ($p < 0.001$) and endpoint ($p < 0.05$) of sustained isometric contraction (Table 4, Fig. 5).

Tables 5 and 6 showed correlations between PPT, MUAP parameters, and MF in both anterior temporal and masseter muscles. Before sustained isometric contraction we could not find any significant correlations between PPT and MUAP parameters/ between PPT and MF in both two muscles. Although no significant correlations were found between PPT and MUAP parameters of anterior temporal muscle, the masseter muscle had significant weak negative correlations between PPT and MUAP parameters after sustained isometric contraction. Each correlation coefficients were as follows; duration ($r = -0.335$, $p < 0.05$), amplitude ($r = -0.359$, $p < 0.05$), area ($r = -0.421$, $p < 0.05$), and phase ($r = -0.331$, $p < 0.05$). In both muscles no significant correlation was found between MF and PPT/ between MF and MUAP parameters, regardless of fatiguing contraction. On the other

Table 4. Median frequency(MF) values before and after sustained isometric contraction. Data presented as mean ± standard error of means. Asterisks indicate statistically significant differences of MF values between before and after sustained isometric contraction (***: p<0.001). The 'a' indicates statistically significant differences of MF values between anterior temporal and masseter muscle (a: p<0.05, aaa: p<0.001).

			MF-start(Hz)	MF-end(Hz)	ΔMF(Hz)	Slope of MF shift
Male	(n=26)	Anterior Temporal	176.2±5.9^{aaa}	117.3±7.5^{a}	58.8±5.1***	-3.74±0.38
		Masseter	152.3±5.9	98.1±6.9	54.2±4.0***	-3.32±0.29
Female	(n=10)	Anterior Temporal	190.0±14.5	118.0±13.4	72.0±11.5***	-4.26±0.59
		Masseter	163.0±8.4	106.0±10.1	57.0±11.1***	-3.48±0.56
Total	(n=36)	Anterior Temporal	180.0±5.8^{aaa}	117.5±6.5^{a}	62.5±4.9***	-3.88±0.31
		Masseter	155.3±4.9	100.3±5.7	55.0±4.1***	-3.37±0.25

MF-start : median frequency at the beginning of sustained isometric contraction
MF-end : median frequency at the end of sustained isometric contraction
ΔMF : the frequency difference between MF-start and MF-end

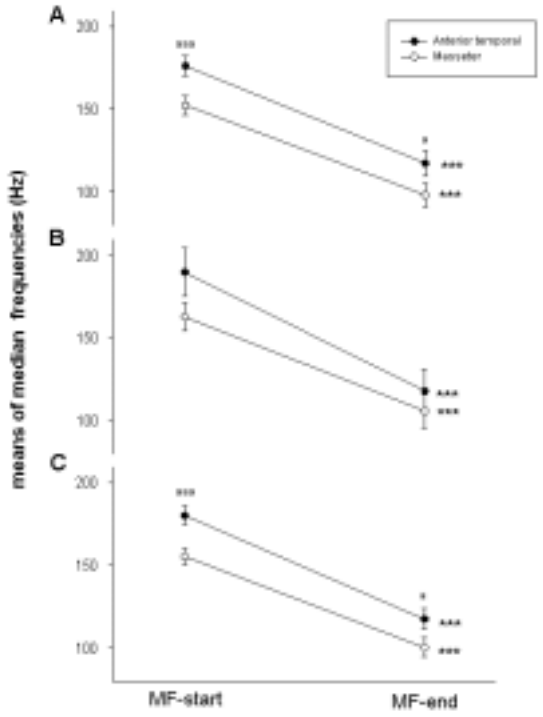

Fig. 5. Median frequency (MF) changes during sustained isometric contraction in the male group (A), the female group (B), and total subjects (C). Asterisks indicate statistically significant decrease of median frequencies during sustained isometric contraction (***: p<0.001). The 'a' indicates statistically significant differences of MFs between anterior temporal and masseter muscle (a: p<0.05, aaa: p<0.001).

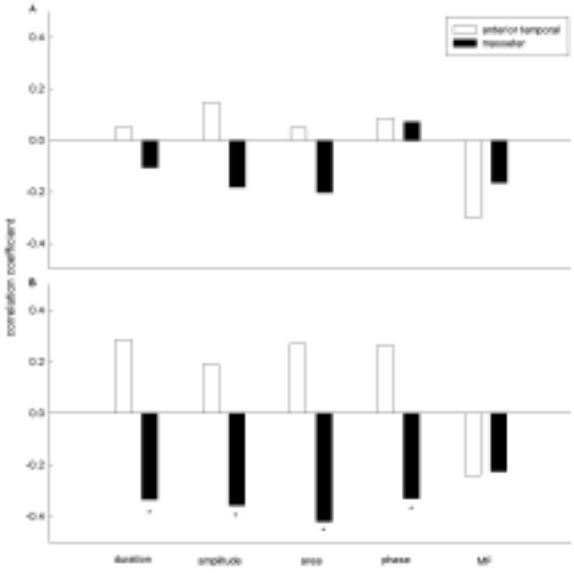

Fig. 6. Correlation coefficients between pressure pain threshold (PPT), motor unit action potential (MUAP) parameters, and median frequency (MF) before sustained isometric contraction (A) and after sustained isometric contraction (B). Asterisks indicate statistically significant correlation coefficients (*: p<0.05).

hand, there were relatively strong correlations between MUAP parameters in both muscles ($p < 0.01$). Figure 6 showed correlation coefficients between PPT, MUAP parameters and MF in both anterior temporal and masseter muscles.

Table 5. Correlation coefficients between motor unit action potential parameters, pressure pain threshold (PPT), and median frequency (MF) in both anterior temporal and masseter muscles before sustained isometric contraction. Values presented as Pearson correlation coefficients (anterior temporal/masseter). Asterisks indicate statistically significant correlation coefficients (**: p<0.01).

	PPT	Duration	Amplitude	Area	Phase
Duration	0.053/-0.106				
Amplitude	0.148/-0.182	0.654^{**}/0.472^{**}			
Area	0.049/-0.202	0.901^{**}/0.884^{**}	0.777^{**}/0.535^{**}		
Phase	0.086/ 0.072	0.880^{**}/0.534^{**}	0.669^{**}/0.620^{**}	0.764^{**}/0.338^{**}	
MF	-0.300/-0.165	-0.075 /-0.168	0.009 /-0.146	0.041 /-0.119	-0.137/-0.217

Table 6. Correlation coefficients between motor unit action potential parameters, pressure pain threshold (PPT), and median frequency (MF) in both anterior temporal and masseter muscles after sustained isometric contraction. Values presented as Pearson correlation coefficients (anterior temporal/masseter). Asterisks indicate statistically significant correlation coefficients (*: $p<0.05$, **: $p<0.01$).

	PPT	Duration	Amplitude	Area	Phase
Duration	0.285/−0.335*				
Amplitude	0.188/−0.359*	0.898**/0.564**			
Area	0.268/−0.421*	0.934**/0.918**	0.934**/0.647**		
Phase	0.263/−0.331*	0.934**/0.897**	0.860**/0.593**	0.853**/0.765**	
MF	−0.242/−0.227	−0.289/−0.174	−0.035/−0.086	−0.121/−0.163	−0.184/−0.136

Ⅳ. DISCUSSION

The definition of muscular fatigue is a failure of the muscle tissues to maintain an expected force. It can be classified into two types. One is central fatigue that is explained as a failure of the command from the motor cortex and the upper motor neuron, the other is peripheral fatigue at the end plate of motor neuron. It may be caused by a disruption in either transmission or contraction[14]. Fatigue by prolonged submaximal voluntary contraction such as 70 % of maximum voluntary isometric contraction in this study resembles contraction fatigue that interferes with excitation-contraction coupling and/or metabolic processes related to these reactions. In contraction fatigue the EMG amplitude is unchanged as the force drops, or the EMG amplitude is increased while the force remains unchanged. Several factors such as low level of Ca^{2+}, glycogen, and adenosine triphosphate (ATP), and high level of lactate are associated with contraction fatigue[14]. Two mechanisms for optimizing force production under fatiguing conditions are recruitment of motor units and modulation of motor unit discharge following recruitment[15].

A motor unit constitutes the smallest functional element of contraction. The anatomic and physiologic properties of a motor unit are based on the innervation ratio, fiber density, propagation velocity, and integrity of neuromuscular transmission[16]. The shape of the MUAP waveform depends upon the motor unit architecture, *i.e.*, the number, size, distribution of muscle fibers in the motor unit and the geometry of the end-plate zone. If the motor unit architecture is changed by disease processes such as loss of fibers and re-innervation, the MUAP waveform will equally be changed. Quantification of the MUAP parameters such as duration, amplitude, area, and phase has been used to characterize neuromuscular disorders. For an ideal MUAP quantification one must count at least 20 different motor units in each muscle using multiple needle insertions[16] . Engstrom *et al.* studied the influence of MUAP sample size tolerance limits, intertrial variability, and diagnostic sensitivity in both myopathic patients and normal control group[17]. They reported that although analysis of 5 potentials might be adequate for diagnosis occasionally, quantitative analysis of 20 MUAPs narrowed tolerance limits, reduced intertrial variability, and improved diagnostic sensitivity. The needle electrode registers muscle action potentials only from a restricted area of the muscle. For an adequate survey frequent needle repositioning is necessary for multiple sampling in small steps. Exploration in various directions from a single puncture site minimizes the patient's discomfort. Therefore the author measured 20 different MUAPs with careful repositioning of the needle electrode within the insertion site in the

tested muscle.

The MUAP abnormalities may be assessed from their peak-to-peak amplitude, duration, and phases of the waveform and the firing rate of the motor unit. A number of parameters have been used to characterize the MUAP. It is important that these parameters reflect morphological or physiological features and can be changed by disease. Not all parameters have proven useful. The most commonly used parameters are duration, amplitude, rise-time, area and phase[18].

In this study amplitude, phase, duration, and area of MUAPs after sustained isometric contraction of masseter muscles were significantly higher than those before contraction. In anterior temporal muscle phase, duration, and area were significantly increased after sustained isometric contraction.

Lindström *et al*. showed that the average change in MUAP duration was linearly related to the change in the mean frequency of the power spectrum[19]. Sandercock *et al.* have observed increases in both MUAP amplitude and duration during low-frequency electrical stimulation of the medial gastrocnemius muscle of the cats[20]. Celichowski *et al.* studied on the changes in amplitude, duration and latency of MUAPs during fatigue in the medial gastrocnemius muscle of the rats[21]. They found the amplitude of MUAPs decreased during fatigue. They explained that redistribution of ions across cell membrane of individual muscle fibers was the main cause of the decreased MUAP amplitude during fatigue.

In this study, there was an increase of MUAP amplitude and duration during fatiguing contraction. It is suggested that the increase in MUAP amplitude resulted from efficient synchronization of negative and positive phases of muscle fiber action potentials within the motor unit and better synchronization may also have some relation to the prolonged duration of muscle fiber action potentials. In most previous studies duration of action potential became prolonged after muscular fatigue[20,21,22]. Those studies also suggested that an increased duration of action potential was related to a decreased muscle fiber conduction velocity after muscular fatigue. Therefore the duration of action potentials can illustrate the feature of muscular fatigue more properly than the amplitude of the potentials.

The higher number of phases in the MUAPs may be due to their higher amplitude and complexity. Slow conduction in terminal axons and increased variability of muscle fiber diameter by muscle fiber recruitment also attribute to an increase of phases[18]. Although amplitude of MUAP in anterior temporal muscle was increased during sustained isometric contraction, the statistical significance did not exist in this study. This result might be due to variable amplitude value dependent upon electrode position within same motor unit territory. Amplitude is determined by less than eight fibers, sometimes one or two fibers, within 0.5 mm from the electrode[18]. Therefore the amplitude may vary considerably within the motor unit territory and the same motor unit can generate many motor unit action potentials with different amplitudes at different recording sites[16]. It is therefore suggested that MUAP amplitude is not a pertinent parameter of muscular fatigue.

Duration reflects the total current generated by the fibers in the motor unit. The total current is determined by the number of muscle fibers and their cross-sectional area that are up to 2.5 mm from the recording electrode. This radius includes a large portion of the motor unit territory in muscles. The MUAP duration is increased in neuropathic patients where the motor unit size is increased due to collateral sprouting and re-innervation of the denervated muscle fibers. In myopathic patients, the MUAP duration decreases due to loss of muscle fibers, atrophy and probably fibrosis. This may alter volume conduction properties[23].

The area that is calculated by integrating the rectified MUAP over the duration reflects the number and the diameter of muscle fibers, as well as the temporal distribution of the single fiber action potentials. The area can reflect the activity of muscle fibers better than the amplitude. Therefore it represents similar motor unit characteristics as

amplitude and duration. In general, increase of both duration and amplitude results in increase of area.

All of amplitude, area, and duration can also reflect the diameter of the muscle fibers, the number of muscle fibers, and the fiber density of the motor units. The main difference between these parameters is the uptake area. Both duration and area are global estimators with a large uptake area while amplitude reflects activity of the fibers closest to the electrode. According to the study of Stålberg *et al.*, amplitude and area were larger in men than in women[18]. This is probably due to larger muscle fiber diameter in men than in women. No gender differences in these MUAP parameters in this study may be due to low statistical power because female subjects were not enough.

In normal motor units arrival times of the single fiber action potentials at the electrode is evenly distributed and the MUAP has a smooth bi- or triphasic shape. During the fatiguing contraction uneven distribution of the single fiber action potentials into separate groups and an increased temporal dispersion of the single fiber action potentials can result in an increased number of phases. In both neuropathic and myopathic conditions there is an increased number of complex or polyphasic MUAPs. These complex MUAPs are due to an increased variation among pathologic muscle fiber diameters causing a wider range of conduction velocities along muscle fiber membranes and, to some degree, are due to the scattered positions of end-plates after re-innervation. These two phenomena produce increased temporal dispersion among individual single fiber action potentials, causing the typical changes in MUAP shape.

Rise-time usually refers to the time it takes for signals to go from the positive to the negative peak. It helps estimate the distance between the recording tip of the electrode and the discharging motor unit. A MUAP accepted for quantitative measurement should have a rise time less than 0.5 ms, preferably 0.1 to 0.2 ms[16]. This study includes the MUAPs whose rise time was less than 0.5 ms.

In this study there were significant correlations within MUAP parameters regardless of muscle contraction. These results consistent with the definition and representative features of each MUAP parameters.

EMG power spectral analysis was used to provide an index of localized muscular fatigue[8,9,10]. There are several frequency parameters such as mean, median, and the ratio of low-frequency components to high-frequency components of the spectrum in EMG power spectral analysis. Among them it has been found that the median frequency was the least sensitive to noise and preferred parameters in most of power spectral analyses[24].

Many investigators proved that the power spectrum of the myoelectric signal is compressed into lower frequency range during sustained muscle contraction[8,9]. Median frequencies in both anterior temporal and masseter muscles were equally found to be compressed significantly to lower frequency range during muscular fatigue[25,26]. Theories of transition of power spectrum to lower frequency range are reduction of action potential conduction velocity, change in action potential shape, recruitment of new motor unit, and synchronization of motor unit or some combinations of these factors [9,19,27]. The relative importance of each theory has not been determined and may vary with different muscles, fatigue protocols, and methods of analysis. Palla *et al.* explained that decrease of the conduction velocity of the action potential along the muscle fiber seemed to be the main cause of the shift[9]. The blood circulation within muscles is arrested during an isometric submaximal contraction and the need for oxygen is not satisfied. As a consequence, there is a change from an aerobic to an anaerobic type of metabolism characterized by a lower efficiency. At the same time, the washout is insufficient and acid metabolites gradually accumulate. The accumulation of metabolites is of greater importance for the development of fatigue than the oxygen deficiency. It is well established that a lowering of the intracellular pH, caused by production of acid metabolites results in a lowering of the excitability

of the membrane[19]. Therefore it might be developed a decrease in the propagation velocity of action potentials and consequent spectral changes.

Some investigators suggest that the synchronization of motor units increase during fatigue, increasing the duration of the EMG fluctuation, thus, the power spectrum shifts to lower frequencies. Palla *et al.* reported that increase in bite force produced a power spectrum shift to lower frequencies, in both anterior temporal and masseter muscles[9]. The shift was probably caused by an increase in synchronization between motor units and newly recruitment of larger motor units. According to Lindström *et al.*, however, synchronization of motor units was not a necessary condition of EMG change but observed phenomenon induced by muscular fatigue[19]. During the sustained contraction, force augmentation is produced by an increase in the motor neuron firing rate and recruitment of new motor units. According to the Henneman's size principle, the motor neurons were recruited not at random but in an orderly manner determined by the fixed central drive that preferentially activates small motor neurons first[16]. As the duration of the MUAP seems to be related to the size of a motor unit, the recruitment of larger motor units at higher bite forces could increase the average duration of the MUAPs and therefore produce the power spectral shift to lower frequency range[9]. In addition Palla *et al.* showed findings that recruitment of larger motor units should produce a power spectral shift to lower frequencies because such motor units have action potentials of longer duration than small ones[9]. During a submaximal voluntary contraction continued until fatigue was reached, larger motor units with action potentials of higher amplitude and longer duration replaced small ones. Thus the power spectral shifts to lower frequencies were found during fatigue elicited by submaximal voluntary contraction, seemed to be caused by an increased duration of the MUAP. DeLuca suggested that changes of action potential shape led to power spectral change as similar pattern of conduction velocity decrease during fatiguing contraction[27].

Median frequencies in anterior temporal muscles were significantly higher than those in masseter muscles, which are consistent with the previous studies that there were differences of median frequencies between different muscles[8,9,25,26]. According to the Lund *et al.*, this phenomenon may be due to the proximity of the muscle fibers to the skin surface because connective tissue and fat act as low-pass filters[28]. The fact that it is nearer to the skin surface may be one reason the high-frequency components are relatively stronger in the anterior temporal muscle than in the masseter.

In this study pressure pain threshold (PPT) values were significantly decreased during sustained isometric contraction in both anterior temporal and masseter muscles. McMillan *et al.* reported that patients with myofascial pain had lower PPTs than the normal pain-free control group[29]. Tension-type headache patients had lower PPTs than the normal control group[13]. Kosek *et al.* reported decrease of PPTs during isometric contraction in fibromyalgia patients and explained that the decrease of PPTs might be due to sensitization of mechanonociceptors caused by muscle ischemia and/or dysfunction in pain modulation during muscle contraction[30]. Dysfunction of one or several of the pain inhibitory mechanisms could explain the abnormal PPT response during fatiguing muscle contraction. Mense *et al.* suggested that muscle ischemia could cause sensitization of mechanonociceptors, and the mechanical component of a contraction would become an effective stimulus for nociceptive afferents, that is, A delta and C fibers[31].

On the other hand, the PPTs on anterior temporal muscle were higher than those of masseter muscle regardless of sustained isometric contraction in this study. This finding is consistent with previous findings in the jaw muscles[5,32,33]. It has been clearly unknown why the PPT of anterior temporal muscle is higher however, it is probably assumed that there may be fewer cutaneous and muscle receptors in the temporal region. There is also variation in the density of connective tissue tendon in the anterior

temporal region compared with the masseter which may have contributed to the difference in PPTs[29].

Several investigators reported that females have lower PPT values than males in craniomandibular disorder patient population[4,32,33]. In this study the PPTs in the male group were also higher than those in the female group before and after sustained isometric contraction, but statistically significance was found on anterior temporal muscle only which measured after sustained isometric contraction because of small sample size.

We could not find any statistically significant correlations between PPTs and MUAP parameters that measured before fatiguing contraction in both muscles. After fatiguing contraction, however, statistically significant negative correlations were found between PPTs and MUAP parameters in masseter muscle. The evaluation of masticatory muscle by means of PPT and MUAP that measured after its function, such as clenching, has more validity and superiority of diagnostic worth. Further researches should take this fact into consideration.

In conclusion in order to investigate muscular fatigue not only PPT and EMG power spectrum but also MUAP analysis which is functional basic unit of muscle contraction may be a useful method. A comparative study between patients with chronic myofascial pain and a normal control group in the level of a motor unit, and further research is thought to be required on the development other MUAP parameters that is more susceptible to fatigability.

V. CONCLUSIONS

The purpose of this study was to investigate the changes in motor unit action potential (MUAP), pressure pain threshold (PPT), EMG power spectrum of human masticatory muscles during experimentally induced muscular fatigue.

Thirty-six healthy volunteers (26 males and 10 females) without past history and present symptoms of temporomandibular disorders were included. MUAP, PPT, and EMG power spectrum were recorded before and after sustained isometric contractions of both anterior temporal and masseter muscles at 70% level of maximum voluntary contraction of EMG activity.

Our data have come to the following conclusions:

1. PPTs of both anterior temporal and masseter muscles were significantly decreased during sustained isometric contraction ($p < 0.001$).
2. PPTs of anterior temporal muscles were significantly higher than those of masseter muscles in both before and after sustained isometric contraction ($p < 0.01$). In general, PPTs of the male group were higher than those of the female group, however, significant gender difference was only shown in PPTs of anterior temporal muscles after sustained isometric contraction ($p < 0.05$).
3. Median frequencies (MF) were significantly shifted to lower frequency range during sustained isometric contraction ($p < 0.001$), and MFs were significantly higher in anterior temporal muscles than those in masseter muscles in both beginning ($p < 0.001$) and endpoint ($p < 0.05$) of sustained isometric contraction.
4. Amplitude, phase ($p < 0.01$), duration, and area ($p < 0.05$) of MUAP in masseter muscles after sustained isometric contraction were significantly increased than those before contraction. Phase ($p < 0.001$), duration, and area ($p < 0.05$) of MUAP in anterior temporal muscles were significantly increased during sustained isometric contraction.
5. In both anterior temporal and masseter muscle there was no significant correlation between PPTs and MUAP parameters before sustained isometric contraction. After sustained isometric contraction significant correlations between PPTs and 4 MUAP parameters[duration ($r = -0.335$, $p < 0.05$), amplitude ($r = -0.359$, $p < 0.05$), area ($r = -0.421$, $p < 0.05$), and phase ($r = -0.331$, $p < 0.05$)] were found in masseter muscles. But there was not any significant correlations between PPTs and MUAP parameters in anterior temporal muscles.

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국문초록

피로를 유발하는 지속적인 근수축 동안 저작근의 운동단위전위, 근전도 power spectrum, 압력통각역치 변화에 대한 연구

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본 연구는 구강안면동통 중에서 빈번히 나타나는 근육성 동통의 주 원인인 저작근의 과활성으로 유발된 근육의 피로 시에 운동단위전위, 압력통각역치, 근전도 power spectrum의 변화 양상과 이들 척도간의 연관성을 조사하기 위해 시행되었다.

두개하악장애의 병력 및 현증이 없고 정상적인 구치부 교합관계를 가진 평균연령 25.8세인 36명의 정상 성인(남자 26명, 여자 10명)을 대상으로 교근과 전측두근의 지속적인 등길이 수축 전후의 압력통각역치 및 운동단위전위를 측정하였고 인내시간까지의 근수축 동안 근전도 power spectrum을 분석하여 다음과 같은 결론을 얻었다.

1. 지속적인 등길이 수축 후 교근과 전측두근의 압력통각역치는 수축 전에 비해 유의하게 감소하였다.
2. 압력통각역치는 수축 전과 수축 후 모두에서 전측두근이 교근보다 유의하게 높게 나타났으며, 전체적으로 남성이 여성보다 높게 나타나는 양상을 보였으나 성별간의 차이는 전측두근의 수축 후 압력통각역치에서만 통계적으로 유의하게 나타났다.
3. 지속적인 등길이 수축말기의 중간주파수는 수축초기에 비하여 유의하게 감소하였고, 전측두근의 수축초기 중간주파수와 수축말기 중간주파수 모두 교근보다 유의하게 높게 나타났다.
4. 교근은 지속적인 등길이 수축 전에 비하여 수축 후의 운동단위전위의 지속시간,진폭, 면적, 상의 4가지 척도에서 유의한 증가를 보였고 전측두근은 진폭을 제외한 나머지 3가지 척도, 즉 지속시간, 면적, 상의 유의한 증가를 보였다.
5. 교근과 전측두근의 지속적인 등길이 수축 전의 압력통각역치와 운동단위전위 척도 사이에는 통계적으로 유의한 상관관계가 없었고 교근에서는 수축 후의 압력통각역치와 운동단위전위의 지속시간, 진폭, 면적, 상 사이에 유의한 상관관계가 존재하였다.

위의 실험결과를 통해 근육피로 검사에 압력통각역치, 근전도 power spectrum 검사 외에 근육수축의 기능적 최소 단위인 운동단위전위의 분석 또한 유용할 수 있고 추후 만성으로 진행된 근막동통환자와 정상 대조군간의 운동단위 수준에서의 비교연구와 근피로에 더욱 민감한 운동단위전위의 다른 척도에 대한 개발과 연구가 필요하다고 사료된다.

Key words: 저작근, 근전도, power spectrum, 압력통각역치, 운동단위전위, 근피로