The Specificity of Response to Experimental Stress in Patients with Myofascial Pain Dysfunction Syndrome

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Comparison of autonomic and muscular response to experimentally induced stress in normal individuals and patients with myofascial pain dysfunction (MPD) syndrome revealed greater masseter and frontalis activity in the patient group, higher gastrocnemius activity in control subjects, and no significant difference in skin conductance and heart rate. This specificity of response to stress supports the psychophysiologic theory of MPD syndrome.

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Introduction.

The nature of the relationship between psychological stress and normal, as well as pathologic, somatic responses has been studied by many investigators. One of the early theories held that certain patients had a vulnerable area of the body ("target organ") which would break down under excessive stress, and that explained why these patients developed certain diseases.[1-3] This proved to be a tautologous assumption which merely oversimplified the issue of cause and effect. Despite its shortcomings, however, the target organ theory became the basis for a subsequent concept of "response specificity".[4] This concept has been validated through extensive research with normal subjects, and also with various patient groups.[5-10]

Response specificity has been defined by Lacey *et al.*[4,5] as a characteristic physiologic response to stress for each individual. Although every person tends to respond to stress of any kind in a number of ways, one type of physiologic response usually predominates over all others. Lacey *et al.* originally studied only autonomic responses to stress such as increases in heart rate, respiration, and galvanic skin resistance. More recently, Goldstein, Shipman, and others have shown a direct relationship between increased muscle tension and increased stress, frustration, aggression, and anxiety.[8-10] They also found the musculature to be the predominant stress response area in certain individuals.

A related concept of "symptom specificity" explains the relationship between psychological stress and certain somatic disorders. Originally proposed by Malmo and Shagass,[3] who found that individuals with psychosomatic symptoms showed heightened responsiveness to stress in the area of their symptoms, this concept also has gained extensive experimental support. The early investigators in this area studied mainly autonomic responses, but later studies of patients with muscular problems (headache, backache, etc.) also showed a strong relationship between stress and increased muscular activity in the symptomatic areas.[11,12]

During the past 20 years, several investigators have studied the effects of stress on activity of the masticatory muscles in normal subjects as well as symptomatic myofascial pain dysfunction (MPD) syndrome patients.[13-17] Johnson[16] and Thomas *et al.*[17] have shown that a significant relationship exists between experimentally induced stress and increased masticatory muscle activity, especially in symptomatic patients. Until now, however, no published studies have dealt directly with the issue of response specificity in patients with MPD syndrome. While it is clear that many of these patients respond to both environmental and experimental stress with masticatory muscle hyperactivity, it has not been demonstrated that this is a predominant response which consistently occurs in a

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large percentage of patients, nor has it been shown whether this is a localized muscular phenomenon or a generalized one.

The purpose of this study, therefore, was to measure various physiologic responses in a group of MPD syndrome patients who were subjected to a series of experimental stresses, and to compare their responses with those of a matched control group. The physiologic responses which were measured included masseter, frontalis, and gastrocnemius activity; heart rate; and galvanic skin response (GSR).

Materials and methods.

Subjects. – Patients admitted to the Temporomandibular Joint and Facial Pain Research Center, University of Illinois, were selected for this study on the basis of having one or more of the cardinal symptoms of myofascial pain dysfunction (MPD) syndrome: 1) pain in the preauricular area or masticatory muscles; 2) limitation of mandibular movement; 3) tenderness in the masticatory muscles; and 4) clicking or popping in the temporomandibular joint (TMJ). However, patients with only joint sounds, or patients with organic pathology of the TMJ, were excluded from the study. The patients ranged in age from 14 to 52 years, with a mean of 30 years. There were 18 females and 2 males.

Control subjects were 18 females and 2 males ranging from 19 to 48 years, with a mean of 30 years. None of these subjects had a history of MPD syndrome symptoms or other TMJ dysfunction.

Apparatus. – Beckman biopotential skin surface electrodes* were used to record simultaneous activity in the right and left masseter, frontalis, and right gastrocnemius muscles, as well as changes in heart rate and galvanic skin resistance. Electrode placement was similar for all patients and control subjects.[16,18,19] A Beckman Offner multichannel Type R Dynograph recorder and Beckman couplers were used to amplify and record all incoming signals.⁺

Procedure. – All subjects scheduled for testing were told they were to have a muscle test. At the time of the test, each subject was brought into a room and seated in a straight-back chair in front of a waist-high table facing away from the recording equipment. No attempt was made to allay apprehension. The subject was told that several minutes of preparation were necessary prior to commencement of the test in order to attach the "microphones" which would pick up and record "muscle noises" through the skin. The skin at each site of the electrode placement was scrubbed to remove the nonconducting keratin layer.

The experimental period was divided into six continuous phases: 1) a one-minute pre-test phase where nothing was done while baseline values were recorded for each parameter; 2) a two-minute phase when the subjects listened to 75±5 decibel white noise delivered through stereo headphones; 3) a one-minute rest period followed by a card sorting exercise devised to provoke stress[16] (this consisted of 45 cards, each of which had a word on it. While some of the words were neutral, most of them were sexually related or identified significant personal relationships. Subjects were instructed to sort these rapidly into three piles – words liked, words disliked and words to which they were indifferent); 4) another one-minute rest period followed by a word association phase (the examiner would start with the word cards that had been placed in the "like" pile and say each word to the subject. The subject then gave an immediate one-word reaction. This was repeated for the indifferent and disliked words); 5) another one-minute rest period was followed by a pain phase using the dolorimeter as described by Poser[20] (the painful stimulus was produced by 94 pointed acrylic projections, 7 mm long, sewn into the cuff of a standard clinical sphygmomanometer. The cuff was placed on the arm and inflated slowly. The subjects were told to say "stop" when it became painful and the pressure was released); 6) a post-test phase of one minute of silent recording to re-establish baseline activity. Thus, the entire test covered a period of approximately 30 minutes.

*Beckman Instruments, Inc., Clinical Instruments Operations, Fullerton, CA

⁺Beckman Offner, Palo Alto, CA

Results.

All changes in heart rate, galvanic skin resistance, and muscle activity which occurred during the six phases were recorded simultaneously and continuously. Each set of measurements during a particular test phase was then averaged to determine the mean response for each physiologic system. Muscle activity scores were recorded in microvolts and converted to logarithms to facilitate the presentation of data, since both groups had such a wide range of activity in microvolts. Heart rate was recorded in beats per minute. Galvanic skin resistance was measured in microohms, converted to skin conductance (reciprocal of resistance), and expressed as log microohms.

The mean log muscle activity for the patient and control groups is presented in Table 1. Since Johnson[16] found no relationship between side MPD symptoms and side of greatest EMG activity in a similar experiment, the present data are reported in terms of left- and right-side activity, rather than symptomatic- and asymptomatic-side activity.

Student's *t*-tests were calculated to locate any significant differences between the two groups, and the values obtained are presented in Table 1. The results were strikingly significant (20 of 24 comparisons at $p<.01$ and the other 4 at $p<.02$) and remarkably consistent. These data are presented graphically in Figures 1 through 4. Figures 1 and 2 show that, for right and left masseters, there was a large difference in muscle activity between the two groups in the pre-test situation. Subsequently, both groups had an increase in masseter activity under the psychologically stressful conditions, but that increase was much greater for the patients with MPD syndrome. When stressed with the dolorimeter, however, normal subjects showed no increase in masseter activity, but the patients again showed considerable increase. White noise did not change the level of muscle activity in either group. The initial difference in muscle activity between the two groups was even greater in the frontalis muscle (Fig. 3). However, the shape of the response curves was similar, indicating that the response to stress for the two groups was similar, and that the pre-test difference was simply maintained during the stress intervals.

In contradistinction to these results, gastrocnemius muscle response of the control subjects was significantly greater than that of the patients with MPD syndrome during the pre-test interval.

TABLE 1
MEAN LOG MUSCLE ACTIVITY AND STUDENT'S *t*-TEST FOR DIFFERENCES BETWEEN MPD PATIENTS AND CONTROL SUBJECTS (DF = 38)

		Pre-test	White Noise	Card Sorting	Word Association	Dolorimetry	Post-test
Right Masseter	MPD pts	1.08	1.16	1.63	1.72	1.58	1.23
	Control *S*s	.44	.45	.52	.51	.44	.44
	t values	4.00**	4.43**	8.21**	7.40**	7.12**	4.32**
Left Masseter	MPD pts	.97	1.12	1.48	1.59	1.44	1.20
	Control *S*s	.47	.47	.49	.54	.47	.47
	t values	3.84**	5.90**	5.89**	7.00**	5.71**	4.05**
Frontalis	MPD pts	1.46	1.49	1.63	1.67	1.62	1.55
	Control *S*s	.51	.51	.56	.63	.63	.54
	t values	6.33**	6.53**	7.13**	6.11**	6.19**	5.94**
Gastrocnemius	MPD pts	.69	.69	.77	.84	.76	.69
	Control *S*s	1.18	1.18	1.33	1.35	1.26	1.18
	t values	2.58*	2.58*	3.11**	2.83**	2.50*	2.58*

*significant at less than the .02 level
**significant at less than the .01 level

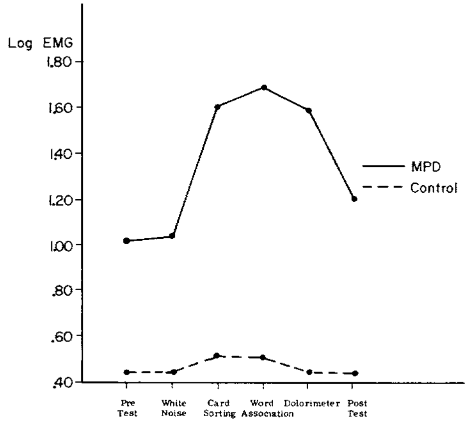

Fig. 1 – Mean log muscle activity in the right masseter of MPD patients and control subjects.

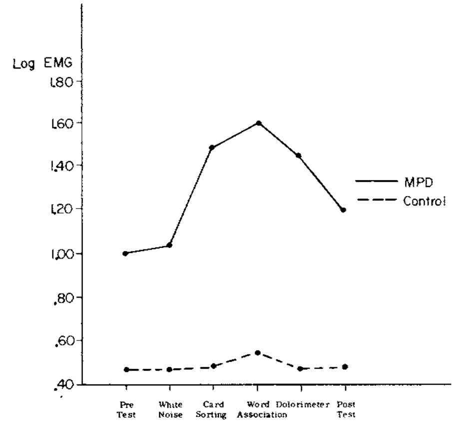

Fig. 2 – Mean log muscle activity in the left masseter of MPD patients and control subjects.

syndrome during the pre-test internal. This initial difference was maintained during the stress intervals (Figure 4).

There was no significant difference between the MPD patients and control subjects in the two autonomic parameters of skin conductance and heart rate (Table 2).

Discussion.

In a study preceding this one, Johnson[16] found that increased masseter and frontalis muscle activity, rather than increased autonomic activity, was the predominant response to experimentally induced stress in a series of 21 patients with MPD syndrome. The present experiment goes one step further in showing that there is a specific response in these muscles when they are compared to a representative general body muscle (gastrocnemius). Since no masticatory muscles other than masseter were studied, however, the question of their involvement must also be considered. Based on previous investigations of muscle activity in patients with MPD syndrome,[16,17] it is reasonable to assume that the response specificity in these patients could involve the masticatory muscles as a group rather than any single jaw muscle.

Although the population of patients with MPD syndrome is heterogeneous in many respects, a significant percentage have been shown to possess personality characteristics which not only predispose them to stressful situations, but also cause them to react to such situations by somatization.[21] In addition, there are biochemical, as well as physiological, data which indicate these patients, as a group, are under greater stress than normal individuals.[22] While experimental stress is not identical to the environmental stress of everyday life, other researchers have shown that similar physiologic responses occur in both situations.[8,9,10]

The present study completes the circle of logic which constitutes the psychophysio-

TABLE 2
MEAN LOG SKIN CONDUCTANCE AND HEART RATE FOR MPD PATIENTS AND CONTROL SUBJECTS (DF = 38)

		Pre-test	White Noise	Card Sorting	Word Association	Dolorimetry	Post-test
Skin Conductance	MPD pts	.13	.15	.13	.18	.18	.18
	Control *S*s	.15	.17	.16	.15	.16	.18
Heart Rate	MPD pts	84	87	92	99	93	88
	Control *S*s	94	94	96	98	96	94

Note: *t* values for all comparisons between group means were non-significant.

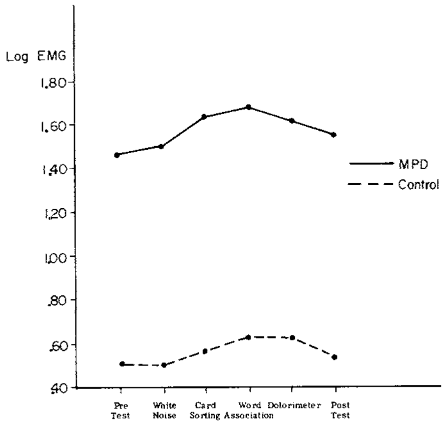

Fig. 3 – Mean log muscle activity in the frontalis of MPD patients and control subjects.

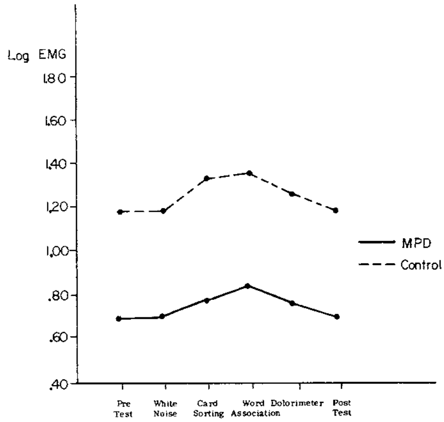

Fig. 4 – Mean log muscle activity in the gastrocnemius of MPD patients and control subjects.

logic theory of etiology for MPD syndrome[23] by showing that, when exposed to stress, these patients respond with increased masticatory muscle activity, rather than with a general increase in body muscle tonus. Such activity, whether centrally generated,[24] peripherally manifested as parafunctional habits, or both, can then result in muscular fatigue and spasm, leading to MPD syndrome. The concept of response specificity not only explains the localization of symptoms in stress-responding individuals, but it also explains why similar personality types may develop different psychophysiologic diseases.

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