Effect of Parafunctional Clenching on TMD Pain

Alan G. Glaros, PhD
Professor
Department of Dental Public Health and Behavioral Science

Kelly N. Tabacchi
Dental Student

Ernest G. Glass, DDS, MS, MSD
Associate Professor
Department of Dental Public Health and Behavioral Science

School of Dentistry
University of Missouri-Kansas City
Kansas City, Missouri

Correspondence to:
Dr Alan G. Glaros
650 East 25th Street
Kansas City, Missouri 64108
E-mail: glarosa@smtpgate.umkc.edu

Parafunctional activities are assumed to play an important role in temporomandibular disorders (TMD), but experimental data in support of this hypothesis are lacking. This study examined the role of parafunctional clenching on various measures of TMD pain. Five subjects participated in daily 17-minute electromyogram biofeedback training sessions structured in three phases. Subjects were instructed to maintain temporalis and masseter muscle activity below 2 μV in the first (decrease) phase of training (10 sessions), above 10 μV in the second (increase) phase (1 to 8 sessions), and below 2 μV in the third (decrease) phase (10 to 15 sessions). Preliminary screening examinations showed that none of the subjects had TMD. Two subjects reported intolerable pain during increase training, and both were diagnosed with a TMD during this phase. No subject was diagnosed with TMD pain during either decrease training phase. The authors conclude that chronic, low-level parafunctional clenching may be a factor in the cause of TMD pain.
J OROFACIAL PAIN 1998;12:145–152.

key words: parafunctions, pain, temporomandibular disorders, clenching, biofeedback

Parafunctional behaviors, especially clenching and grinding, are presumed to be important initiating and perpetuating factors in temporomandibular disorders (TMD).[1] However, direct experimental evidence in support of the hypothesis that parafunctional clenching produces TMD-like pain is limited. Studies of maximum bite force, for example, show that this activity can produce both fatigue and pain.[2] The pain produced by such maximum bite forces can be significant,[2–4] but short-lived.[4–6] However, reports by Christensen[7,8] have shown that the pain produced by 30-minute sessions of intensive experimental bruxism can last about 2 days.

Maximum bite force can only be maintained for a brief period of time.[9] In one study,[10] individuals without TMD engaged in maximum voluntary clenching until they reported muscle fatigue. After a 10-minute intermission, subjects again engaged in maximum clenching. Subjects were able to maintain maximum bite forces for an average of 128 seconds before muscle fatigue set in and bite force diminished. Experimental clenching at somewhat less than maximum levels produces the sensation of fatigue in 5 minutes or less.[11,12]

Studies of maximum bite force and pain may be poor models for understanding the role of parafunctions in the pain reported by some TMD patients. There are no data to indicate that TMD patients typically create maximum bite forces during parafunctional activities or that they do so for extended periods of time.[13] Furthermore, studies show that TMD patients typically have lower

maximum bite forces than individuals without TMD and that maximum bite forces increase as the problem is successfully treated.[14]

However, self-reports of TMD patients suggest that their parafunctional clenching can last for hours at a time.[15] Since the values reported by patients are far in excess of the few minutes reported by maximum bite force studies for the appearance of fatigue and pain, it is likely that the parafunctional activity reported by patients occurs at significantly lower intensities than those produced by efforts at maximum biting.

Rugh and Drago[16] showed that only minor changes in jaw position could result in large increases in the activity of the masseter. Their study indicated that the masseter muscle was least active when the posterior teeth were separated by 4.5 to 12.6 mm (mean = 8.6 mm). Bringing the posterior teeth from this opening to the point of zero clearance increased masseter activity 1.3 to 3.5 times over the level observed when the muscle was least active. Any clenching beyond mere contact would increase masseter activity to even higher levels, potentially increasing the likelihood of pain.

These findings suggest that chronic, low-level parafunctional activity may produce the muscle pain of TMD. To test this hypothesis, individuals without TMD participated in a three-phase, repeated measures study in which they were asked to decrease the activity of the temporalis and masseter muscles, increase the activity of these muscles, and decrease the activity of these muscles over a 6-week period. The level of electromyographic (EMG) activity requested of subjects during increase training was similar to that produced by tooth contact or light clenching. Various measures of pain were used as the dependent measures.

Materials and Methods

Subjects

Three women and two men ranging in age from 23 to 29 years participated in the study. All subjects were students enrolled at the University of Missouri-Kansas City (UMKC) School of Dentistry. No subject reported a facial pain problem, chronic headaches, or other painful condition of the head or neck prior to their participation in the study; no subject reported the use of interocclusal appliances prior to their participation. According to a screening examination (see Procedure, below), no subject received a TMD diagnosis before the study began. Informed consent was obtained from all subjects by means of a written form approved by the university's Institutional Review Board.

Apparatus

Miniature silver/silver-chloride surface electrodes were used to collect EMG data. The electrodes were connected to electromyographic modules (M-501, J & J Instruments, Poulsbo, WA). The module filters were set for 20 to 1000 Hz bandpass with notch filtering at 58 to 62 Hz. The bandpass setting was consistent with the authoritative recommendations of Fridlund and Cacioppo[17] for EMG research. The full-wave rectified outputs (20 millisecond time constant) from the modules were fed into an I-330 interface (J & J Instruments) that converted the analogue signals to digital form. The interface can sample 15 different physiologic signals, up to 1000 samples per second, at a resolution of 14 bits. The operation of the I-330 interface was controlled by its USE software.

Electrodes were placed on the left and right temporalis and masseter areas using templates constructed according to the recommendations of Kawazoe et al[18] for the temporalis and the recommendations of Fridlund and Cacioppo[17] for the masseter. All surface sites were cleansed with alcohol and abrasive gel on gauze pads. Resistances between electrode pairs were checked with an EZM 5A electrode impedance meter (Grass Instruments, Quincy, MA).

Threshold Selection

EMG data from prior studies[15,19] that used similar or identical settings, equipment, and procedures were used to set the threshold for decrease training at 2.0 μV. In these studies, EMG values for the temporalis and masseter muscles obtained from non-TMD subjects at rest ranged from 2.32 μV to 3.94 μV (SD = 1.55 to 2.92). Several studies have shown that TMD patients have higher levels of temporalis and masseter activity at rest than non-TMD individuals.[20,21] When these patients participate in a treatment regimen of relaxation and EMG biofeedback in the authors' clinic, they are frequently able to reduce temporalis and masseter activity to 2.0 μV or less. It was therefore assumed that the non-TMD subjects used in this study would be able to attain temporalis and masseter muscle activity at or below 2.0 μV in this biofeedback task, at least some of the time, by the use of self-initiated relaxation strategies accompanied by separation of the posterior teeth.

The ratios describing the difference between the resting values just noted and the 10.0-μV threshold

value requested for increase training ranged from 2.5 to 4.3 μV. These values are somewhat higher than the ratios of 1.3 to 3.5 μV reported by Rugh and Drago[16] for masseter activity between maximally relaxed and tooth-contact values. In order to create the 10.0-μV threshold value requested for increase training, subjects needed to create posterior tooth contact or engage in light clenching.

Procedure

This study was structured in a three-phase, repeated measures design. The three phases were: *(1)* biofeedback training during which subjects were instructed to decrease temporalis and masseter EMG activity; *(2)* biofeedback training during which subjects were instructed to increase temporalis and masseter EMG activity; and *(3)* biofeedback training during which subjects were instructed to decrease temporalis and masseter EMG activity. Subjects had a maximum of 28 training sessions over a 6-week period. The first, decrease phase consisted of 5 training sessions per week for 2 weeks, for a total of 10 sessions. Ten sessions were selected for this phase because this value falls within the range of sessions reported in studies examining biofeedback training for TMD.[22–24] The second, increase phase was scheduled to last 8 sessions (5 training sessions in the first week, and 3 training sessions followed by two holidays in the second week). Subjects who reported excessive or intolerable pain during increase training were given the option to return immediately to decrease training. A subject who terminated increase training was asked to undergo a screening examination (see Procedures, below) within 24 hours of their decision. The third, decrease phase was scheduled to last at least 10 sessions (5 sessions per week for 2 weeks). Subjects who terminated increase training before the 8 scheduled sessions had been completed underwent additional days of decrease training.

At each session, subjects were placed in a light- and sound-attenuated chamber and seated in a comfortable recliner with the foot rest extended. After the electrodes were applied, the training session was started. Subjects viewed their EMG activity on a computer monitor. EMG activity from the temporalis and masseter muscles was represented by colored lines that traversed the screen once per minute. The monitor also showed a threshold line. Subjects were asked to try to keep their temporalis and masseter muscle activity below the threshold line during decrease training and to maintain their temporalis and masseter muscle activity above the threshold line for increase training. During decrease training the threshold line was set at 2.0 μV; for increase training the threshold line was set at 10.0 μV.

When subjects successfully kept all muscle activity below the threshold during decrease training or above the threshold during increase training, they were given additional feedback in the form of a tone that "chimed" periodically. The pitch of the chime tone varied directly as a function of the EMG activity. EMG biofeedback training sessions for TMD patients treated in the authors' clinic typically last between 15 and 20 minutes; therefore, the duration of the training sessions in this study was set at 17 minutes. Feedback was provided throughout the training session.

Prior to each training session, subjects completed four visual analogue scales (VASs), which measured worst pain since the last training session, least pain since the last training session, amount of clenching since the last training session, amount of stress since the last training session, and, for those who indicated the presence of a headache, the intensity of the headache since the last session. Each VAS was 100 mm long. For the three pain-related questions, the end-points of the scales were "No pain" and "Worst pain possible"; for the clenching question, the end-points of the scales were "No clenching" and "Constant clenching." At the end of each training session, all subjects completed two additional VASs measuring worst and least pain levels during the training session.

Prior to participation, all subjects underwent a screening examination administered by the UMKC Facial Pain Center. During the screening examination, six muscle sites accessible extraorally (anterior temporalis, middle temporalis, posterior temporalis, masseter, sternocleidomastoid, and posterior digastric) and four muscles accessible intraorally (masseter, temporalis tendon, lateral pterygoid, and medial pterygoid) were palpated according to the techniques described by Dworkin and LeResche.[25] Subject report of pain during muscle palpation was scored on a 0 to 3 scale, with 0 signifying no pain. The presence of reproducible clicking on vertical opening, closing, lateral excursion, and protrusion was determined by auscultation; as was the presence of coarse crepitus. The presence of pain in the temporomandibular joint (TMJ) was determined by palpation and rated on a 0 to 3 scale. Pain-free unassisted mandibular opening and maximum unassisted opening were measured in millimeters. All subjects also completed the McGill Pain Questionnaire during the screening examination. The screening examinations were repeated at the end of each week of training and within 24 hours of a decision to terminate increase training early. The examiner was blind to the subjects' training condition.

Table 1 Number of Sessions Completed by Each Subject in Each Phase of Training

Subject no.	Decrease 1	Increase	Decrease 2
1	10	1	15
2	10	4	13
3	10	8	10
4	10	8	10
5	10	8	10

Table 2 Diagnostic Outcomes From Screening Examinations

Subject no./ gender	Initial exam*	Decrease 1*	Increase*	Decrease 2*
1/F	No facial pain (1)	No facial pain (2)	Myofascial pain arthralgia (1)	No facial pain (3)
2/F	No facial pain (1)	No facial pain (2)	Arthralgia (1)	No facial pain (3)
3/F	No facial pain (1)	No facial pain (2)	No facial pain (2)	No facial pain (2)
4/M	No facial pain (1)	No facial pain (2)	No facial pain (2)	Disc displacement with reduction; no facial pain† (2)
5/M	No facial pain (1)	No facial pain (2)	No facial pain (2)	No facial pain (2)

*Total number of exams for each phase indicated in parentheses.
†Diagnosis of disc displacement made in the first screening examination conducted in this phase; no TMD diagnosis made in the second screening examination conducted in this phase.

Data were analyzed by means of analysis of variance (ANOVA) with repeated measures (electromyographic and visual analogue scale data) or Friedman ANOVA (McGill Pain Questionnaire data); $P < 0.05$ was considered significant.

Results

Table 1 reports the number of sessions in each of the training phases for each subject. Two of the five subjects terminated increase training early as a result of high levels of pain. Consequently, the number of exams in the increase and in the second decrease training phases varied across subjects because of the early termination of increase training by these two subjects. The diagnostic results from the screening examinations are summarized in Table 2. Two of the five subjects received a diagnosis of arthralgia or arthralgia and myofascial pain during increase training.

The mean EMG values for each training session were used as the measure of muscle-related activity in the decrease and increase sessions. These data are summarized in Table 3; due to intermittent equipment failure, data from the right masseter are not presented. All three muscles showed significant changes in activity across the three phases of the study. Worst and least pain levels as reported by subjects immediately following each training session are presented in Table 4. Worst pain levels varied significantly as a function of training phase. The overall correlation between worst pain level and the average (for each participant) of the three EMG values was 0.729 ($P < 0.01$). Table 5 presents worst and least pain levels for the 24-hour periods preceding each training session; these variables did not differ significantly over the three training phases. Mean stress levels (and standard deviations) for the 24-hour periods preceding each training session were 2.90 (3.11), 11.52 (14.90), and 1.89 (1.42), respectively; clenching activity (and SDs) for the same time periods were 0.88 (0.77), 3.23 (5.75), and 1.14 (0.87), respectively. Neither measure changed significantly across training phases.

Table 3 EMG Activity (μVSecond Root-Mean-Square) for Three Training Phases

Site/ subject no.	Decrease 1		Increase		Decrease 2		F(2, 8)
	Mean	SD	Mean	SD	Mean	SD	
Left temporalis							
1	2.19	0.61	14.86	0.00†	6.63	5.93	
2	3.48	1.20	11.94	2.10	8.58	7.19	
3	2.04	1.48	16.61	7.37	2.77	1.40	
4	2.46	1.17	23.58	4.88	3.96	2.33	
5	2.56	1.18	23.70	8.13	8.94	6.92	
Mean	2.55	0.56	18.14	5.29	7.01	4.09	23.43**
Left masseter							
1	2.63	1.35	28.42	0.00†	6.41	6.88	
2	8.22	5.83	11.26	2.54	5.64	3.03	
3	2.60	1.11	20.67	5.69	4.92	3.79	
4	1.88	0.69	22.90	6.54	3.17	1.42	
5	2.06	1.61	17.61	4.24	3.53	1.44	
Mean	3.48	2.67	20.17	6.36	4.73	1.37	21.91**
Right temporalis							
1	2.58	1.31	9.45	0.00†	4.11	1.60	
2	4.35	2.14	9.55	3.08	3.93	2.06	
3	1.83	0.71	13.34	3.66	2.26	1.12	
4	2.87	1.60	29.66	5.43	3.93	2.77	
5	2.34	1.36	19.73	4.95	4.80	3.44	
Mean	2.79	0.95	15.47	9.13	3.81	0.94	9.06*

*$P < 0.01$.
**$P < 0.001$.
†This subject had only one session of increase training (see Table 1).

Table 4 Self-Reported Pain Rating at End of Training Sessions*

Pain rating/ subject no.	Decrease 1		Increase		Decrease 2		F(2, 8)
	Mean	SD	Mean	SD	Mean	SD	
Worst pain							
1	0.89	0.78	60.00	0.00†	0.07	0.26	
2	0.70	0.95	20.75	7.50	0.46	0.52	
3	0.70	1.34	17.25	13.48	0.00	0.00	
4	0.20	0.42	15.63	3.89	0.20	0.42	
5	1.50	4.40	32.75	18.90	0.10	0.32	
Mean	0.80	0.47	29.28	18.44	0.17	0.18	12.29**
Least pain							
1	0.89	0.93	61.00	0.00†	0.07	0.26	
2	1.20	2.82	4.50	3.42	0.33	0.89	
3	0.20	1.34	4.13	5.79	0.00	0.00	
4	0.10	0.32	2.50	0.93	0.00	0.00	
5	1.40	4.43	5.25	5.23	0.20	0.42	
Mean	0.76	0.59	15.48	25.47	0.12	0.14	1.75

*Pain rating obtained from 100-mm visual analogue scale.
**$P < 0.01$.
†This subject had only one session of increase training (see Table 1).

Table 5 Self-Reported Pain Rating for 24-Hour Period Preceding Training Sessions*

Pain rating/ subject no.	Decrease 1 Mean	Decrease 1 SD	Increase Mean	Increase SD	Decrease 2 Mean	Decrease 2 SD	F(2, 8)
Worst pain							
1	0.63	0.92	19.00	0.00†	0.00	0.00	
2	0.25	0.46	0.00	0.00	0.11	0.33	
3	0.63	1.77	0.00	0.00	0.13	0.35	
4	0.75	1.75	2.33	1.86	0.50	0.76	
5	0.00	0.00	5.67	8.45	0.00	0.00	
Mean	0.45	0.32	5.40	7.95	0.15	0.21	2.05
Least pain							
1	0.50	0.76	22.00	0.00†	0.36	0.67	
2	0.00	0.00	0.00	0.00	0.11	0.33	
3	0.88	2.10	0.00	0.00	0.00	0.00	
4	0.00	0.00	0.00	0.00	0.00	0.00	
5	0.25	0.71	0.00	0.00	0.13	0.35	
Mean	0.33	0.37	4.40	9.84	0.12	0.15	0.92

*Pain rating obtained from 100-mm visual analogue scale.
†This subject had only one session of increase training (see Table 1).

Table 6 Results of Screening Examinations

	Decrease 1 Mean	Decrease 1 SD	Increase Mean	Increase SD	Decrease 2 Mean	Decrease 2 SD
No. of muscles with pain to palpation (external)	0.00	0.00	0.60	0.89	0.00	0.00
No. of muscles with pain to palpation (internal)	0.00	0.00	1.00	2.24	0.00	0.00
No. of activities producing TMJ clicking	0.00	0.00	0.00	0.00	0.40	0.89
Pain-free maximum opening (mm)	47.47	3.25	49.70	7.78	48.53	5.71
Maximum opening (mm)	56.60	7.63	56.80	7.74	56.03	7.91
McGill Pain Questionnaire (no. of words circled)	3.00	2.45	3.00	1.58	0.00	0.00

The results from the screening examinations are presented in Table 6. Because of the lack of variability in the decrease phases for muscle palpation pain and joint clicking, no inferential statistics could be computed on these variables. Of the remaining three dependent variables listed in Table 6, only the McGill Pain Questionnaire (number of words circled) changed significantly across the three phases of the study (Friedman F_r [3,5] = 8.40; $P < 0.01$).

Discussion

The data indicate that increase training resulted in two of the subjects receiving a TMD diagnosis involving pain. Both of these subjects reported joint pain characteristic of arthralgia, while one reported muscle pain characteristic of myofascial pain.[25] As expected, reports of pain increased as subjects performed increase training. However, the pain reported by subjects tended to resolve in the 24 hours following increase training.

The EMG data show considerable variability during increase training. An examination of the individual subject data suggests that some subjects maintained EMG levels around 10 μV during increase training, while others showed considerably more activity in these muscles. Nonetheless, the correlation between EMG activity and self-reported pain was .729, suggesting that EMG activity accounted for a significant proportion of the variance in the self-reported pain scores. The decision to terminate increase training early, however, may not have been

a function of EMG levels alone; of the two subjects who terminated increase training prematurely, one had the lowest mean EMG activity (for the three sites combined) during increase training, while the other had the median level of activity.

The reports of pain immediately postsession indicate that worst pain levels increased significantly during increase training, while pain levels assessed for the 24 hours preceding each training session did not change significantly during any phase. As with the EMG data, there was significant variability in the postsession and 24-hour pain ratings. As indicated by Table 5, only one of the two subjects who terminated increase training early showed persistent pain following increase training.

Two of the five subjects, both women, terminated increase training early because of self-reported intolerable pain. These findings suggest that the experimental protocol succeeded in increasing TMD pain in a subset of the subjects who participated in this study. A variety of factors might account for the presence of pain in this subset of subjects, including differing levels of pain tolerance, differing levels of muscle activity during increase trials, and differing biochemical/physiologic responses in the musculature to sustained, low-level activity.[26] All three of the women participating in this study used oral contraceptives, and the use of these medications may have increased their susceptibility to experimental clenching.[27] Considerably more research would be needed to identify the characteristics of individuals who responded with pain to the protocol used here. The possibility that experimenter bias and subject expectation effects were in part responsible for the results on pain should also be considered.

The findings from this study suggest that low-level parafunctional activity may be a mechanism producing pain in some TMD patients. According to this model, some individuals engage in low-level parafunctional activity for lengthy periods of time. The activity might consist of tooth contact, more intense clenching, or other kinds of parafunction. In any case, the activity of the masseter[16] and other elevator muscles is likely to be significantly greater than the activity recorded when the muscles are at rest. The data from the present study suggest that this low-level activity can result in arthralgia or myofascial pain. The 17 minutes of daily training performed by these subjects (for a maximum of 8 days) may be only a minor approximation of the amount of time that some TMD patients engage in parafunctional activities. Unfortunately, nonreactive, in vivo measures of parafunctional clenching in TMD patients are not available. Individuals diagnosed with myofascial pain appear to have deficits in proprioceptive awareness,[15,28] and this may account for their failure to recognize that they engage in parafunctional activity.

Further research is needed to investigate the hypothesis that chronic, low-level parafunctional activity produces TMD pain. It would be desirable to conduct the study with a larger sample size. To reduce subject expectation effects, subjects could be randomly assigned to only increase or decrease training. Alterations of the protocol (eg, more training sessions per day or raising the threshold for increase training) may be more successful in producing pain. To ensure that subjects were engaged in the same level of effort during increase training, the threshold for increase training could be set as a fixed percentage of a resting, most relaxed EMG value or of EMG activity produced during maximum clenching. Additionally, it would be useful to identify the factors that distinguish individuals who respond with pain to the experimental protocol from those who do not.

Acknowledgments

We thank the UMKC Summer Dental Scholar program for their support, and Dr Nagamani Narayana for her assistance.

References

1. Glaros AG, Glass EG. Temporomandibular disorders. In: Gatchel RJ, Blanchard EB (eds). Psycho-physiological Disorders: Research and Clinical Applications. Washington, DC: American Psychological Association, 1993:299–356.
2. Christensen LV. Progressive jaw muscle fatigue of experimental tooth clenching in man. J Oral Rehabil 1981; 8:413–420.
3. Waltimo A, Könönen M. A novel bite force recorder and maximal isometric bite force values for healthy young adults. Scand J Dent Res 1993;101:171–175.
4. Christensen LV. Integrated electromyography of experimental tooth clenching in man. J Dent Assoc S Afr 1978; 33:477–480.
5. Christensen LV. Some subjective-experiential parameters in experimental tooth clenching in man. J Oral Rehabil 1979;6:119–136.
6. Clark GT, Adler RC, Lee JJ. Jaw pain and tenderness levels during and after repeated sustained maximum voluntary protrusion. Pain 1991;45:17–22.
7. Christensen LV. Eksperimentelt provokerede ansigtssmerter fra kaebernes bevaegesystem ved bruksismei en forelobig meddelelse [Danish]. Tandlaegebladet 1967;71:1171–1181.
8. Christensen LV. Facial and internal pressure of masseter muscle in experimental bruxism in man. Arch Oral Biol 1971;16:1021–1031.
9. Lyons MF, Rouse ME, Baxendale RH. Fatigue and EMG changes in the masseter and temporalis muscles during sustained contractions. J Oral Rehabil 1993;20:321–331.

10. Christensen LV. Quantitative observations on maximum static work efforts and associated pain of the human masseter muscle. J Oral Rehabil 1988;15:561–573.
11. Christensen LV. Jaw muscle fatigue and pains induced by experimental tooth clenching: A review. J Oral Rehabil 1981;8:27–36.
12. Christensen LV. Progressive jaw muscle fatigue of experimental tooth clenching in man. J Oral Rehabil 1981; 8:413–420.
13. Gay T, Maton B, Rendell J, Majourau A. Characteristics of muscle fatigue in patients with myofascial pain dysfunction syndrome. Arch Oral Biol 1994;39:847–852.
14. Helkimo E, Carlsson GE, Carmeli Y. Bite force in patients with functional disturbances of the masticatory system. J Oral Rehabil 1975;2:397–406.
15. Glaros AG. Awareness of physiological responding under stress and non-stress conditions in temporomandibular disorders. Biofeedback Self-Regul 1996;12:261–272.
16. Rugh JD, Drago CJ. Vertical dimension: A study of clinical rest position and jaw muscle activity. J Prosthet Dent 1981;45:670–675.
17. Fridlund AJ, Cacioppo JT. Guidelines for human electromyographic research. Psychophysiology 1986;23: 567–589.
18. Kawazoe Y, Kotani H, Hamada T. Relation between integrated electromyographic activity and biting force during voluntary isometric contraction in human masticatory muscles. J Dent Res 1979;58:1440–1449.
19. Fogle LL, Glaros AG. Contributions of facial morphology, age, and gender to EMG activity under biting and resting conditions: A canonical correlation analysis. J Dent Res 1995;78:1496–1500.
20. Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain dysfunction syndrome. J Behav Med 1989;12:397–406.
21. Rao SM, Glaros AG. Electromyographic correlates of experimentally induced stress in diurnal bruxists and normals. J Dent Res 1979;58:1872–1878.
22. Dalen K, Ellertsen B, Espelid I, Grönningsæter AG. EMG feedback in the treatment of myofascial pain dysfunction syndrome. Acta Odontol Scand 1986;44:279–284.
23. Funch DP, Gale EN. Biofeedback and relaxation therapy for chronic temporomandibular joint pain: Predicting successful outcomes. J Consult Clin Psychol 1984;52: 928–935.
24. Olson RE, Malow RM. Effects of biofeedback and psychotherapy on patients with myofascial pain dysfunction who are nonresponsive to conventional treatments. Rehabil Psychol 1987;32:195–204.
25. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, Criteria, Exams and Specification, Critique. J Craniomandib Disord Facial Oral Pain 1992;6:301–355.
26. Christensen LV, Tran KT, Mohamed SE. Gum chewing and jaw muscle fatigue and pains. J Oral Rehabil 1996; 23:424–437.
27. LeResche L, Saunders K, Von Korff MR, Barlow W, Dworkin SF. Use of exogenous hormones and risk of temporomandibular disorder pain. Pain 1997;69:153–160.
28. Glaros A. Evidence for proprioceptive deficits in temporomandibular disorders. J Dent Res 1997;76:116.

Resumen

Efecto del apretamiento parafuncional en el dolor de los desórdenes temporomandibulares

Se supone que las actividades parafuncionales juegan un papel importante en los desórdenes temporomandibulares (DTM), pero no existe información experimental que sustente esta hipótesis. Este estudio examinó el papel del apretamiento parafuncional sobre varias medidas de dolor por DTM. En este estudio participaron 5 personas, quienes fueron sometidas a sesiones de 17 minutos consistentes en entrenamiento de bioretroinformación electromiográficas. Estas sesiones fueron estructuradas en tres fases. La instrucción de los participantes incluyó las siguientes fases de entrenamiento: Durante la primera fase de 10 sesiones se mantuvo la actividad de los músculos temporal y masetero debajo de 2μV (disminución), durante la segunda fase (1 a 8 sesiones), se mantuvo la actividad por encima de 10 μV (aumento), y en la tercera fase de 10 a 15 sesiones, la actividad se mantuvo por debajo de 2 μV (disminución). Los exámenes de selección preliminares indicaron que ninguno de los participantes sufría de DTM. Dos personas se quejaron de dolor intolerable durante el entrenamiento cuando se aumentó la actividad, y ambas fueron diagnosticadas con DTM durante esta fase. Ninguna de las personas fue diagnosticada de dolor por DTM durante las fases cuando la actividad fue disminuida. Los autores concluyen que el apretamiento parafuncional de bajo nivel, crónico, puede ser un factor relacionado a la etiología del dolor de los DTM.

Zusammenfassung

Auswirkung des parafunktionellen Pressens auf den TMD Schmerz

Es wird angenommen, dass parafunktionelle Aktivitäten eine wichtige Rolle bei den temporomandibulären Erkrankungen (TMD) spielen, aber es fehlen experimentelle Daten zur Stützung dieser Hypothese. Diese Studie untersucht die Rolle des parafunktionellen Pressens auf verschiedene Ausmasse des TMD Schmerzes. Fünf Personen nahmen an täglichen 17 minütigen elektromyographischen Trainingssitzungen, strukturiert in drei Phasen, teil. Die Personen wurden instruiert, die Temporalis- und Masseteraktivität in der ersten (Abnahme) Phase des Trainings (10 Sitzungen) unter 2 μV, in der zweiten (Zunahme) Phase über 10 μV (1 bis 8 Sitzungen), und in der dritten (Abnahme) Phase unter 2 μV (10 bis 15 Sitzungen), zu halten. Einleitende Screeninguntersuchungen zeigten, dass keine der Personen eine TMD aufwies. Zwei Personen berichteten über unerträgliche Schmerzen während des Zunahmetrainings, und bei beiden wurde während dieser Phase eine TMD diagnostiziert. Bei keiner Person wurde während beider Abnahmetrainingsphasen ein TMD Schmerz diagnostiziert. Die Autoren schliessen daraus, dass chronisches, schwaches parafunktionelles Pressen ein Faktor für die Ursache von TMD Schmerz sein kann.