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Stress Induced Oral Behaviors and Facial Pain

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Facial pain is frequently associated with environmental stress and emotional distress. One hypothetical mechanism by which stress is translated into pain is through stress induced motor function (*e.g.*, *teeth clenching*, *grinding*, *nail biting*). *Existent data partially supports these stress-hyperactivity models although they have also come under theoretical and empirical attack. The purpose of this study was to examine the relationship between oral behaviors and pain in an analog sample of facial pain sufferers and student controls. Subjects engaged in a controlled clenching task and reported on subjective facial pain intensity and unpleasantness at 5 specified times over the subsequent 48 hours. A one-way ANCOVA indicated group differences in self reported oral habits* ($p < .05$) *with the facial pain group reporting great frequency of oral habits. Two repeated measures ANCOVAs* (*i.e.*, *pain intensity and pain unpleasantness*), *controlling for baseline pain ratings*, *indicated a between groups effect with facial pain sufferers experiencing significantly greater pain over the 48 hours postexperiment* ($p < .05$). *This study supports a hyperactivity model of facial pain and provides clues about relevant factors in facial pain development.*

KEY WORDS: facial pain; myofascial pain; oral habits; hyperactivity.

Hyperactivity models of facial pain (e.g., myofascial pain, headache) postulate that psychological stress is translated into maladaptive motor function (i.e., oral behaviors such as teeth clenching, teeth grinding, nail biting, gum chewing) that is directly and causally related to the development of pain. This relationship first gained general acceptance following the publication of Laskin's (1969)

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seminal paper that outlined a "stress-oral behavior-myospasm-pain" model. This model, and the derivative models that followed (e.g., Eversole & Machado, 1985; Haber, Moss, Kuczmierczyk, & Garrett, 1983; Parker, 1990; Scott, 1981), attempt to account for the higher than normal degree of emotional distress often seen in facial pain patients (Butterworth & Deardorff, 1989; McCreary, Clark, Merril, Flack, & Oakley, 1991; Rudy, Turk, Zaki, & Curtin, 1989; Schnurr, Brooke, & Rollman, 1990; Schwartz & Gramling, 1997) and the efficacy of treatments such as biofeedback/relaxation training and cognitive/behavioral interventions (e.g., Crider & Glaros, 1999; Funch & Gale, 1984; Moss, Wedding, & Sanders, 1983; Scott & Gregg, 1980), although such relationships do not imply causation (Stohler, 1999).

Critical to these hyperactivity models is the causal chain that links stress, mandible use, and consequent pain (Haber et al., 1983; Laskin, 1969; Moss et al., 1984; Parker, 1990). While this causal chain is generally assumed, there is little empirical data to confirm or refute its validity. One early body of research that has directly addressed the link between mandible use and facial pain was conducted by Christensen and others (e.g., 1976, 1981a,b). This series of influential studies examined the mechanical operation of the mandibular system via experimental teeth clenching and pain in the masticatory musculature. Several of these studies required healthy subjects with normal dentition and no history of facial pain to engage in a teeth clenching exercise and self monitor for subsequent pain during a specified postexperimental period. The authors found asymptomatic subjects developed pain about the face and head following experimental teeth clenching. In addition, the pain persisted in several subjects for days (up to 7 days) after experimental teeth clenching. More recent work (e.g., Glaros, Tabacchi, & Glass, 1998; Nicholson, Lakatos, & Gramling, 1999) has tended to support these earlier findings.

Aside from bruxism (i.e., teeth grinding), little research has been directed toward delineation of the in vivo oral behaviors that may promote facial pain. Identification of specific oral behaviors would provide concrete targets for behavioral interventions. Moss, Sult, and Garrett (1984) were among the first to differentiate facial pain sufferers from asymptomatic controls in a student sample based on the self reported frequency of in vivo oral habits (e.g., nail biting, gum chewing, diurnal clenching, and grinding). Specifically, facial pain subjects endorsed engaging in many of these oral habits more frequently than controls. A related study by Villarosa and Moss (1985) had subjects engage in one of four common oral behaviors (i.e., jutting the jaw forward, jutting the jaw to one side, cupping the chin in the hand, teeth clenching) and self monitor for pain several hours postexperiment. They also found that the oral behaviors led to localized head and face pain. Moss and colleagues have extended these findings to tension headache and common migraine as well (Moss, Ruff, & Sturgis,

1984; Moss 1987; Moss et al., 1988; Moss, Lombardo, Hodgson, & O'Carroll, 1989).

These two avenues of research represent important empirical steps in support of the stress-hyperactivity model and make a compelling, albeit incomplete, case for the role of oral behaviors in the etiology of some facial pain conditions. In addition, several methodological changes might be instituted in order to enhance external validity and strengthen conclusions. First, the clenching procedure employed by Christensen (1979, 1981a,b) allowed subjects to define for themselves the degree of masticatory muscle contraction in which they were willing to engage. A number of mediating variables—including demand characteristics, individual differences in pain tolerance, or inherent resilience of the masticatory musculature—might affect the degree to which a given subject contracts the masticatory muscles under experimental conditions. Christensen (1981b) himself confirms that there was considerable intersubject variability in the degree of contraction and acknowledges this as a study limitation. In addition, subjects were at times employing a level of tonic muscle contraction that far exceeded what is likely to occur in vivo (e.g., often exceeding 200 microvolts; Christensen, 1971). Villarosa and Moss (1985) did not assess EMG activity during their laboratory oral behavior exercises and may also be vulnerable to the problem of intersubject variability or excessive levels of muscle contraction. This problem might be resolved by providing subjects with EMG feedback that would reduce intersubject variability by directing subjects to maintain a specified force band (Glaros et al., 1998). The use of EMG levels required for the various in vivo oral habits identified by Moss and associates (1984, 1984, 1987, 1989) could provide a rational benchmark for defining such a force band range.

The following study was undertaken with the principal purpose of studying facial pain development in participants with and without self reported facial pain symptoms following an experimental clenching procedure in much the same manner as Christensen (1970, 1971, 1981a,b). A secondary purpose was to replicate the work of Moss and colleagues (1984) by assessing the differential frequency of self reported oral behaviors in participants with and without facial pain symptoms. The methodology employed here was designed to (1) closely replicate the findings of these two important lines of research, and (2) better control the experimental clenching parameter. First, subjects were required to maintain their degree of masticatory muscle contraction within a specified force band. Second, the force band employed was rationally derived via pilot testing of the oral habits found by Moss and colleagues to differentiate facial pain samples from asymptomatic controls (Moss et al., 1984, 1987, 1989). Third, EMG feedback was provided using a commercially available biofeedback system in order to enhance the clinical feasibility and relevance of the procedures.

The intent was to maintain greater control over how subjects responded to the experimental task demands and to make those demands consistent with what little is known about potential behaviors occurring in the natural environment.

METHOD

Participants

Forty-seven individuals (11 men and 36 women) recruited from undergraduate university classes participated in this study. Student subjects received course credit for participation. Subjects were placed into two diagnostic groups (Facial Pain = 18 and Asymptomatic Controls = 29) based on self report of symptoms adapted from Bush, Whitehill, and Martelli (1989), Moss et al. (1984), and self report portions of the Research Diagnostic Criteria for Temporomandibular Disorders (Widmer, Huggins, & Friction, 1992). Questions inquired about facial pain symptomatology such as a history and frequency of pain in and about the ears, jaw, temples or cheeks, headache, mandibular joint sounds, uncomfortable bite, limited range of motion, and trismus (i.e., tonic muscle contractions). Facial pain subjects were identified if they reported recent and persistent (although not necessarily continuous) headache and/or pain in the temporomandibular joint or surrounding musculature, pain in the ears, temples, or cheeks, headache of any type, and at least one of the following concomitant symptoms: trismus, mandibular joint sounds, trigger points in the muscles of mastication, or an uncomfortable bite. Individuals were excluded if they reported a history of head or facial injury, other chronic pain condition, recent dental treatment, use of prescription medications for pain or related conditions, use of prescription medications for psychiatric conditions, or pregnancy. Control subjects had to report no symptoms of facial or other chronic pain condition and not satisfy exclusion criteria. It should be noted that the intent here was not to study a specific diagnostic entity but rather to focus on the relationship of oral habits to a specific symptom (i.e., pain located in the head and neck region).

The sample was predominately white (79%) and female (77%) with a mean age of 26.56 years ($SD = 8.81$). Group differences on demographic variables (age, gender, race) were assessed, and age was the only demographic variable that showed significant group differences via t test $t\ (1,46) = 2.6$, $p < .05$ with the facial pain group ($X = 25.5$ years, $SEM = 1.67$) being significantly older than the controls ($X = 21.63$ years, $SEM = .58$). Thus, age was used as a covariate in subsequent analyses. Group differences for gender and race were assessed using two Pearson chi-square analyses and were not significant for either gender or identified racial affiliation.

Self Report Measures

Oral Habits Questionnaire

Oral habits were assessed with a questionnaire adapted from the work of Moss et al. (1984). The questionnaire consists of 11 oral habit items (e.g., chewing gum, biting pens/pencils.) rated for the frequency with which each habit was generally engaged on a 10-point Likert scale verbally anchored at each extreme with "Never" and "Almost Always." This questionnaire has shown adequate test-retest reliability and successfully differentiated TMD sufferers from controls in a previous study (Schwartz, Gramling, Mancini, & Baldwin, 1990).

Pain Assessments

Pain assessments employed two Visual Analog Scales (VAS) for each recording period tapping both the intensity and unpleasantness dimensions of pain. VAS line endpoints were verbally anchored with "Not At All Intense" on the left endpoint and "The Most Intense Pain Imaginable" on the right for the intensity dimension. The unpleasantness dimension was labeled similarly with "unpleasant" substituted for "intensity." These two dimensions of pain have been demonstrated to be consistent and independent aspects of the pain experience using VAS scales (Price, McGrath, Raffi, & Buckingham, 1983). The VAS format is straightforward, easily understood, and affords greater sensitivity and variability than many other assessment formats (Ohnhaus & Adler, 1975). Numerous studies have demonstrated that the VAS format is a valid measure of pain intensity and other pain related sequela (e.g., Kremer, Atkinson, & Ignelzi, 1981; Price et al., 1983; Jensen, Karoly, & Braver, 1986; Gramling & Elliott, 1992).

Procedures

Preexperimental Preparation

Following informed consent, all participants completed the oral habits questionnaire and were then prepared for psychophysiological recording and feedback via standard procedures (Sturgis & Gramling, 1997). EMG responses were collected from the masseter muscle on the subject's dominant side (based on hand preference) using disposable silver/silver chloride electrodes. Electrode placement was over the masseter muscle using a vertical electrode placement as recommended by Basmajian and Blumenstein (1989). The system was grounded to the back of the subject's dominant hand.

Psychophysiological recording and feedback was accomplished via the 1991 version of the Biocomp 2001 biofeedback system. The Biocomp 2001 is a commercially available IBM compatible psychophysiological recording/biofeedback system that is capable of collecting eight channels of physiological data via modality specific sensor leads. System control is accomplished through the computer using a specialized Biocomp interface coupler card that fits any 8-bit computer slot. Psychophysiological feedback can be either audio and/or visual. Visual feedback was used for the purposes of this study. A bandwidth of 80–200 Hz was used to filter out EEG and other electrical artifact. This is the default setting for the system software and most likely to be used in its clinical applications.

Experimental Clenching

Participants were instructed to bite down hard on their molars for a 20-second interval followed by a 20-second resting intermission for an 8-minute experimental period. Alternating contraction/resting periods were signaled by both the experimenter and data appearing on the computer monitor. Participants were instructed to maintain a force band of 20–50 microvolts during the contraction phase, and EMG levels were visually displayed numerically for subjects on the computer monitor. The EMG force band was determined based on pilot testing of the various oral habits identified by Moss et al. (1984, 1987) with a separate sample. The force band is somewhat broad in order to account for the variability inherent in the various oral behaviors assessed. The resting intermission required immediate cessation of contraction such as to create minimal EMG activity.

Pain Assessment

Using VAS diaries provided during the experiment, participants reported on both pain intensity and unpleasantness at a preclenching baseline, immediately following the experimental clenching procedure, and at for four specified times over the subsequent 48 hours. Instructions for using each dimension of VAS scale were drawn from procedures used by Price et al. (1983). Scores for pain intensity and unpleasantness were determined by measuring the distance in millimeters from the left anchor point to the hash mark made by the subject.

RESULTS

This study employed a 2×5 (group $\times$ time of pain assessment) mixed model design. Group membership (student facial pain, asymptomatic controls)

constituted the between groups factor, and pain ratings over the 48 hours postexperimental clenching (immediately following, 1/2 hour post, at bedtime night one, the following morning, and at bedtime night two) served as the within subjects factor. Because we were interested in the change in pain ratings over time for the time period post clenching, baseline pain ratings were covaried out of the data analyses (along with age). Both age and baseline pain ratings served as significant covariates in all analyses ($p < .05$). Results of an evaluation of assumptions of normality, homogeneity of variance-covariance, linearity, and multicollinearity were satisfactory for all the dependent measures.

A one-way ANCOVA was used to evaluate group difference in total score for self reported oral habits. A significant between groups effect was found [$F(1,46) = 14.26$, $p < .05$], with the facial pain group reporting more oral habits than controls. Group means are presented in Table 1 for the individual oral habits and cumulative score. Note that the individual oral habits were not specifically analyzed, but uncorrected univariate p values are provided for descriptive purposes.

EMG data for both the clenching and relaxing periods of the clenching task were analyzed for group differences using ANCOVA. EMG was calculated as the arithmetic mean of the 20-second on/off epochs. Each epoch data point represented a mean of EMG activity for that 20-second interval. All subjects were able to maintain an average of at least 20 microvolts during the clenching phase of the procedure. Groups did not differ significantly in their mean "on" (clenching) phase or "off" (relax) phase. Therefore, groups did not differ significantly in the degree of motor function engaged in during the clenching task.

Table 1. Group Means and Standard Deviations for Self Reported Oral Habits

	Facial pain subjects		No pain controls	
Variable	Mean	SD	Mean	SD
Diurnal Bruxing	5.4	2.8	2.4	1.8*
Pipe Smoking	1.0	0.0	1.4	1.5
Lip Biting	5.4	2.4	4.4	2.4
Gum Chewing	4.9	2.9	3.9	2.4
Instrument Playing Involving Lips/Chin	1.4	1.1	1.8	2.3
Telephone Receiver B/W Chin & Shoulder	5.6	2.9	3.5	2.3
Tongue Biting	5.7	2.8	3.9	2.9
Pen/Pencil Chewing	5.6	3.3	3.3	2.5*
Jaw Posture	3.3	2.2	1.8	1.6*
Resting Head in Hands	7.3	2.3	5.8	2.8
Nail Biting	4.8	3.6	3.4	3.1
Total	50.4	18.0	35.6	12.1*

Note: Facial Pain Students $N = 18$; Student Controls $N = 29$.
*$p < .05$.

Table 2. Group Means and Standard Deviations for VAS Ratings of Pain Intensity Following the Bruxing Exercise

	Facial pain subjects		No pain controls	
Variable	Mean	SD	Mean	SD
Baseline	22.6	28.3	2.2	4.0*
Immediate Rating	51.4	34.9	23.1	26.6
30 min. Post Bruxing	37.9	36.1	11.2	19.6*
Night One	34.4	29.8	7.3	3.9*
Next Morning	26.1	32.6	3.9	4.0*
Night Two	25.0	29.1	3.6	4.3*

Note: Facial Pain Subjects $N = 18$; No Pain Controls $N = 29$.
*$p < .05$.

Tables 2 and 3 present the group means and standard deviations by time period for pain intensity and unpleasantness ratings respectively. The intensity and unpleasantness dimensions of pain were tested separately. Results of the repeated measures ANCOVA for pain intensity indicated a significant between groups effect using Hotellings criteria [$F(4,37) = 5.65$, $p < .05$]. Univariate ANCOVAs for each time period, using a Bonferroni adjustment, were also significant for each of the time periods except immediately after clenching: 30 minutes post, $F(1,40) = 10.65$, $p < .05$; first night, $F(1,40) = 13.06$, $p < .05$; next morning $F(1,40) = 79.17$, $p < .05$; second night, $F(1,40) = 48.05$, $p < .05$. The facial pain group reported significantly greater pain intensity over the 48-hour recording period. Results of the repeated measures ANCOVA for pain unpleasantness also indicated a significant between groups effect using Hotellings criteria [$F(4,37) = 14.79$, $p < .05$]. Univariate ANCOVAs for pain unpleasantness at each time period, using a Bonferroni adjustment, were also significant for each of the

Table 3. Group Means and Standard Deviations for VAS Ratings of Pain Unpleasantness Following the Bruxing Exercise

	Facial pain subjects		No pain controls	
Variable	Mean	SD	Mean	SD
Baseline	28.2	30.2	2.8	3.3*
Immediate Rating	71.6	33.6	46.3	33.3
30 min. Post Bruxing	45.8	26.2	17.0	20.2*
Night One	38.3	30.4	8.0	16.8*
Next Morning	30.6	32.7	4.1	4.2*
Night Two	27.0	28.9	3.9	4.4*

Note: Facial Pain Subject $N = 18$; No Pain Controls $N = 29$.
*$p < .05$.

assessment periods except immediately after clenching: 30 minutes post, $F(1,37) = 10.65$, $p < .05$; first night, $F(1,40) = 13.78$, $p < .05$; next morning, $F(1,40) = 50.83$, $p < .05$; second night, $F(1,40) = 51.54$, $p < .05$. The facial pain group also reported significantly greater pain unpleasantness over the 48-hour recording period.

The effect for time was also significant using Hotellings criteria [$F(8,39) = 4.91$, $p < .05$], indicating that both pain intensity and unpleasantness ratings decreased over time. The univariate post hoc tests for the time factor of both pain intensity and unpleasantness employed Reverse Helmert contrasts with a Bonferroni confidence interval. This analysis indicated significant change ($p < .05$) in both pain intensity and unpleasantness at the level over the 48-hour recording period. Pain reductions occurred primarily within the first 30 minutes postclenching for all groups and then began to level off. The group by time interaction was not significant.

DISCUSSION

The purpose of this study was to examine the relationship between masticatory muscle use and pain development in and about the face and head, as has been suggested by the various stress-hyperactivity models of facial pain development (Eversole & Machado, 1985; Haber et al., 1983; Laskin, 1969; Moss et al., 1984; Parker, 1990; Scott, 1981). The goal was to replicate and expand on the two literatures cited by integrating them conceptually and empirically with certain methodological improvements. The data presented here support the hyperactivity notion of facial pain development and is consistent with existing literatures of both Christensen and associates (1976, 1981a,b) and Moss et al. (1984, 1987, 1988, 1989). Importantly, both facial pain subjects and no pain controls manifested pain when engaging in a clenching task designed to correspond to the types of oral behaviors found in vivo.

These data indicate that both healthy asymptomatic controls and analog facial pain sufferers developed facial pain following a teeth clenching task (even after accounting for differences in baseline pain ratings in the facial pain group). In addition, both groups report that this pain is experienced as considerably unpleasant (the affective component of pain). Facial pain subjects continued to experience considerable increases in pain after 48 hours, even after accounting for initial pain ratings. Pain duration for asymptomatic subjects was relatively short lived and they reported little residual pain at the end of the 48-hour assessment period. Given that the group by time interaction was not significant, pain intensity and unpleasantness declined for both groups at a similar rate over the 48 hour assessment period. This finding differs somewhat from Christensen (1971) whose findings indicated that pain persisted for a much longer duration

in some "healthy" subjects. This is probably in part due to modifications in the clenching procedure, which required less mandibular work from subjects than the procedure used by Christensen. Additionally, data reported here are consistent with the work of Moss et al. (1984, 1987, 1988, 1989). Facial pain subjects drawn from the student population reported considerably more frequent oral habits (e.g., chewing gum, nail biting) than did the asymptomatic controls.

Several caveats must be acknowledged. First, this was an analog study and no collateral diagnostic procedures aside from self report were used. Therefore no specific formal diagnosis is associated with group membership, making extrapolation to a specific diagnostic clinical group problematic. However, the focus of this study was primarily on the relationship of oral behavior to the symptom of facial pain, rather than on a specific diagnostic group per se. Nevertheless, application of this methodology and these results to a more specifically operationalized clinical sample would be a logical extension of this study. Second, while some speculation about the etiological relationship of oral behaviors to facial pain might be gleaned from this study, the data presented here cannot address directly the etiological role these oral habits play in facial pain development. It does seem reasonable, however, to suggest they are likely to play a critical role in the maintenance of facial pain in some clinical cases. In addition, this study did not include a control manipulation, and we cannot be entirely certain facial pain subjects would not have experienced increased pain in the absence of the clenching procedure. However, given the temporal contiguity of pain increases with clenching, we do feel there is a causal relationship. Finally, and perhaps most importantly, pain ratings (i.e., the magnitude of pain change being reported) could have been subject to experimenter demands as participants clearly understood that increases in pain were an expectation. However, this almost certainly does not relate to the existence of pain itself.

Recently, hyperactivity models have come under criticism for being inconsistent with the pattern of EMG activity observed in specific masticatory muscle groups during agonist and antagonist activities (Chapman, 1986; Lund & Widmer, 1989; Lund, Donga, Widmer, & Stohler, 1991; Stohler, 1999). In other words, EMG activity during tonic hyperactivity of muscle (agonist muscle activity) is often not found to be higher in TMD sufferers when compared with controls (Moller, Sheikh-ol-eslam, & Lous, 1984; Christensen & Hutchins, 1992). This follows from the commonsense notion that sore, fatigued muscles are less capable of the same degree of work, and fatigue is viewed as a protective factor (Lund et al., 1991). This literature has implications for hyperactivity models (i.e., equivocal results in EMG activity and reactivity studies), but does not necessarily weaken the underlying assumption that stress induced oral behaviors are causally related to the pain. For example, Christensen (1981) has pointed out that while reduced capacity inherent in fatigue may protect muscle tissue, it does not necessarily and correspondingly reduce subjective pain experience. Therefore two convergent causal factors may be at work. The first is the

greater work load exhibited by facial pain sufferers in terms of the frequency with which they engage in various oral habits; the second is the reduced capacity of the masticatory musculature to tolerate such work. The reduction in work capacity could result from an inherent weakness of the masticatory system (e.g., degenerative joint diseases), oral habits induced over time (e.g., myofascial pain, tension headache), or an interaction. Unfortunately, little is really known about the natural history of the various disorders that produce facial pain. Importantly, these relationships have yet to be adequately tested, but may account for the debate over the validity of hyperactivity models. There is certainly no reason to expect that these relationships would be identified by or reflected in typical EMG activity/reactivity studies for any given set of oral behaviors and related oral mechanics.

Finally, one critical aspect of the hyperactivity models that has not been adequately addressed within the context of this or any other study concerns critical factors that maintain the oral habits that foster the pain, given their obvious punishing consequences. Hyperactivity models have assumed that engaging in the oral habits is reinforced by their tension reduction (arousal reduction) qualities or traditional secondary gain (Haber et al., 1983). Nevertheless, no study to date has conceptually or empirically addressed the specific mechanisms by which these oral behavior patterns might reduce arousal and therefore be reinforced despite the consequent pain. Identification of such biobehavioral mechanisms (e.g., schedule-induced behaviors, Gramling, Grayson, Sullivan, & Schwartz, 1997; Nicholson et al., 1999) and their adaptive function (if any) would have important treatment implications for behavior management of such conditions and provide powerful support for hyperactivity models of facial pain development. In fact, a habit reversal program based on the schedule induction paradigm has shown initial efficacy (Gramling, Neblett, Grayson, & Townsend, 1996).

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