Emotional and Physiologic Responses to Laboratory Challenges: Patients With Temporomandibular Disorders Versus Matched Control Subjects

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This study explored psychologic and physiologic factors differentiating patients with temporomandibular disorders (n = 23) from sex-, age-, and weight-matched asymptomatic control subjects. Each subject completed several standard psychologic questionnaires and then underwent two laboratory stressors (mental arithmetic and pressure-pain stimulation). Results indicated that patients with temporomandibular disorders had greater resting respiration rates and reported greater anxiety, sadness, and guilt relative to control subjects. In response to the math stressor, patients with temporomandibular disorders reacted with greater anger than did control subjects. There were no differences between patients with temporomandibular disorders and control subjects on pain measures or any other measured variable for the pressure-pain stimulation trial. In addition, there were no differences in electromyography levels between patients with temporomandibular disorders and control subjects. The results are discussed in terms of their implications for the etiology and treatment of this common and debilitating set of disorders.

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Interest in the psychologic and physical aspects of facial pain began with Moulton's[1,2] writings and clinical reports concerning the potential linkages between anxiety and such pain syndromes. Empirical research since then has focused on differentiating individuals with chronic facial pain or temporomandibular disorders (TMD) from asymptomatic, healthy control subjects to identify possible psychologic factors associated with the etiology, maintenance, and/or treatment of TMD. The most common findings are increased levels of anxiety and psychologic stress in patients with TMD as compared to control subjects.[3–5] However, efforts to define unique personality characteristics or psychologic risk factors associated with TMD have not yielded a consistent set of findings.[4,6]

Questions regarding the mechanisms by which anxiety and stress are linked to the onset and maintenance of TMD have led researchers to focus on a psychophysiologic model.[7,8] This model is based on the concept of autonomic response stereotypy that states that each person reacts to stressors with a unique physiologic response involving a specific system or area of the body.[9] In much of the dental literature, the effort to define unique respond-

ing in patients with TMD is based on Laskin's theory[10] relating the etiology of TMD to spasms and excessive tension in the masticatory muscles. As a result, electromyographic (EMG) activity in various masticatory muscles at rest and/or in response to laboratory stressors has often been measured in patients with TMD and in control subjects.[11–13] Methodologic errors (eg, nonmatched control groups with regard to age, sex, and body weight; no adaptation period; head movement; misunderstanding of EMG signal characteristics) and inconsistent results in many of these studies, however, have led to controversy regarding the role of muscle activity in the production and maintenance of TMD.[3,8,14,15]

Flor and Turk[16] reviewed the chronic pain literature generally, and TMD research specifically, and identified several important interpretational problems for physiologic data reported in the existing literature. These problems include inadequate diagnostic criteria for patient inclusion, poor sample description, use of single physiologic measures, use of inadequate stressors, inadequate adaptation and baseline measures for physiologic variables, and inferior data analyzing strategies. According to their review, much of the published research is generally flawed in at least several of these dimensions. To address these weaknesses, Flor et al[17] conducted a study comparing patients with TMD to patients with back pain and asymptomatic control subjects. Their results indicated that while imagining a personally relevant stressor, patients with TMD displayed more EMG activity in the right masseter than did the other experimental groups. These findings were interpreted to suggest that patients with TMD do display greater EMG activity in masseter muscles during imagination of stressful memories than do people who do not have TMD. The linkage between such overactivity and the development of painful muscles, however, has not been demonstrated as of yet.

In addition to monitoring EMG activity, research has expanded to include other parameters exploring the nature of the increased levels of anxiety commonly associated with TMD. Some studies have differentiated patients with TMD from control subjects on heart rate and skin conductance responses to laboratory stressors,[18] whereas others have shown no differences based on these methods.[19–21] Recently, Carlson et al[3] compared the responsivity of patients with TMD and masticatory muscle pain to that of age- and sex-matched control subjects. Subjects' heart rate, blood pressure, skin temperature, and masseter EMG levels were monitored at rest and in response to a standard laboratory stressor involving mental arithmetic. The results indicated that patients with TMD had greater heart rate and systolic blood pressure increases in response to the stressor as compared to matched asymptomatic control subjects. Overall, the findings in these small number of studies represent initial efforts to define the scope of the role of the autonomic nervous system in facial pain and to search for reliable parameters to measure such activity.

The majority of the laboratory research in chronic facial pain has used cognitively oriented laboratory stressors; few studies have explored how TMD may affect an individual's sensitivity and responsivity to painful stimuli. Three studies[22–24] reported lower pain threshold levels in patients with TMD as compared to control subjects. Another study[25] reported opposite results. The use of different experimental pain stimuli (pressure versus electric), varied pain-dependent measures, and unclear patient selection criteria in these studies likely contributed to the lack of consistency regarding distinctions between patients with TMD and matched control subjects.

Although many orofacial pain researchers and clinicians agree that patients with TMD experience increased anxiety, there are many questions regarding the origin and scope of this psychologic component and its ultimate relationship to facial pain. In the present investigation, it was hypothesized that patients with TMD would be more anxious in general and more sensitive to pain stimulation than would a group of carefully matched asymptomatic control subjects. Along with controlling for many of the methodologic problems in previous studies employing EMG measures, the present study included several physiologic parameters for a more comprehensive examination of the activity of the autonomic nervous system in people with TMD.[8,14,15]

Materials and Methods

Subjects

The subjects in this study included 23 female facial pain patients recruited from the Orofacial Pain Center at the University of Kentucky, College of Dentistry (Lexington, KY), and 23 female weight-matched (1 to 10 lb) and age-matched (0 to 3 years) asymptomatic control subjects recruited from the university population (including introductory psychology courses for research credit). The mean age of the pain sample was 26.9 years,

and the mean age of the asymptomatic control subjects was 27.4 years. In a small number of cases (six cases), the age range was broadened slightly (within 5 years) to accommodate the weight limits. Pain patients with a primary diagnosis of masticatory muscle pain[26] of at least 6 months in duration, with no clinical evidence of joint pathology or dysfunction, were included in this study. This was determined by an evaluating dentist in the Orofacial Pain Center who conducted a thorough dental examination to ensure patients met the inclusion criteria for masticatory muscle pain as set forth in the "Research Diagnostic Criteria" of Dworkin and LeResche.[26] Patients with joint involvement in their pain symptoms were specifically excluded in this study to ensure that the patients with TMD in this sample represented a homogeneous group of persons reporting exclusively masticatory muscle pain.

All subjects were screened on the following criteria: mean resting blood pressure less than 140/90 mm Hg, no previous injury to the nondominant hand, and overall good health (with the exception of the muscle pain in pain patients). In addition, asymptomatic control subjects were also screened for any TMD or pain. Subjects who met the screening criteria were asked for further information including: age, any medication use, awareness of bruxism, use of oral contraception, and the date of the first day of their last menstrual cycle. Subjects with regular menstrual cycles did not participate in the experiment during the ovulation phase of their menstrual cycle because of potential changes in pain sensitivity at this phase.[27]

Design

A quasiexperimental design was employed to compare patients with chronic facial muscle pain to asymptomatic control subjects. Based on psychologic and physiologic measures, any baseline differences between these two groups as well as differences in responsivity to a stressor, pain perception, and recovery from the stressors were investigated. The research was approved by the Institutional Review Board for the Protection of Human Subjects.

Laboratory Stressors

Stressor 1: Mental Arithmetic (MA). This stressor consisted of a serial subtraction task (subtracting "13" from a four-digit number) and lasted 1 minute per trial. There were a total of three consecutive trials with 1-minute rests between trials. Physiologic data were collected throughout this period, and the Emotion Assessment Scale (EAS stress[3,28]) was given after the third trial to assess any physical or emotional changes resulting from this stressor. To control for head and jaw movement, EMG activity was also recorded during the 1-minute rest periods between each math trial.

Stressor 2: Pain Stimulus (PS). A modification of the Forgione-Barber Pressure Pain Stimulator was used to induce acute tonic pain for up to a 1-minute period. With this device, focal pressure is applied with a 4.5-lb weight concentrated on the second phalanx of the middle finger of the nondominant hand. The device provides a standard PS and has been used successfully in previous studies.[22,23,29,30] It is a tonic PS in which the pain intensity builds gradually, resembling clinical pain more so than a phasic PS.[31]

Dependent Physiologic Measures

Heart Rate and Blood Pressure. Heart rate (HR) and systolic (SBP), diastolic (DBP), and mean arterial (MAP) blood pressures were measured using a Paramed 9200 automated blood pressure cuff (Paramed Technology, Mountain View, CA).[32] The cuff was placed on the subject's dominant arm to prevent possible inaccurate measurements during the PS to the nondominant hand.

Skin Temperature (ST). Skin temperature was measured using a thermistor probe (J & J Manufacturing, Poulsbo, WA) attached to the index finger of the nondominant hand. Data were recorded during 1-second intervals and averaged over each experimental period.

Electromyography. Using a computerized physiograph (I-330, J & J), EMG activity was recorded using silver/silver chloride miniature surface electrodes attached according to standard laboratory guidelines.[33] The EMG band pass filter setting was at 25 to 1,000 Hz. Activity in the left and right masseter regions and in the left and right temporalis regions was recorded. The data were integrated over 1-second epochs, and a mean score was computed for each experimental phase. Since EMG data were managed similarly for both groups, and each subject contributed mean scores for each experimental phase, no attempt was made to remove artifacts from the EMG recordings, since they were likely to effect both group means.

Respiration Rate (RR). Respiration rate was recorded with a J & J I-330 respiration module. A strain gauge was attached across the chest and abdomen of the subject. Data were recorded over 1-minute epochs. Because of the unavailability of this equipment at the beginning of the project, res-

piration rate was only collected in a subset of the total sample of subjects (15 patients, eight control subjects).

Continuous Heart Rate (ContHR). Continuous heart rate was recorded using a J & J I-330 photoplethysmograph. The photosensor was place on the fourth finger of the nondominant hand. Data were averaged over 1-minute periods. Because of the unavailability of this equipment at the beginning of the project as well, the continuous heart rate data were collected for only a subset of the total sample of subjects (16 patients, 15 control subjects).

Psychologic Measures

State-Trait Personality Inventory (STPI). This inventory was designed to measure anxiety, anger, and curiosity. The state scales measure the degree to which an individual is experiencing these three emotional states at the time of testing, and the trait scales measure an overall tendency to experience these emotions.[34]

Emotion Assessment Scale (EAS). This scale was designed to measure eight fundamental dimensions of emotional responses (surprise, fear, disgust, anger, guilt, anxiety, sadness, and happiness). The EAS contains 24 visual analog scale (VAS) items and has a split-half reliability of .94.[28] This measure was given before the math stressor, after the math stressor, after the PS, and after the postbaseline period.

Pain Indexes

Visual Analog Pain and Expectancy/Coping Ratings. Visual analog scales have been found to be sensitive measures of pain intensity.[35] Four separate measures containing 10-cm VAS were used in this study. One measure concerned the intensity of current facial pain symptoms that the individual was experiencing (VAS-CPI). Another measure was used to determine the intensity of pain expected by the individual prior to both stressors (VAS-E). A VAS measure was also used at this time to measure the individual's perceived ability to cope with the PS (VAS-C). The third measure was used during the pain stressor to assess the intensity of pain (PR). The VAS-CPI, the VAS-E, and the PR were anchored at one end with "no pain" and "worst possible pain" at the other end. The VAS-C was anchored at one end with "no coping ability" and "most coping ability" at the other end. The VAS-E and VAS-C were also administered after the pain stressor (eg, indicate how much pain you experienced).

Pain Threshold (PTHRESH). Subjects were asked to report when they initially felt the sensation of pain. This measure was recorded as the time (in seconds) from the beginning of the PS until the subjects said "now."

McGill Pain Questionnaire–Short Form (MPQ-SF). This questionnaire is a shorter version of the original MPQ, which was designed to measure the qualitative aspects of pain. This short form contains four measures of pain, including sensory and affective scores as well as two indexes of total pain experience, and it correlates highly with the original MPQ.[36]

Procedure

Prior to the experimental session, subjects gave their informed consent and passed all screening criteria. Based on information collected at the initial session, subjects did not participate in the experimental session during the ovulation phase of their menstrual cycle (day 15 to day 21), with the exception of any subjects using oral contraception. In addition, subjects with the consent of the prescribing health professional were asked to refrain from any pain medication use 8 hours before the experimental session.[37,38] At the experimental session, subjects completed a battery of psychologic measures (STPI, VAS-E, VAS-C, VAS-CPI) while the experimenter attached the blood pressure cuff and physiologic recording leads according to standard laboratory guidelines.[33]

After the recording leads were attached and the questionnaires were completed, the subject rested quietly for a 5-minute adaptation period followed by a 5-minute baseline period. After the baseline period the EAS (pre-math stressor) was given. The MA period then occurred after an anticipatory period lasting 1 minute. After the third trial of the MA, the EAS (after the math stressor) was again administered.

After the subject had completed the EAS (after the math stressor) and had rested for 5 minutes, the PS period began. Each subject placed the middle finger of her nondominant hand in the finger pressure device. Each subject was asked to keep her finger in the device as long as possible without exceeding 60 seconds. (Subjects were informed that they could withdraw their fingers at any time prior to the end of the 60-second trial). The time (seconds) was recorded when the subjects initially reported pain (PTHRESH). When the trial was finished, subjects completed the pain indexes (MPQ-SF, EAS [after the pain stimulus], PR, VAS-E, VAS-C).

After completion of the pain indexes, subjects rested quietly for a 5-minute postbaseline period. Subjects then completed the EAS (postbaseline period). The experimental session was then finished and subjects were given a debriefing form.

Statistical Analyses

Initial baseline differences between patients and control subjects were analyzed via a series of *t* tests using the psychologic and physiologic indexes as dependent measures. Repeated measures of analysis of variance (ANOVA) were used to evaluate the impact of each stressor across groups (within subjects) and to assess potential group differences in response to the stressors. Responsivity to the MA stressor was evaluated by comparing the EAS and physiologic measures collected during the MA period to the corresponding baseline measures. Recovery from the math stressor was measured by comparing the physiologic measures that were collected during the period following the stressor with the initial baseline period measures. Responsivity to the PS was evaluated by comparing the physiologic measures collected during the PS period to the corresponding baseline measures. The postbaseline EAS and physiologic measures collected following the PS were compared to the corresponding baseline measures. The *t* tests to compare pain perception indexes between the two groups were also computed.

Results

Baseline Comparisons

As expected, pain patients (TMD) reported current pain symptoms more so than did matched control subjects (MC) (TMD $\bar{X}$ = 2.61, MC $\bar{X}$ = 0.13) ($t[45] = 5.63$, $P < .001$). The patients with TMD also indicated greater anxiety based on the trait measure of the STPI than did the control subjects (TMD $\bar{X}$ = 23.83, MC $\bar{X}$ = 19.35) ($t[45] = 2.40$, $P < .03$). The patients with TMD indicated greater levels of anxiety ($\bar{X}$ = 24.70) relative to control subjects $\bar{X}$ = 12.87) ($t[45] = 2.55$, $P < .02$) on the EAS. Based on the EAS, patients with TMD had greater self-ratings of sadness (TMD $\bar{X}$ = 9.96, MC $\bar{X}$ = 3.87) ($t[45] = 2.47$, $P < .02$) and guilt (TMD $\bar{X}$ = 5.65, MC $\bar{X}$ = 2.04) ($t[45] = 2.02$, $P < .05$) than did control subjects. Patients with TMD also had greater respiration rates than did control subjects (TMD $\bar{X}$ = 15.17, MC $\bar{X}$ = 12.49) ($t[22] = 2.81$, $P < .02$). There were no other baseline physiologic or psychologic differences between the two groups (Table 1).

Table 1 Physiologic Data for Experimental Session

	Baseline	MA	MA recovery	Pain stimuli	Postbaseline
TMD patients					
SBP (mm Hg)	115.00	122.70	155.35	119.87	114.26
DBP (mm Hg)	62.83	68.61	62.70	67.09	64.74
MAP (mm Hg)	88.04	94.65	87.35	92.04	88.39
HR (bpm)	71.65	82.52	71.87	74.56	72.17
RR (rpm)	15.17*	13.69	15.40	15.38	15.46*
ST (°F)	80.48	79.91	80.06	78.18	77.86
ContHR (BPM)	70.43	77.64	72.17	72.66	71.05
R masseter EMG (μV)	2.24	4.56	2.38	5.38	2.10
L masseter EMG (μV)	2.27	4.93	2.48	6.46	2.19
R temporalis EMG (μV)	4.24	6.28	4.23	7.62	4.02
L temporalis EMG (μV)	3.72	6.52	4.07	6.67	3.77
Control subjects					
SBP (mm Hg)	109.78	117.04	109.70	115.52	110.17
DBP (mm Hg)	66.48	70.96	64.04	68.61	65.13
MAP (mm Hg)	86.78	95.48	87.00	93.00	87.44
HR (bpm)	69.30	79.65	67.52	71.96	68.09
RR (rpm)	12.49*	13.59	13.74	12.77	12.73*
ST (°F)	80.80	80.26	80.22	78.30	77.09
ContHR (bpm)	68.01	72.71	67.79	69.49	67.27
R masseter EMG (μV)	2.08	4.01	1.87	5.54	1.70
L masseter EMG (μV)	2.28	4.53	2.04	5.72	2.01
R temporalis EMG (μV)	4.45	6.73	4.05	7.31	3.42
L temporalis EMG (μV)	3.54	5.06	3.28	6.46	3.43

*$P < .05$ in between-groups comparison.

Reactivity to MA

The experimental groups displayed statistically significant ($P < .05$) emotional and physiologic reactivity to the math stressor (see Table 1), indicating that the task imposed a notable demand on the participants. Differences in emotional responses during the math stressor were found between the two groups, with patients who had TMD reporting greater self-ratings of anger ($F[1,44] = 4.08$, $P < .05$) as compared to control subjects (Table 2). There were no other statistically significant differences in emotional reactivity to the math stressor. There was a statistically significant difference between respiration rates of the patients with TMD during the math task as compared to the control subjects ($F[1,21] = 9.41$, $P < .01$) (see Table 1); there were no other statistically significant differences between patients and control subjects in physiologic reactivity to the math stressor.

Recovery From the MA Stressor

Physiologic and emotional measures indicated that there were no statistically significant differences between patients with TMD and control subjects during the recovery from the math stressor.

Reactivity to the PS

Each of the experimental groups displayed statistically significant ($P < .05$) emotional and physiologic reactivity to the PS (see Table 1). However, there were no group differences between patients and control subjects on emotional or physiologic responses to the PS.

Table 2 Emotional Reactivity to Laboratory Challenges

	TMD patients		Control subjects	
	Baseline	MA stressor	Baseline	MA stressor
Surprise	12.87	29.74	9.13	27.48
Fear	10.65	20.34	7.61	14.74
Disgust	6.09	28.78**	2.78	16.09**
Anger	7.04	34.47**	2.70	15.74**
Guilt	5.65*	35.56	2.04*	20.87
Anxiety	24.70*	45.52	12.87*	36.52
Sadness	9.96*	28.39	3.87*	16.22
Happiness	23.61	10.91	23.35	13.13

*$P < .05$ baseline between-groups comparison.
**$P < .05$ repeated measure ANOVA between groups.

Postbaseline Differences

There were no differences in emotional measures between the two groups when compared between the initial baseline and postpain levels. In addition, there were no postbaseline differences on the physiologic measures of BP, HR, skin temperature, respiration, or EMG activity.

Pain Indexes

A measure labeled as pain tolerance was added in the investigation of pain sensitivity after it was noted that many (n = 28) individuals did not keep their finger in the PS for the entire 60-second trial period. Pain tolerance was defined as the number of seconds the subject kept her finger in the finger pressure device during the 60-second trial. There were no statistically significant differences between patients with TMD and control subjects based on the pain tolerance measure (TMD $\bar{X}$ = 37.00 seconds, MC $\bar{X}$ = 43.09 seconds) ($t[45] = 1.06$, $P < .30$) or pain threshold levels (TMD $\bar{X}$ = 13.17 seconds, MC $\bar{X}$ = 10.96 seconds) ($t[45] = .54$, $P < .60$). These groups also did not differ in terms of reporting the intensity of the pain experienced during the trial (PR: TMD $\bar{X}$ = 7.04, MC $\bar{X}$ = 6.39) ($t[45] = 1.06$, $P < .30$). Based on the McGill Pain Questionnaire, the groups indicated no differences in the sensory (TMD $\bar{X}$ = 12.96, MC $\bar{X}$ = 13.78) ($t[45] = 0.43$, $P < .70$) or affective (TMD $\bar{X}$ = 2.04, MC $\bar{X}$ = 1.83) ($t[45] = 0.30$, $P < .80$) aspects of the pain stimulation, as well as present pain intensity levels (TMD $\bar{X}$ = 2.96, MC $\bar{X}$ = 2.52) ($t[45] = 1.53$, $P < .20$). Patients did not significantly differ from control subjects in terms of expectancies for the intensity of the PS (TMD $\bar{X}$ = 4.00, MC $\bar{X}$ = 3.74) ($t[45] = 0.38$, $P < .80$) or in self-ratings of coping ability (TMD $\bar{X}$ = 7.91, MC $\bar{X}$ = 8.49) ($t[45] = 1.08$, $P < .30$).

Discussion

Anxiety has consistently been a factor differentiating patients with chronic facial pain from asymptomatic control subjects in previously published reports.[3,39] In this study, patients with masticatory muscle pain also reported greater anxiety based on self-report trait and state measures than did matched asymptomatic control subjects. Additionally, patients with muscle pain described themselves as feeling greater guilt and sadness at the beginning of the session than did control subjects. The potential relevance of these differences in emo-

tional states between patients and asymptomatic control subjects was illustrated in a recent study by Kinney et al.[40] Kinney et al[40] assessed possible psychologic disorders in 50 patients with chronic TMD. Their findings indicated that 84% of the patients met lifetime criteria for an Axis I disorder (DSM III-R[41]), as compared to general population base rates of 29% to 38% (somatoform pain disorder was excluded). Based on lifetime diagnostic criteria, the most frequently diagnosed Axis I disorders among patients with TMD were major depression (74%), substance disorders (30%), and anxiety disorders (24%). Although many etiologic questions remain regarding the co-occurrence of psychologic disorders and chronic pain, the results of the present study and others[3,39,42] highlight the potential utility of including a formal assessment of psychologic status in the initial evaluation of chronic facial pain.

Heightened emotional reactivity to environmental stressors may contribute to the greater levels of distress, anxiety, and depression often found in patients with facial pain.[24,39,42] The patients in the present sample, for example, responded to the math stressor with greater anger than did the asymptomatic control subjects. Such heightened response patterns during stressors may limit or obstruct various coping strategies available to patients with facial pain. In addition, this may influence their abilities to manage subsequent life stressors, which may, in turn, lead to increased levels of anxiety and depression.

The results of this study emphasize the importance of assessing emotional responses to laboratory stressors; however, this has often been neglected in favor of using primarily physiologic measures of stress responsivity in studies of this patient population. A majority of the research and clinical treatment of chronic facial muscle pain for almost four decades has focused on facial muscle EMG activity in an attempt to link increased muscle activity and pain/dysfunction. This study controlled for many of the problems often found in past research such as matching control subjects based not only on sex, but also age and weight criteria, including an adaptation period, and controlling for head (jaw) movement during the math stressor task.[14,15] With the implementation of these control subjects, the results of this study indicated no differences between patients and control subjects in bilateral masseter muscle and temporalis muscle EMG activity. These results are consistent with other studies that have included an adaptation period prior to baseline recording[16,43] and suggest the importance of exploring the contributions of other factors, such as emotional reactivity to the onset and maintenance of muscle pain disorders.

Flor and Turk[16] have emphasized the importance of using laboratory tasks that subjects perceive as stressful; we concur with that recommendation, but we also believe it is valuable for subjects, both symptomatic and asymptomatic, to be exposed to standardized stressors so that meaningful comparisons of physiologic reactivity can be made. Our data, both physiologic and emotional, indicated that the standard laboratory tasks used in the present study were experienced as stressful by both groups of the study. The use of standard laboratory stressors is an effective and efficient way of ensuring that subjects and patients are exposed to equivalent laboratory challenges.

Based on the present findings and those of others,[14,15] laboratory EMG activity may not generally be a useful measure in differentiating patients with facial pain from control subjects using current reactivity protocols involving standard stressors. Although the use of the personally relevant stressor approach may distinguish patients with pain from asymptomatic subjects for individual muscle sites,[17] consideration must be given to controlling for *(1)* the intensity of the stressor and *(2)* the variability associated with EMG measurements. The large degree of individual variability and the ability to voluntarily control masticatory muscles during experimental sessions may further complicate the use of EMG in monitoring TMD patients and control subjects. Generally, the available research indicates that EMG monitoring is more effective when used in direct treatment such as in biofeedback training for patients with acute muscle spasms or in patients with observable muscle hyperactivity.[44]

The inconsistent support for the links between muscle activity and facial pain has led to a search for alternative mechanisms to explain chronic facial muscle pain. A previous study in this laboratory found that patients with facial pain had higher heart rate and systolic blood pressure responses to a standard stressor as compared to control subjects.[3] This finding was not replicated in the present study and may reflect the difference in the experimental protocol in which a pain stimulus was added, possibly inducing a ceiling effect for reactivity measurements. This ceiling effect could be obscuring the differences in reactivity between patients with pain and control subjects. Another possibility is that there truly are no differences in autonomic functioning between patients with facial pain and asymptomatic control subjects.

Further research is needed to explore the role of autonomic function in the pathogenesis of deep muscle pain.

The differences in respiration rates between patients with TMD and control subjects, while representing a subset of the overall sample, may be an important measure to include in evaluating patients with TMD. The results indicated greater baseline respiration rates in patients compared to control subjects. Although this measure did not differentiate patients from control subjects in responsivity to the stressors, the baseline elevation in respiration rate found in patients with TMD was maintained throughout most of the session. Chronic, increased respiration rates can lead to pH changes in the blood, which is associated with increased neuronal excitability, decreased peripheral blood flow, and hyperirritability.[45] Future studies should include measures of respiratory function to further explore this domain of physiologic functioning as it relates to patients with TMD.

In this study, there were no differences between patients with TMD and control subjects on various standard pain measures associated with the acute pain stimulation. In addition, expectations regarding the intensity of the pain stimulus and the ability to cope with the pain stressor did not differ between patients with TMD and control subjects. These findings suggest that patients with facial pain psychologically and physically experience acute pain stimuli similarly to the asymptomatic matched subjects. Previous research involving this issue has produced inconsistent results.[22–25] Possible sources of variability in these earlier studies include the use of different experimental pain stimuli, application to different anatomic sites, and lack of formal operationalization of the dependent variables associated with pain stimulation. Various terms such as *detection threshold*, *discomfort threshold*, *pain threshold*, and *tolerance* have been used. The definitions and use of these terms vary and likely contribute to the mixture of reported findings. Further research is needed to evaluate possible variations in facial pain patients' sensitivity and responsivity to pain associated with the intensity of current symptoms and/or site of the application of the experimental pain stimulus.

Overall, this study represents a controlled examination of the potential differences between patients with TMD and asymptomatic control subjects. Features of the present study that could be improved include counterbalancing the presentation of stressors and increasing the sample size. Nonetheless, patients with TMD demonstrated greater levels of emotional distress overall and overresponded emotionally to stressful stimuli. The psychophysiologic nature of TMD, however, does not appear to include increased levels of facial muscle activity during evaluations with standard stressors but may be linked to other physiologic variables such as respiration pattern. These present data emphasize the need to include both emotional and physiologic indexes to further examine the factors associated with the differentiation of patients with TMD from asymptomatic control subjects.

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References

1. Moulton RE. Oral and dental manifestations on anxiety. Psychiatry 1955a;18:261–273.
2. Moulton RE. Psychiatric considerations in maxillofacial pain. J Am Dent Assoc 1955b;51:408–414.
3. Carlson CR, Okeson JP, Falace DA, Nitz AJ, Curran SL, Anderson D. Comparison of psychologic and physiologic functioning between patients with masticatory muscle pain and matched controls. J Orofacial Pain 1993;7:15–22.
4. Moss RA, Garrett JC. Temporomandibular joint dysfunction syndrome and myofascial pain dysfunction syndrome: A critical review. J Oral Rehabil 1984;11:3–28.
5. Rugh JD, Solberg WK. Psychological implications in temporomandibular pain and dysfunction. Oral Sci Rev 1976; 7:3–30
6. Marbach JJ. The TMPDS personality: Fact or fiction? An overview. NY State Dent J 1992;58:23–26.
7. Malow RM, Olson RE, Greene CS. Myofascial pain dysfunction syndrome: A psychological disorder. In: Golden CJ, Alcapparras SS, Strider FD, Graber B (eds). Applied Techniques in Behavioral Medicine. New York: Grune and Stratton, 1981:101–133.
8. Rudy TE. Psychophysiological assessment in chronic orofacial pain. Anesth Prog 1990;37:82–87.
9. Lacey JI, Lacey BC. Verification and extension of the principle of autonomic response-stereotypy. Am J Psychol 1958;71:50–73.
10. Laskin DM. Etiology of the pain-dysfunction syndrome. J Am Dent Assoc 1969;79:147–153.
11. Dolan EA, Keefe FJ. Muscle activity in myofascial pain-dysfunction syndrome patients: A structured clinical evaluation. J Craniomandib Disord Facial Oral Pain 1988;2:101–151.
12. Gervais RO, Fitzsimmons GW, Thomas NR. Masseter and temporalis electromyographic activity in asymptomatic, subclinical, and temporomandibular joint dysfunction patients. J Craniomand Pract 1989;7:52–57.
13. Kydd WL. Psychosomatic aspects of temporomandibular joint dysfunction. J Am Dent Assoc 1959;59:31–44.

14. Lund JP, Widmer CG. An evaluation of the use of surface electromyography in the diagnosis, documentation, and treatment of dental patients. J Craniomandib Disord Facial Oral Pain 1989;3:125–137.
15. Lund JP, Widmer CG, Schwartz G. What is the link between myofascial pain and dysfunction? In: Van Steenberghe DV, De Laat A (eds). EMB of Jaw Reflexes in Man. Leuven, Belgium: Leuven University Press, 1989:427–444.
16. Flor H, Turk DC. Psychophysiology of chronic pain: Do chronic pain patients exhibit symptom-specific psychophysiological responses? Psychol Bull 1989;105: 215–259.
17. Flor H, Birbaumer N, Schugen MM, Lutzenberger W. Symptom-specific psychophysiological responses in chronic pain patients. Psychophysiology 1992;29:452–460.
18. Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain-dysfunction syndrome. J Behav Med 1989;12:397–406.
19. Katz JO, Rugh JD, Hatch JP, Langlais RP, Terezhalmy GT, Borcherding SH. Effect of experimental stress on masseter and temporalis muscle activity in human subjects with temporomandibular disorders. Arch Oral Biol 1989;34:393–398.
20. Mercuri LG, Olson RE, Laskin DM. The specificity of response to experimental stress in patients with myofascial pain dysfunction syndrome. J Dent Res 1979;58: 1866–1871.
21. Rugh JD, Montgomery GT. Physiological reactions of patients with TM disorders vs. symptom-free controls on a physical stress task. J Craniomandib Disord Facial Oral Pain 1987;1:243–250.
22. Malow RM, Grimm L, Olson RE. Differences in pain perception between myofascial pain dysfunction patients and normal subjects: A signal detection analysis. J Psychosom Res 1980;24:303–309.
23. Malow RM, Olson RE. Changes in pain perception after treatment for chronic pain. Pain 1981;11:65–72.
24. Molin C, Edman G, Schalling D. Psychological studies of patients with mandibular pain dysfunction syndrome. 2: Tolerance for experimentally induced pain. Swed Dent J 1973;66:15–23.
25. Hagberg C, Hellsing G. Perception of cutaneous electrical stimulation in patients with craniomandibular disorders. J Craniomandibular Disord Facial Oral Pain 1990;4:120–125.
26. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examinations and Specifications, Critique. J Craniomandibular Disord Facial Oral Pain 1992;6:301–355.
27. Goolkasian P. Cyclic changes in pain perception: An ROC analysis. Percept Psychophys 1980;27:499–504.
28. Carlson CR, Collins FL, Porzelius J, Stewart JF, Nitz AJ, Lind C. The assessment of emotional reactivity. J Psychopathol Behav Assess 1989;11:313–325.
29. Breuhl S, Carlson CR, McCubbin JA. The relationship between pain sensitivity and blood pressure in normotensives. Pain 1992;48:463–467.
30. Forgione AG, Barber TX. A strain gauge pain stimulator. Psychophysiology 1971;8:102–106.
31. Chen ACN, Dworkin SF, Haug J, Gehrig J. Human pain responsivity in a tonic pain model: Psychological determinants. Pain 1989;37:143–160.
32. Theisen V, Peterson E. Accuracy and acceptance of an automated blood pressure device in hypertension screening. Presented at the National Conference of High Blood Pressure Control, Las Vegas, 1987.
33. Fridlund AJ, Cacioppo JT. Guidelines for human electromyographic research. Psychophysiology 1986;23:567–589.
34. Spielberger CD, Gorsuch RL, Lushene R, Vagg P, Jacobs GA. State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychology Press, 1977.
35. Huskisson EC. Visual analog scales. In: Melzack R (ed). Pain Measurement and Assessment. New York: Raven Press, 1983:33–77.
36. Melzack R. The short-form McGill Pain Questionnaire. Pain 1987;30:191–197.
37. Brennum J, Kjeldsen M, Jensen K, Stachelm Jensen T. Measurements of human pressure-pain thresholds on fingers and toes. Pain 1989;38:211–217.
38. Yang JC, Richlin D, Brand L, Wagner J, Clark WC. Thermal sensory decision theory indices and pain threshold in chronic pain patients and healthy volunteers. Psychosom Med 1985;47:461–468.
39. Marbach JJ, Lennon MC, Dohrenwend BP. Candidate risk factors for temporomandibular pain and dysfunction syndrome: Psychosocial, health behavior, physical illness and injury. Pain 1988;34:153–165.
40. Kinney RK, Gatchel RJ, Ellis E, Holt C. Major psychological disorders in chronic TMD patients: Implications for successful management. J Am Dent Assoc 1992;123: 49–54.
41. Diagnostic and Statistical Manual of Mental Disorders (DSM III-R). Washington, DC: American Psychiatric Association, 1987.
42. Schnurr RF, Brooke RI, Rollman GB. Psychosocial correlates of temporomandibular joint pain and dysfunction. Pain 1990;42:153–165.
43. Moss RA, Adams HE. Physiological reactions to stress in subjects with and without myofascial pain dysfunction symptoms. J Oral Rehabil 1984;11:219–232.
44. Funch DP, Gale EN. Biofeedback and relaxation therapy for chronic temporomandibular joint pain: Predicting successful outcomes. J Consult Clin Psychol 1984;52:928–935.
45. Fried R. The Hyperventilation Syndrome: Research and Clinical Treatment. Baltimore, MD: JohnS Hopkins, 1987.

Resumen

Respuestas Emocionales y Psicológicas a Pruebas de Laboratorio: Pacientes con Desórdenes Temporomandibulares Versus Sujetos de Control

Este estudió exploró los factores psicológicos y fisiológicos que diferencian a los pacientes con desórdenes temporomandibulares (n = 23), de los sujetos de control asintomáticos acoplados con el grupo experimental, en cuanto a su sexo, edad y peso. Cada persona completó varios cuestionarios psicológicos estandard y luego fue sometida a dos pruebas de laboratorio (aritmética mental y estímulo al dolor-presión). Los resultados indicaron que los pacientes con desórdenes temporomandibulares tenían mayores frecuencias respiratorias en reposo y se habían quejado de mayor ansiedad, tristeza y culpabilidad en comparación a los sujetos de control. Como respuesta al examen matemático, los pacientes con desórdenes temporomandibulares, reaccionaron con mayor enfado en comparación a los pacientes de control. No hubo diferencias entre los pacientes con desórdenes temporomandibulares y los sujetos de control, en cuanto a las medidas de dolor u otra variable medida en el examen de estímulo de dolor-presión. Además, no se detectaron diferencias en los niveles electromiográficos entre los pacientes con desórdenes temporomandibulares y los sujetos de control. Los resultados son discutidos en relación a sus implicaciones en la etiología y tratamiento de esta serie de desórdenes que es común y debilitante.

Zusammenfassung

Emotionale und physiologische Reaktionen auf Herausforderungen im Labor bei Patienten mit Myoarthropathien gegenüber einer entsprechenden Kontrollgruppe

Diese Studie untersuchte die psychologischen und physiologischen Faktoren, welche 23 Patienten mit Myoarthropathie von geschlechts-, alters- und gewichtsentsprechenden asymptomatischen Kontrollpersonen unterschieden. Alle Personen füllten einige psychologische Standardfragebögen aus und wurden anschliessend zwei Stressoren im Labor unterworfen (Mentalarithmetische Herausforderung und Druckschmerz). Die Resultate zeigten, dass Patienten mit Myoarthropathien höhere Ruheatmungsfrequenzen, stärkere Ängstlichkeit, Unwohlsein und Schuldgefühle im Vergleich zu den Kontrollpersonen hatten. Auf den Mathematik-Stressor reagierten die Myoarthropathiepatienten mit stärkerer Verärgerung als die Kontrollpersonen. Es traten keine Unterschiede zwischen den 2 Gruppen bezüglich Schmerzempfindlichkeit auf. Zusätzlich waren auch keine Unterschiede der EMG-Aktivitäten beider Gruppen festzustellen. Die Resultate werden hinsichtlich ihrer Auswirkung auf die Ätiologie und die Behandlung von Myoarthropathien diskutiert.