# Changes in Pressure-Pain Thresholds of the Jaw Muscles During a Natural Stressful Condition in a Group of Symptom-Free Subjects

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**Aims:** *To investigate the effects of a natural emotional stressor on pressure-pain thresholds (PPTs) of the masticatory muscles of symptom-free subjects.* **Methods:** *Sixteen healthy dental students were selected before they undertook an academic examination. Sixteen gender-matched students who were not exposed to an examination served as controls. The 2 groups of students were monitored in parallel on 5 separate days over a 1-month period: 2 days before the examination (T1), on the day of the examination (T2), 2 days after (T3), 1 month after (T4), and again after another 2 days (T5). On the day of the examination (T2), the control students were only required to complete a brief, non-demanding questionnaire. On each day, the following parameters were assessed: PPTs of the masseter, the anterior temporalis, and the Achilles tendon; state anxiety; and present stress (measured on a visual analog scale [VAS]). Furthermore, in the students undergoing the examination, venous blood samples for assessment of ß-endorphin levels were obtained at T2 and T5.* **Results:** *In the stressed students, the PPTs of the masticatory muscles and the Achilles tendon were significantly lower (analysis of variance [ANOVA], $P \leq 0.02$) on the day of the examination (T2) and on the days nearest the exam (T1, T3), and state anxiety and present stress were significantly higher (ANOVA, $P \leq 0.003$). No significant change was found in ß-endorphin plasma levels ($P > 0.05$). In the control group, PPTs, state anxiety, and present stress did not change significantly (ANOVA, $P > 0.05$).* **Conclusion:** *The results support a relationship between psychologic stress and pressure-pain sensitivity of the masticatory muscles.*

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**Key words:** masticatory muscles, sensory thresholds, pain measurement, psychological stress, beta-endorphin

Psychologic stress has been implicated in several aspects of temporomandibular disorders (TMD). Stressful and unpleasant life events have been shown to be very prevalent in patients suffering from TMD.[1–3] Interestingly, TMD patients report their facial pain as being exacerbated or aggravated during stressful conditions.[4] Furthermore, the outcome of several treatments for TMD may be modified by psychologic factors such as anxiety, stress, and depression.[5–7]

In an attempt to identify the key mechanisms by which TMD pain symptoms are linked to psychologic stress, extensive research has focused on the stress-induced motor responses of the masticatory muscles in both healthy subjects and TMD patients.[8,9] Yet despite the evidence that stress and anxiety may play an important role in affecting pain sensitivity in human subjects,[10–12] little

attention has been paid to the perceptual responses of the masticatory muscles to psychologic stress.

The pain sensitivity of the masticatory muscles may be assessed by determining their pressure-pain threshold (PPT), which is defined as the amount of applied pressure necessary for a subject to report the onset of pain. Pressure algometry has proven a reliable and valid measurement method in patients with a variety of musculoskeletal pain syndromes and in asymptomatic subjects.[13–15]

It has been emphasized that the validity of stress responses in human subjects is strongly influenced by the type of stress stimuli and the degree of personal relevance.[8] Indeed, a standard laboratory stimulus may not have a perceived "stressfulness" equivalent to that of an ecological stimulus (eg, a real life event). Furthermore, the experiential and physiologic correlates of an acute laboratory stress may be considerably different from those elicited during a prolonged period of natural stress.

The purpose of the present study was to assess whether a naturally stressful condition induced changes in the PPTs of the human jaw muscles in a group of symptom-free dental students. The stressor selected consisted of a very difficult academic examination. The effectiveness and the validity of this emotional stressor have already been demonstrated.[16,17] Since pain perception can be influenced by peripheral endogenous opioids,[18] we further investigated whether plasma β-endorphin levels were related to this stressful condition. One brief communication of this study has appeared elsewhere.[19]

## Materials and Methods

### Subjects

Sixteen healthy subjects (9 men and 7 women), students in the Dental School at the University of Naples, were recruited into this study before they undertook a very difficult academic examination. The mean age (± SD) was 22 ± 2 years (range 21 to 31 years). Sixteen gender-matched subjects, postgraduate students not undergoing any examination, were selected as controls. The mean age (± SD) was 26 ± 2 years (range 24 to 30 years). All the subjects had a complete natural dentition and were free from orofacial pain and TMD as assessed by means of Research Diagnostic Criteria.[20] Informed consent was obtained from all subjects prior to the start of the study.

### Procedure

The 2 groups were monitored in parallel on 5 separate days over an approximately 1-month period: 2 days before the examination (T1), immediately after the examination (T2), 2 days after (T3), 1 month after (T4), and again after another 2 days (T5). On the day of the examination (T2), the control students were only required to complete a brief, non-demanding questionnaire. The last 2 days (T4 and T5) corresponded to a period during which the students were not exposed to examinations, immediately after the summer holidays.

On each day, the following parameters were assessed in a sequential order: PPTs of the masseter, anterior temporalis, and Achilles tendon; state anxiety; and present stress. In addition, in the examination group, venous blood samples for assessment of β-endorphin plasma levels were obtained about 1 hour after the exam and again 1 month later during the stress-free period. To avoid learning bias, all the subjects underwent a preliminary training session a few days before the start of the study. During this session, the subjects became acquainted with and learned about the procedure and measurements. All the assessments were taken blindly by a single examiner. All the sessions lasted about half an hour and took place in the morning in a warmed room. During the sessions, each subject was seated upright on a dental chair with his/her head leaning on a headrest. The subjects received no information about the aim and/or expectations of the study.

**Algometric Measurements.** The PPTs were determined with an electronic algometer (Somedic AB). The instrument and the procedure are described in detail elsewhere.[21] Briefly, the tip of the algometer applied to the skin had a surface of 1 $cm^2$ and a rate of pressure increase of approximately 20 kPa/sec was chosen. The PPT was determined as the point at which the subject sensed a change from a feeling of pressure to a feeling of pain. Pressure-pain thresholds were assessed at 4 muscular sites, 2 on the masseter (M1 and M2) and 2 on the anterior temporalis (T1 and T2). To ensure precise relocation of these sites in each session, a transparent pliable plastic template was aligned to the ear, to the labial margin, and to the eye, and the location of each site was marked. An additional measurement was performed over the Achilles tendon, which was selected as a non-muscular control site. The sites were measured in the order M1, M2, T1, T2, followed by the Achilles tendon, with approximately a 5-second interval between sites. Four PPT

measurements were made at each recording site, with a 2-minute rest interval between trials. A total of 10 minutes elapsed for the recording of PPT at all sites. Since the first PPT of a session is generally higher than consecutive measurements,[13] this value was discarded, and each PPT was determined as the mean value of the successive 3 trials. In previous studies,[21,22] within-muscle comparison of sites (M1 versus M2 and T1 versus T2) did not reveal significant differences; consequently, the data from the 2 sites of each muscle investigated were averaged to obtain single estimates of masseter and temporalis PPT values.

**Stress and Anxiety Assessment.** Psychometric and self-report measures were used to evaluate subjects' psychologic changes during the study. State anxiety was assessed by means of the state part of Spielberger's State-Trait Anxiety Inventory (STAI).[23] This consists of a brief questionnaire of 20 statements, with raw scores ranging from 20 to 80, that is designed to assess how anxious the subject is "currently." The trait part of the STAI was omitted because it was not relevant to the hypothesis of the present study.

Present stress was measured with a visual analog scale (VAS) after careful instruction. The scale consisted of a 100-mm line oriented horizontally, with the left endpoint indicating "I do not feel stressed at all" and the right endpoint corresponding to "I could not feel more stressed."[5,24] The VAS is reliable, valid, and sensitive to change when used to assess subjective states such as pain intensity and unpleasantness or different emotions.[25,26] Ratings were made by the subjects marking the position on the scale that best represented the assessment of their present stress.

**Plasma β-Endorphin Assessment.** Venous blood samples for β-endorphin measurements were obtained in the examination group immediately after the examination (stress period) and 1 month later (stress-free period). Plasma levels of immunoreactive β-endorphin were determined with a radioimmunoassay kit (Peninsula Laboratories Europe, Ltd). Briefly, blood samples were collected into a chilled syringe and then transferred into a polypropylene tube containing ethylenediaminetetraacetic acid (EDTA) (1 mg/mL of blood) and aprotinin (500 KIU/mL of blood) at 0°C. After centrifugation at 0°C for 15 minutes at 1,600 rpm, the plasma was decanted, aspirated, immediately frozen in dry ice at −70°C, and stored until assayed. Two plasma aliquots were extracted in parallel and assayed in duplicate. Each aliquot was added with an equal amount of 0.1% trifluoroacetic acid (TFA) (buffer A) and centrifuged at 6,000 rpm for 20 minutes at 4°C. The plasma solution was loaded onto a separation column. Before the samples were layered, the separation column containing 200 mg of C18 (Cat. No. Riksepcoli) was equilibrated 3 times by washing it with 60% acetonitrile in 0.1% TFA (buffer B) (1 mL once) followed by buffer A (3 mL 3 times). Next, the column was slowly washed with buffer A (3 mL twice), the peptide was slowly eluted with buffer B (3 mL once), and the eluant was collected in a polypropylene tube. The eluant was then evaporated to dryness in a centrifugal concentrator. The residue was then dissolved in 250 μL radioimmunoassay buffer included in the radioimmunoassay kit. Additional tubes containing known amounts of β-endorphin standard were analyzed in parallel.

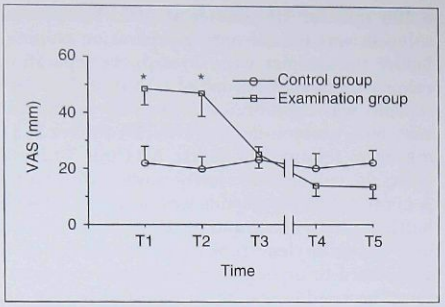
### Statistical Analyses

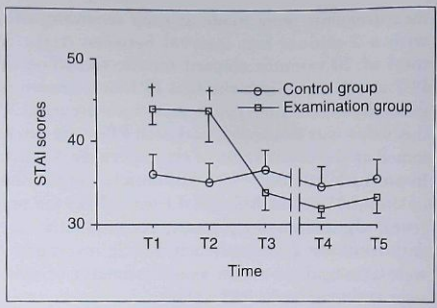
The data were analyzed by means of parametric and non-parametric statistical tests. Repeated-measures analysis of variance (ANOVA) was used to evaluate changes in PPTs within subjects. The VAS and STAI scores were analyzed by Friedman ANOVA because several samples could not pass the Kolmogorov-Smirnov normality test. The assumption of sphericity for the repeated-measures ANOVA was tested by means of the Mauchly's test[27] and, where appropriate, degrees of freedom were corrected with the Greenhouse-Geisser epsilon.[27]

When statistical significance was obtained, post-hoc multiple comparisons were performed by the Newman-Keuls test and by the Dunns test. Correlations were performed by Spearman correlation analysis. Paired and unpaired comparisons were performed by the Wilcoxon-Pratt and Mann-Whitney tests. All tests were 2-tailed. Statistical significance was considered when the $P$ value was less than 0.05. All the data were analyzed with commercial statistical software packages (Prism 2.01 GraphPad Software and SPSS for Windows 8.0).

## Results

Both present stress and state anxiety scores (Figs 1a and 1b) differed significantly across the 5 sessions in the examination group (Friedman ANOVA, $P \leq 0.003$). The ratings of present stress and state anxiety did not change significantly in the control group (Friedman test, $P > 0.05$). Multiple-comparison tests, performed by means of the Dunns test, are summarized in Figs 1a and 1b.




**Figs 1a and 1b** Mean present stress (*left*) and state anxiety (*right*) in the students taking the examination versus the control subjects. Vertical bars indicate 1 standard error. A 1-month interval elapsed between T3 and T4, and all the other sessions were separated by a 2-day interval. Both present stress and state anxiety ratings differed significantly across the 5 sessions in the examination group (Friedman analysis of variance, $P \leq 0.003$), whereas the ratings did not change significantly in the control group (Friedman test, $P > 0.05$). *Significantly different ($P < 0.05$) from T4 and T5; †significantly different ($P \leq 0.05$) from T3, T4, and T5 (both post-hoc multiple-comparison test).

The PPTs of the masseter, the anterior temporalis, and the Achilles tendon (Figs 2a to 2c) differed significantly across the 5 sessions in the examination group (ANOVA, $P \leq 0.02$). The PPTs in the control group did not show significant changes (ANOVA, $P > 0.05$). Multiple-comparison tests, performed by means of the Newman-Keuls test, are summarized in Figs 2a to 2c.

In the examination group, the relative changes in PPTs between the stressful period and the stress-free period amounted to about 20% for the masseter muscle, 15% for the anterior temporalis, and 20% for the Achilles tendon. A Spearman correlation analysis was performed between the relative changes in PPTs and stress/anxiety obtained on the day of examination (T2) and 1 month later (T4). The correlation coefficients were generally low; a significant correlation was obtained only between the changes in present stress and the changes in the PPT of the anterior temporalis (Table 1). The relative changes in both PPT and stress/anxiety measurements did not differ significantly between males and females (Mann-Whitney test, $P > 0.05$).

Plasma levels of β-endorphin (mean ± SD) assessed on the day of the examination (7.3 ± 3.2 pg/mL) and during the stress-free period (8.1 ± 3.0 pg/mL) did not differ significantly (Wilcoxon-Pratt, $P > 0.05$).

## Discussion

The findings of the present study revealed that the PPTs of the jaw muscles are lowered during a natural stressful event. The significant elevation of present stress and state anxiety ratings on the day of the examination indicates that the students forming the examination group were much more stressed during this session and supports the validity of the emotional stressor selected for this study. Stress and anxiety had decreased 2 days after the examination and reached almost a steady level on subsequent days. Conversely, the control group showed a fairly constant level of present stress and state anxiety, and the PPTs did not change significantly during the study.

As revealed by post-hoc multiple comparisons, the decreases in PPTs found in the examination group were not restricted to the day of the academic examination, but they were also evident in the days leading up to and following the examination (ie, T1 and T3). This finding may be ascribed to the type of stimulus chosen as a stressor. In previous studies, stress has been induced mainly by means of challenging laboratory tasks, including mental arithmetic,[28] stressful imagery,[29] and psychomotor tasks.[30] Although a "standard" laboratory stimulus is relatively robust for inducing a predictable acute stress, psychologic and physiologic responses to stress occur suddenly and begin to fade away immediately after the cessation of

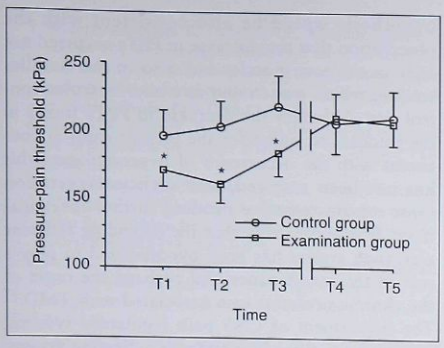



Fig 2a Masseter.

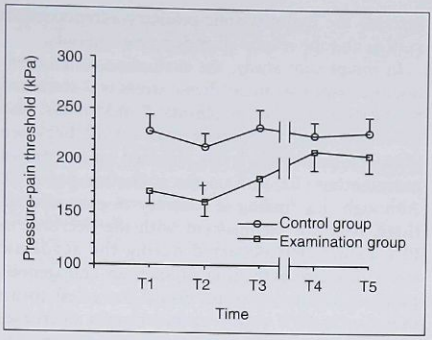



Fig 2b Anterior temporalis.

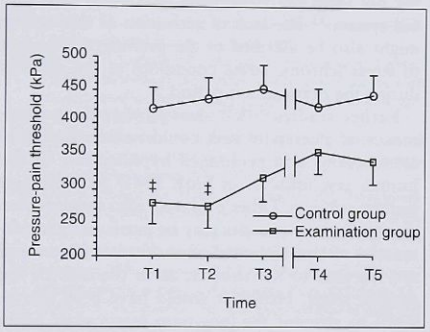



Fig 2c Achilles tendon.

**Figs 2a to 2c** Mean algometric measurements of the masseter, anterior temporalis, and Achilles tendon in the students taking the examination versus the control subjects. Vertical bars indicate 1 standard error. A 1-month interval elapsed between T3 and T4, and all the other sessions were separated by a 2-day interval. The PPTs of the masseter, anterior temporalis, and Achilles tendon differed significantly across the 5 sessions in the examination group (ANOVA, $P \leq 0.02$), whereas the PPTs did not show significant changes in the control group (ANOVA, $P > 0.05$). *Significantly different ($P < 0.05$) from T4 and T5; †significantly different ($P < 0.01$) from T4 and T5; ‡significantly different ($P < 0.05$) from T4 and T5 (post-hoc multiple-comparison test).

stimuli.[8,24] In the present study, we have attempted to ensure that the stressful stimulus endured for a long time and had a personal relevance to the subjects by using a real-life event, ie, students undergoing an examination. Such a natural stressor may elicit emotional and cognitive states different from those associated with an acute laboratory stress.[31] The quality, magnitude, and persistence of this stressor can explain its prolonged effect on both psychologic and algometric measures.

The decrease in PPTs found in this study may be ascribed to several factors. A large number of physiologic responses involving the central and peripheral nervous system are activated during stress. Stress and anxiety increase sympathetic activity and the release of epinephrine at the sympathetic terminals, which may sensitize or directly activate nociceptors.[32] Stress and anxiety may also

**Table 1** Spearman Correlation Coefficients for Changes (T2 to T4) in PPT and Stress/Anxiety Measurements

| | Location | | |
|---|---|---|---|
| Measurement | Masseter | Anterior temporalis | Achilles tendon |
| Present stress | –0.26 | –0.55* | –0.48† |
| State anxiety | 0.06 | –0.28 | –0.33 |

*$P < 0.05$; †$0.05 < P < 0.1$.

activate the hypothalamic-pituitary-adrenocortical system and the release of endogenous opioids.

In the present study, the evaluation of neuroendocrine responses to academic stress was restricted to the measurement of plasma β-endorphins, and no significant difference was found between plasma β-endorphin levels obtained on the day of examination (T2) and on the stress-free day (T5). Although this finding is contrary to our expectations, it is not inconsistent with the decrease in PPT values that occurred during the academic stress, because activation of such an endogenous pain regulatory system would have led to an increase in PPT values rather than a decrease. Hence, although the academic examination was considered an "extremely stressful" event by the majority of the students investigated, it was probably not sufficient to activate the endogenous opioid system.[33] The lack of activation of this system might also be ascribed to the prolonged exposure to stress (chronic stress condition) of the subjects during the examination period.[31]

Earlier studies[30,34,35] showed that the performance of a stressful task could induce abnormal contractions and prolonged hyperactivity in the human jaw muscles of both TMD patients and healthy subjects. Other evidence[8] also suggests that the masticatory muscles may be particularly hyperreactive to stressful conditions that last longer and are relevant to the subject, as in the case of the present study. Hence, it would have been worthwhile to monitor the long-term masticatory activity of the students in their natural environment by means of portable electromyographic recorders.[36] Unfortunately, this was not done in the present study, and a potential relationship between pressure-pain sensitivity of the jaw muscles and masticatory motor activity therefore cannot be inferred.

It should be emphasized that the changes in PPTs found in the present study were only weakly correlated with the changes of VAS ratings, and no significant correlation was found with anxiety levels (STAI). Other psychologic factors may correlate better with variations in pain thresholds. According to the perceptual disruption theory,[12] stress and anxiety contribute to alterations in pain perception by disinhibiting central nervous system (CNS) structures involved in the regulation of attention (eg, ascending reticular activating system). This disruption may result in hypervigilance, which is associated with an increased attention to or amplification of nociceptive stimuli. It could be hypothesized that such hypervigilance would occur in students undergoing an academic examination that is a long-lasting stressful condition. This hypothesis would be also consistent with the observation that the decrease in PPTs occurred not only in the jaw muscles but also in the Achilles tendon, which was chosen as a non-muscular control site. Although the decrease in PPTs found in the students recruited for the present study is consistent with the occurrence of hypervigilance, this has not been assessed, and such interpretation must remain tentative pending further investigations. It is noteworthy that the ascending reticular activating system has been hypothesized to play a role in the maintenance and perhaps the onset of the chronic orofacial pain associated with TMD.[37] The impairment of CNS pain regulatory systems, including the ascending reticular activating system, may be also responsible for the generalized enhanced pain sensitivity found in TMD patients and for the lower PPTs of their masticatory muscles.[21,38]

## Conclusions

The findings of the present study suggest that the PPTs of the jaw muscles in healthy subjects are influenced by a naturally emotionally stressful situation. Changes in PPTs were not related to β-endorphin plasma levels. Psychologic changes during the experimental procedures showed only a weak correlation with changes in PPTs. Additional studies should be carried out to replicate these findings and to investigate the potential role of other psychologic and physiologic measures that may be altered during a natural stress condition.

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## References

1. Rugh JD, Solberg WK. Psychological implications in temporomandibular pain and dysfunction. Oral Sci Rev 1976;7:3–30.
2. Speculand B, Hughes AO, Goss AN. The role of recent stressful life events experience in the onset of TMJ dysfunction pain. Community Dent Oral Epidemiol 1984;12:197–202.
3. Dahlstrom L. Psychometrics in temporomandibular disorders. An overview. Acta Odontol Scand 1993;51:339–352.
4. Suvinen TI, Hanes KR, Gerschman JA, Reade PC. Psychophysical subtypes of temporomandibular disorders. J Orofac Pain 1997;11:200–205.

5. Widerström-Noga E, Dyrehag LE, Börglum-Jensen L, Åslund PG, Wenneberg B, Andersson SA. Pain threshold responses to two different modes of sensory stimulation in patients with orofacial muscular pain: Psychologic considerations. J Orofac Pain 1998;12:27–34.
6. Fricton JR, Olsen T. Predictors of outcome for treatment of temporomandibular disorders. J Orofac Pain 1996;10:54–65.
7. De Leeuw JR, Ros WJ, Steenks MH, Lobbezoo-Scholte AM, Bosman F, Winnubst JA. Craniomandibular dysfunction: Patient characteristics related to treatment outcome. J Oral Rehabil 1994;21:667–678.
8. Flor H, Turk DC. Psychophysiology of chronic pain: Do chronic pain patients exhibit symptom-specific psychophysiological responses? Psychol Bull 1989;105: 215–259.
9. Lund JP, Donga R, Widmer CG, Stohler CS. The pain adaptation model: A discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 1991;69:683–694.
10. Malow RM. The effects of induced anxiety on pain perception: A signal detection analysis. Pain 1981; 11:397–405.
11. Rhudy JL, Meagher MW. Fear and anxiety: Divergent effects on human pain thresholds. Pain 2000;84:65–75.
12. Cornwall A, Donderi DC. The effect of experimentally induced anxiety on the experience of pressure pain. Pain 1988;35:105–113.
13. Isselée H, De Laat A, Lesaffre E, Lysens R. Short-term reproducibility of pressure-pain thresholds in masseter and temporalis muscles of symptom-free subjects. Eur J Oral Sci 1997;105:583–587.
14. Ohrbach R, Gale EN. Pressure-pain threshold in normal muscles: Reliability, measurement effects, and topographic differences. Pain 1989;37:257–263.
15. Ohrbach R, Gale EN. Pressure-pain thresholds, clinical assessment, and differential diagnosis: Reliability and validity in patients with myogenic pain. Pain 1989; 39:157-169.
16. Malarkey WB, Hall JC, Pearl DK, Kiecolt-Glaser JK, Glaser R. The influence of academic stress and season on 24-hour concentrations of growth hormone and prolactin. J Clin Endocrinol Metab 1991;73:1089–1092.
17. Malarkey WB, Pearl DK, Demers LM, Kiecolt-Glaser JK, Glaser R. The influence of academic stress and season on 24-hour mean concentrations of ACTH, cortisol, and β-endorphin. Psychoneuroendocrinology 1995;70:499–508.
18. Basbaum AI, Fields HL. Endogenous pain control systems: Brainstem spinal pathways and endorphin circuitry. Ann Rev Neurosci 1984;7:309–338.
19. Michelotti A, Farella M, Tedesco A, Cimino R, Martina R. Psychological stress and pressure-pain threshold of the masticatory muscles [abstract]. J Dent Res 1998;77:773.
20. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Examination and Specifications, Critique. J Orofac Pain 1992;6:301–355.
21. Farella M, Michelotti A, Steenks MH, Romeo R, Cimino R, Bosman F. The diagnostic value of pressure algometry in myofascial pain of the jaw muscles. J Oral Rehabil 2000;27:9–14.
22. Michelotti A, Farella M, Martina R. Sensory and motor changes of the human jaw muscles during induced orthodontic pain. Eur J Orthod 1999;21:397–404.
23. Spielberger CD, Gorsuch R, Lushere R. STAI Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press, 1970.
24. Ohrbach R, Blascovich J, Gale EN, McCall WD Jr, Dworkin SF. Psychophysiological assessment of stress in chronic pain: Comparison of stressful stimuli and of response systems. J Dent Res 1998;77:1840–1850.
25. LeResche L, Burgess J, Dworkin SF. Reliability of visual analog and verbal descriptor scales for objective measurement of temporomandibular disorder pain. J Dent Res 1988;67:33–36.
26. Carlson CR, Collins FL, Porzelius J, Stewart JF, Nitz J, Lind C. The assessment of emotional reactivity. J Psychopathol Behav Assess 1989;11:313–325.
27. Lindsey SK. Models for Repeated Measurements, ed 2. London: Oxford University Press, 1999.
28. Curran SL, Carlson CR, Okeson JP. Emotional and physiologic responses to laboratory challenges: Patients with temporomandibular disorders versus matched control subjects. J Orofac Pain 1996;10:141–150.
29. Flor H, Birbaumer N, Schulte W, Roos R. Stress-related electromyographic responses in patients with chronic temporomandibular pain. Pain 1991;46:145–152.
30. 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 TMD. Arch Oral Biol 1989;34:393–398.
31. Lariviere WR, Melzack R. The role of corticotropin-releasing factor in pain and analgesia. Pain 2000;84:1–12.
32. Dubner R, Hargreaves KM. The neurobiology of pain and its modulation. Clin J Pain 1989;5:1–6.
33. Levine JD, Feldmesser M, Tecott L, Lane S, Gordon NC. The role of stimulus intensity in opioid-mediated analgesia. Brain Res 1984;304:265–269.
34. Yemm R. Variation in the electrical activity of the human masseter muscle occurring in association with emotional stress. Arch Oral Biol 1969;14:873–878.
35. 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.
36. Gallo LM, Palla S. Activity recognition in long-term electromyograms. J Oral Rehabil 1995;22:455–462.
37. Maixner W, Fillingim R, Booker D, Sigurdsson A. Sensitivity of patients with painful temporomandibular disorders to experimentally evoked pain. Pain 1995;63:341–351.
38. Reid KI, Gracely RH, Dubner RA. The influence of time, facial side, and location on pain-pressure thresholds in chronic myogenous temporomandibular disorders. J Orofac Pain 1994;8:258–265.