Waking and Sleeping Temporalis EMG Levels in Tension-Type Headache Patients

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Temporalis muscle activity in tension-type headache subjects (n = 36) and in matched nonheadache controls (n = 36) was evaluated in this study. Subjects' cumulative temporalis muscle activity was recorded every 30 minutes for 3 days and nights using an electromyographic recorder. Analysis of variance showed that neither the waking nor the sleeping overall muscle activity levels for these two groups were statistically different. When the waking EMG data were dichotomized into function and nonfunction activities, a significant difference was found between groups during jaw function (ie, chewing and talking). These data suggest that headache subjects are using their temporalis muscles with less efficiency than nonheadache subjects during function. This elevated EMG is more likely a consequence of pain (via protective splinting or guarding) rather than a cause in tension-type headache sufferers.
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A tension-type headache is the diagnosis when a patient complains of longstanding, continuous, dull, aching pain of variable intensity in the temporal, frontal, or suboccipital region. The consensus opinion in 1962 regarding the etiology for nonmigrainous continuous daily headache problems was that myogenic or muscle contraction headaches were caused by sustained muscle tension.[1] In recent years, the traditional hypothesis that elevated muscle tension induces muscle pain (also known as myogenic headache) has been seriously challenged.

Previous research on this topic has the limitation of being largely based on resting electromyographic (EMG) evaluations made in the laboratory, while other forms of muscle activity that might cause pain have not been extensively studied.[2–11] For example, waking-state muscle activities, which might induce or be associated with tension-type headache, include any habitual behavior exhibited by a patient (eg, clenching of the teeth, poor posture of the head or neck). Unfortunately, such habits are not easily identified during a laboratory experiment because patients being observed typically do not demonstrate such behaviors. In contrast to the laboratory studies, several reports in the literature use ambulatory EMG recording techniques.[12,13] Studies using the early portable, natural-environment EMG recording methods are described below.

In one article using the ambulatory EMG technique, Rugh and colleagues reported the mean daytime level of neck muscle activity in headache sufferers versus nonheadache sufferers during 3 stressful and 3 nonstressful days.[14] They found a significant influence of stress on the EMG level in both groups but no significant difference in the mean daily EMG levels between the headache and nonheadache subjects. One limitation of this study was that physical activity levels were not reported. Since physical activity can strongly influence EMG levels, the actual activities engaged in by subjects during the stress period versus the nonstress period may have been quite different. In 1991, a second study appeared on the relationship among ambulatory EMG activity, stress, and pain in tension-type headache subjects versus nonheadache subjects.[15] In this experiment, EMG levels from the frontalis muscle and the posterior neck region (sixth cervical vertebrae) were monitored during both the waking and sleeping periods. In addition, daily pain and stress levels were measured every 30 minutes. The results of the EMG level analysis showed no significant differences in overall waking or sleeping EMG levels between the headache and nonheadache subjects. Analysis also revealed that the subjects' posture had a significant influence on the relationship between neck muscle activity and pain, but neither neck nor frontalis EMG was substantially correlated to pain or stress. The conclusion of this study was that EMG activity did not covary with stress or pain, but pain and EMG were influenced by posture. Unfortunately, this study did not provide any clear information about how long the subjects were engaged in each posture or about which posture and to what extent these body postures (sitting, standing, and reclining) influenced the subjects' pain or EMG levels.

Another form of waking-state muscle activity not likely to be observed in laboratory studies investigating resting muscle tension levels in headache subjects is protective muscle activity (PMA).[16] Protective muscle activity is best defined as an involuntary contraction generated in response to a regionally painful condition in an effort to prevent or avoid painful movement. Protective muscle activity (eg, jaw muscle trismus) would only manifest itself if a painful, deliberate movement of the involved body part were requested as a part of the experiment. Several investigators have documented these abnormal skeletal muscle activity patterns during movement in the masticatory system.[17–20] Only occasionally will regional pain produce low levels of PMA at rest. In these cases, elevated muscle activity in painful muscles is thought to be the consequence and not the cause of the pain.[21] For example, several studies have demonstrated that orofacial muscle activity levels were more elevated in migraine sufferers during an attack than in so-called "muscle contraction" headache patients with pain.[2,22,23]

A further consideration is that sleeping-state muscle activity (eg, bruxism) may contribute to headache disorders. For example, masticatory muscle pain problems are reported to result from bruxism, strong and often rhythmic contractions of the jaw muscles during sleep.[24] These contractions can be rhythmic or continuous isometric contractions lasting from several seconds to as much as 5 minutes.[25] Bruxism contractions are probably not learned or habitual behaviors, but uncontrolled, patterned outflows of central nervous system activity during sleep.[26] Several studies have monitored nocturnal masseter muscle activity levels and successfully related increases in EMG levels to daytime pain and dysfunction in the muscles of mastication.[27,28] Despite evidence of the relationship of bruxism behavior and masticatory muscle pain, few references in the headache literature discuss teeth grinding or clenching during sleep (bruxism) as a source of headache problems. This may be because many patients are unaware of their nighttime teeth-grinding activity. The above questions can only be answered by analysis of ambulatory and sleeping-state EMG recordings of headache patients in their natural environment.

The purpose of this study was to evaluate the null hypothesis that there is no difference in waking or sleeping temporalis muscle activity levels between tension-type headache and matched nonheadache subjects. Several brief reports on this research have been published previously.[29–32]

Materials and Methods

Study Sample

This study involved the following two groups of subjects: *(1)* headache subjects who had tension-type headaches in the temporal region, and *(2)* nonheadache subjects who were individually matched in age, gender, race, and education to headache subjects. Temporal region pain was selected to ensure that all subjects in the study had pain and EMG recordings from the same anatomical region. All subjects, recruited from newspaper advertisements, were sent a screening questionnaire and examined for eligibility. The specific

inclusion, exclusion, and matching criteria used in this study were that all nonheadache subjects had to be relatively headache-free (< 1 mild headache per month) and not exhibit any substantive signs or symptoms of a craniocervical or temporomandibular joint (TMJ) disorder. The headache subjects had to have the following: *(1)* a positive complaint of a strong, frequent (≥ 3 times per week) dull aching pain of a protracted nature in the temporal region, *(2)* a negative past treatment history and examination for substantive signs or symptoms of a TMJ or craniocervical disorder, *(3)* no more than one of the migraine-like symptoms of photophobia or nausea. No subject in this study exhibited a warning aura, a visual scintillating scotoma, or a severe throbbing sensation. Finally, no active treatment (including medication) was allowed 1 week prior to or during the recording period. All acceptable subjects were then given a full and detailed explanation of the nature of research and asked to sign the consent form.

Diary Recordings

For 6 consecutive days, subjects recorded in a pain diary their perceived pain, stress, and physical activity levels on a 100-mm visual analog scale (VAS) for each 30 minutes of waking time. These three VAS scales used the words "No Pain, No Stress, or No Physical Activity" to anchor the left side of the line. On the right side of the line, the words "Most Pain Imaginable, Highest Stress Imaginable, and Most Physical Activity Imaginable" were used. The VAS-based ratings for pain, stress, and physical activity were recorded in separate diaries for each day. Subjects also marked the predominant behavior they were engaged in for each 30 minutes. Last, the EMG level for the temporalis muscle (method described below) was also recorded in the diary every 30 minutes during the last 3 days. To encourage compliance, a phone call was made to the subjects at least one time per day, and a timer that sounded an alarm every 30 minutes was issued to each subject.

Electromyographic Recording

The EMG recording was made by the portable EMG integrator (AL 200B muscle activity integrator).[13] This EMG integrator is designed to record muscle activity levels in ambulatory subjects over extended time periods. Cumulatively stored muscle activity is viewed on a digital display panel by pressing the display switch. The resulting muscle activity units are shown as microvolts per second and are a function of the duration and amplitude of the input signal. The threshold was set at 10 μV to avoid recording any electrical activity during swallowing. This threshold level is also high enough to exclude noise from minor head and neck movement or from inadvertent touching of the electrode or cable. The stored information can be erased by turning the reset switch to "off" and can be recorded by setting it to "on." The anterior temporalis on the pain side was selected from headache subjects and was matched for nonheadache control subjects. The right side was selected when pain intensity was the same in both sides unless the skin condition was not healthy at the site of electrode placement. The bipolar silver/silver chloride surface electrode pair was placed parallel (2 cm of interelectrode distance) to the anterior portion of the temporalis muscle fibers between the hairline and the anterior border of the temporal fossa using clear adhesive collars. The ground electrode was placed over the base of the mastoid process. The skin surface was lightly abraded with alcohol gauze. Electrode paste was lightly massaged into the skin in a 3- to 5-mm area where the electrode would be placed. The impedance level was set below 10,000 ohms. Proper operation was verified by having the subject fully contract and relax the jaw muscles once every second for 5 seconds. All subjects reported to the Clinical Research Center each morning for the final 3 consecutive days of the study to allow the experimenter to confirm that the pain diary had been filled out properly. Thereafter, electrodes were replaced in the same locations as for the previous recording. Impedance was measured to ensure it was within 10,000 ohms before the subject was dismissed. The morning after the final recorded sleep period, impedance was checked again, and the subject was dismissed. Additional recording was required in the event of loose attachment of electrodes.

Data Reduction and Analysis

The primary data analyzed in this report were the temporalis EMG levels recorded every 30 minutes during the waking and sleeping periods. The weighted daily mean EMG level and one standard deviation of the mean were computed for each group to obtain least-squares estimates of the true group mean waking EMG per 30 minutes. The weights were proportional to the number of observations taken per subject, and the standard deviations are estimates of between-subject variability. Time plots of the weighted average waking EMG were made from these data.

Table 1 Weighted Waking- and Sleeping-Period Temporalis Muscle EMG Levels (μV/sec)

	Headache group EMG (n = 36)			Nonheadache group EMG (n = 36)			
Period	Mean	SD	m	Mean	SD	m	*P* value*
Waking	6642	1088	2734	5136	642	2825	.237
Sleeping	13392	6968	107	943	161	103	.133

n = number of subjects; m = number of observations.
*P value computed from matched sign test.

The sleep period mean EMG level per 30 minutes of sleep was also determined by dividing the total sleep time EMG level by the number of 30-minute periods spent sleeping. The sleep EMG distribution is skewed by a few very large values that do not appear to be errors. Comparisons between the mean 30-minute EMG levels per subject for the headache and nonheadache groups' sleeping EMGs were performed using a nonparametric matched sample sign test that weighed all subjects equally.

The headache and nonheadache subjects' mean waking EMG levels per 30 minutes are compared using a weighted analysis of variance (ANOVA).[33] In addition, to capture those time periods when subjects ate and talked (the two major jaw functions), the waking period EMG data were dichotomized in two ways: *(1)* chewing versus nonchewing, and *(2)* function versus nonfunction (function was the combination of all chewing plus talking data points). This allowed testing of the null hypothesis that jaw function period EMG levels were not different between the groups. The two group means for each of these four subsets of the waking EMG data were also compared using weighted ANOVA. The multiple comparisons for the five analyses of the waking EMG data are adjusted for by a Bonferroni correction, so that a 1% statistical significance level per analysis assures the joint statistical significance level of 5% for the complete set of waking EMG analyses.[34]

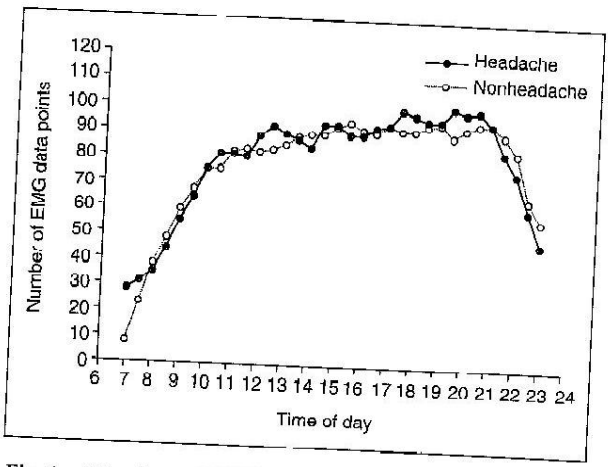

Fig 1 Number of EMG data points available at each 30-minute time point across the day for the 36 headache and for the 36 nonheadache subjects.

Results

Seventy-two subjects (36 headache, 36 nonheadache) completed this protocol. Both groups were comprised of 30 women and 6 men. The mean age for the headache group was 24 years (range 19 to 35) while the nonheadache group mean age was 23 years (range 18 to 36). The education levels for the headache group were as follows: 1 high school, 20 partial college, 8 college graduate, and 7 graduate school. The education levels for the nonheadache group were 2 high school, 25 partial college, 8 college graduate, and 1 graduate school. There were 31 Caucasians, 4 Asians, and 1 African-American in each study group.

Weighted means and standard deviations for sleeping and waking period temporalis EMG levels per 30-minute recording interval are presented in Table 1. The nonparametric test of equality of medians for the sleeping EMG scores gives a two-tailed *P* value of 13.3%. The weighted ANOVA on the overall waking EMG scores has a *P* value of 23.7%. Thus, the centrality measures for the distributions of the headache and nonheadache subjects' EMG scores do not appear to differ when the subjects are awake or asleep.

The total number of available EMG data points at each time point for each subject group is displayed in Fig 1. For any subjects who failed to monitor and record their 30-minute EMG for 3 or more consecutive data points other than for a brief daytime sleep period on a day after beginning the recording, the day was dropped from the data set. If more than 1 day was dropped, the subject was

Table 2 Weighted Waking-Period Chewing and Nonchewing Temporalis Muscle EMG Levels (μV/sec)

	Headache group EMG (n = 36)			Nonheadache group EMG (n = 36)			
Period	Mean	SD	m	Mean	SD	m	*P* value
Chewing	16408	2217	344	9863	987	418	.009
Nonchewing	5236	932	2390	4315	666	2407	.424

n = number of subjects; m = number of observations.

Table 3 Weighted Waking-Period Jaw Function and Nonfunction Temporalis Muscle EMG Levels (μV/sec)

	Headache group EMG (n = 36)			Nonheadache group EMG (n = 36)			
Period	Mean	SD	m	Mean	SD	m	*P* value
Function	12525	1938	820	6470	987	418	.004
Nonfunction	4122	578	1914	4264	730	1708	.879

n = number of subjects; m = number of observations.

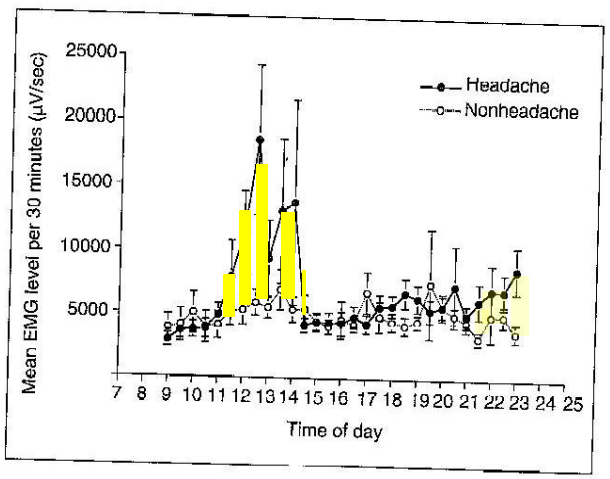

Fig 2 Mean (+ 1 SD) temporalis EMG level recorded on 36 headache and on 36 nonheadache subjects. Each subject's data were gathered within a natural environment across a 3-day period.

dropped. There were five subjects who started the experiment protocol but whose data were dropped as a result of missing data.

A decision was made to analyze only data between 8 AM and 11 PM because prior to or after these time points few subjects were recording (Fig 1). The mean for waking period temporalis muscle EMG is seen in Fig 2. These data show moderately strong elevations between 11:30 AM and 2 PM for the headache subjects and a clear but less evident elevation in the nonheadache group.

The mean value plus one standard deviation for the chewing period (eating soft foods, eating hard foods, chewing gum) versus nonchewing period (all other activities) EMG levels between the hours of 8 AM and 11 PM are presented in Table 2. The ratio (headache EMG/nonheadache EMG) for this difference was 1:7 for the chewing EMG level and 1:2 for the nonchewing EMG level. The weighted ANOVA showed significant differences, and post-hoc analysis showed that the two groups were not significantly different for the nonchewing periods but were significantly different during the chewing periods.

The mean value plus one standard deviation for the jaw-function period (eating soft food, eating hard food, chewing gum, talking) versus nonfunction period (all other activities) EMG levels (with and without baseline adjustment) between the hours of 8 AM and 11 PM are presented in Table 3. The headache EMG/nonheadache EMG ratio was 1:9 for the function EMG level and 1:0 for the nonfunction EMG level. As with the chewing versus nonchewing data, the weighted ANOVA of average function versus average nonfunction EMG levels showed significant differences, and post-hoc analysis showed that the two groups were not significantly different for the nonfunction periods but were significantly different during the function periods.

Discussion

There are major differences between this study and a majority of the research efforts reviewed in this paper. First, our experiment was performed on ambulatory subjects in a natural environment, not in a research laboratory. Our data are not limited to resting "postural muscle tone" evaluations, but include both functional and nonfunctional behaviors (eg, subjects ate and talked, among other routine daily behaviors). We used a more narrowly defined population of headache subjects than is traditionally used by headache researchers, ie, pain was limited to the temporal region. The time period of data collection used in our study was considerably longer than in earlier EMG experiments. Finally, our EMG data were slightly different from most resting EMG studies in that all EMG activity below 10 mV was filtered out. We selected this threshold criteria based on work by Burgar and Rugh[13] and because it removed unwanted swallowing and minor facial muscle expressions from the EMG recording of the temporalis muscle.

We recognize that the approach taken in this study allows for the possibility that the study itself might alter the behavior being monitored. In general, such a possibility is an inherent feature of the type of study we conducted (ie, a natural environment study of pain, stress, and EMG). Although this possibility is of concern, we believe that it does not invalidate the findings of this study. We did not measure the resting tone levels only, but functional behaviors such as talking and eating, which tend to be so routine as to be almost automatic. Subjects repeatedly reported that they completely forgot about the recorder and were amazed by how frequently the 30-minute alarm went off during a day. This indicates that the subjects were indeed following their instructions to go about their daily routines without altering their activities.

Of course, the only way to control such possibilities would be to conduct the entire experiment in a laboratory under careful observation by the experimenter. Since it was the goal of this experiment to have a large number of subjects perform within a *natural environment* while being monitored for stress, pain, and EMG levels, a laboratory-based experimental setting was not possible. We are fully aware of the potential limitations of such data; however, conducting experiments in a natural environment should be seen as the correct and logical extension of the laboratory work that has already been done on tension-type headache subjects. Actually, in spite of the possible shortcomings of this study, we are of the opinion that pain, stress, and especially EMG data collected in the natural environment is far more relevant than that collected in an obviously artificial environment such as an experimental lab, where all data is highly controlled.

In spite of these potential confounders and substantial differences in experimental design, our finding of no overall difference in EMG level between headache and nonheadache subjects is consistent with prior research reports. One extremely interesting finding illustrated by our data can be seen in the plots of the waking period temporalis muscle EMG. This plot clearly shows marked elevations in the headache group data at specific time points during the day. The largest of the elevations (11:30 AM to 2:00 PM) is undoubtedly associated with a period (lunch time) when most subjects were chewing. The breakfast and dinner elevations were not as clear, possibly because of the more variable nature of the timing of these meals. Between the periods of increased function (eg, eating), EMG levels of the headache subjects returned to the levels seen in the nonheadache subjects. The strong statistical differences found for both chewing versus nonchewing and function versus nonfunction analyses are interpreted to mean that subjects with headaches are either *(1)* consistently eating harder foods and talking with more vigor, *(2)* eating and talking more frequently, and/or *(3)* using their muscles less efficiently during eating and talking. We believe that the latter assumption is a more logical probability.

The implications of these data are that tension-type headache subjects exhibit clear elevation of their temporalis muscle during function as a probable consequence of their headache pain. This finding was upheld in a recently published cross-correlation analysis on the same data described in this study.[34] In that paper, we found virtually no correlation between pain, stress, or physical activity with EMG for either group. The cross-correlation analysis also showed that temporalis muscle activity levels were not related to the rise and fall of the subjects' pain or stress levels. Conversely, elevated stress appeared to be highly related to pain; it occurred as both an antecedent and simultaneous event with elevated headache pain. Considering the data in the current study and the results of the prior cross-correlation analysis, it seems highly unlikely that the EMG elevations are a cause of the pain.

Actually, the EMG data in our study are quite similar to the EMG data gathered by researchers who used a similar ambulatory EMG monitoring

method to evaluate regional (frontalis and posterior neck) muscle activity levels in tension-type headache subjects.[15,35] These earlier studies showed that mean daily ambulatory EMG levels for non-headache sufferers versus tension-type headache sufferers were not greatly different. Although we recorded a different muscle than was recorded in these previous studies, our data also failed to demonstrate a significant group difference in the mean overall daily waking or sleeping period EMG levels as a result of the large individual subject variability in the data. On the other hand, our data did allow us to reject the modified null hypothesis that "jaw function–associated EMG levels were not different between the groups." That is, we did see a difference between the groups when the muscle activity levels during function were separated from the nonfunctional activity. We attribute this result to the fact that high- and low-function periods for the temporalis muscle were easily identified using the behavioral categories, while the same distinction is not as easily made for posterior neck and frontalis muscles. Our data do not provide a chew-by-chew EMG analysis for the headache subjects, but the relationship between EMG elevations and eating and talking behaviors is unmistakable. We do not interpret our data to suggest a causal relationship between the elevated waking levels and pain, but rather that the EMG elevations seen in the headache subjects are protective muscle activity responses induced by the pain and are not the cause of the pain.

With regard to the sleeping EMG levels, it is clear from the individual subject data that very high EMG levels were not present in all subjects. This finding implies that two subgroups of patients—headache subjects with and headache subjects without bruxism—may exist in our study sample. Many have speculated that high levels of bruxism during sleep are causally related to headache pain experienced during the following day. This assumption is difficult to accept or reject from the data in this study, since the majority of the headache subjects did not exhibit elevated sleeping EMG, yet clearly suffered from headaches. Moreover, strong bruxism patterns are frequently present in patients without serious signs or symptoms of masticatory muscle pain. This information suggests that the mechanism for the daily tension-type headache is not bruxism alone but some other pathophysiologic process such as neurogenic-induced transient inflammation. However, when the temporalis and, to a lesser extent, other head and neck muscles exhibit these pathophysiologic changes, it is likely that short, strong, repetitive bruxism events during sleep add insult to the muscles and/or temporomandibular joint and thereby result in increased pain and stiffness in the morning. Analysis of individual subjects' sleeping EMG data revealed that a close-to-significant trend towards a difference between the two groups was accounted for by the influence of several subjects in the headache group who had at least one night of very high sleeping-period EMG. The criterion for "very high" sleeping-period EMG was arbitrarily set as a level 10 times greater than the median level seen in the entire subject data set, and this designation was considered evidence of bruxism. Whether more bruxism subjects are to be found in a tension-type headache population than in a nonheadache population is not proven by these data, since a larger true probability sample of headache subjects would be needed to prove this hypothesis.

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References

1. Ad Hoc Committee on the Classification of Headache. Classification of Headache. JAMA 1962;179:717–718.
2. Bakal DA, Kaganov JA. Muscle contraction and migraine headache: Psychophysiologic comparison. Headache 1977;17:208–215.
3. Martin PR, Mathews AM. Tension headaches: Psychophysiological investigation and treatment. J Psychosom Res 1978;22:389–399.
4. Anderson CD, Franks RD. Migraine and tension headache: Is there a physiologic difference? Headache 1981; 21:63–71.
5. Sutton EP, Belar CD. Tension headache patients versus controls: A study of EMG parameter. Headache 1982; 22:133–136.
6. Pikoff H. Is the muscular model of headache still viable? A review of conflicting data. Headache 1984;24:189–198.
7. 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.
8. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. Cephalgia 1988; 8(suppl):1–96.
9. Kapel L, Glaros AG, McGlynn FD. Psychophysiological responses to stress in patients with myofascial pain-dysfunction syndrome. J Behav Med 1989;12:397–406.
10. Pritchard DW. EMG cranial muscle levels in headache sufferers before and during headache. Headache 1989;29: 103–108.
11. Montgomery GT, Rugh JD. Psychophysiological responsivity on a laboratory stress task: Methodological implications for a stress-muscle hyperactivity pain model. Biofeedback Self Regul 1990;15:121–134.

12. Rugh JD. Electromyographic analysis of bruxism in the natural environments. In: Weinstein P (ed). Advances in Behavioral Research in Dentistry. Seattle: University of Washington Press, 1978:67–83.
13. Burgar CB, Rugh JD. An EMG integrator for muscle activity studies in ambulatory subjects. IEEE Trans Biomed Eng 1983;30:66–69.
14. Rugh JD, Hatch JP, Moore PJ, Cyr-Provost M, Boutros NN, Pellegrino CS. The effects of psychological stress on electromyographic activity and negative affect in ambulatory tension-type headache patients. Headache 1990; 30:216–219.
15. Hatch JP, Prihoda TJ, Moore PJ, Cyr-Provost M, Borcherding S, Boutros NN, et al. A naturalistic study of the relationships among electromyographic activity, psychological stress, and pain in ambulatory tension-type headache patients and headache-free controls. Psychosom Med 1991;53:576–584.
16. Clark GT. Muscle hyperactivity pain and dysfunction. In: Klineberg I, Sessle B (eds). Orofacial pain and neuromuscular dysfunction: Mechanisms and clinical correlates. Sydney: Pergamon, 1985:93–111.
17. Steinhardt G. Clinical, radiographic and therapeutic aspects of jaw trismus. Quintessence Int 1978;May:1–10.
18. Tovi F, Zirkin H, Sidi J. Trismus resulting from a parotid hemangioma. J Oral Maxillofac Surg 1983;41:466–469.
19. Stohler C, Yamada Y, Ash MM. Antagonistic muscle stiffness and associated reflex behaviour in the pain-dysfunctional state. Helv Odontol Acta 1985;95:13–20.
20. Yamada Y, Stohler CS, Ash MM. Comparative study of reflexes associated with tooth tapping and painful clicking. J Dent Res 1985;64:217.
21. 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.
22. Phillips C, Hunter M. The treatment of tension headache I. Muscular abnormality and biofeedback. Behav Res Ther 1981a;19:485–498.
23. Phillips C, Hunter M. The treatment of tension headache II. EMG normality and relaxation. Behav Res Ther 1981b; 19:499–507.
24. Glaros AB, Rao SM. Bruxism: A critical review. Psychol Bull 1977;84:767–781.
25. Rugh JD, Ohrbach R. Occlusal parafunction. In: Mohl ND, Zarb GA, Carlsson GE, Rugh JD (eds). A Textbook of Occlusion. Chicago: Quintessence, 1988:249–261.
26. Satoh T, Harada Y. Electrophysiological study on tooth-grinding during sleep. Electroencephalogr Clin Neurophysiol 1973;35:267–275.
27. Clark GT, Beemsterboer PL, Rugh JD. Nocturnal masseter muscle activity and the symptoms of masticatory dysfunction. J Oral Rehabil 1981;8:279–286.
28. Rugh JD, Solberg WK. Electromyographic studies of bruxist behavior before and during treatment. J Calif Dent Assoc 1975;3:56–59.
29. Clark GT, Sakai S. Temporalis EMG level in temporal region headache patients [abstract #P-154]. Canadian and American Pain Societies Joint Meeting, Toronto, Canada, 11 Nov 1988.
30. Sakai S, Clark GT, Flack VF. Stress pain and temporalis muscle EMG in temporal headache subjects [abstract 347]. J Dent Res 1989;68:225.
31. Merrill RL, Sakai S, Clark GT, McCreary CP. Pain, stress and physical activity in chronic daily temporalis region headache patients. Abstracts of the Annual Meeting of the American Association for the Study of Headaches, Los Angeles, 22 June 1990:29.
32. Sakai S, Merrill RL, Clark GT, McCreary CP. Waking and sleeping EMG levels in chronic daily temporalis region headache patients. Abstracts of the Annual Meeting of the American Association for the Study of Headaches, Los Angeles, 22 June 1990:31.
33. Dunn OJ, Clark VA. Applied Statistics: Analysis of Variance and Regression, 2nd ed. New York: Wiley and Sons, 1987.
34. Clark GT, Sakai S, Merrill R, Flack VF, McCreary CP. Cross correlation between stress, pain, physical activity and temporalis muscle EMG in tension type headache. Cephalgia 1995;15:511–518.
35. Arena JG, Sherman RA, Bruno GM, Smith JD, Meador KJ. Effect of movement and position on muscle activity in tension headache sufferers during and between headaches. J Psychosom Res 1991;35:187–195.

Resumen

Niveles Electromiograficos del Musculo Temporal Durante Actividad Diurna y Nocturna en Pacientes con Cefaleas Tensionales Cronicas en la Region Temporal

Este estudio evaluó la actividad del músculo temporal en sujetos con cefalea de tipo tensional (n = 36) y sujetos-control sin cefalea (n = 36). Los pacientes registraron la actividad acumulada del músculo temporal cada 30 minutos por 3 días y 3 noches usando un detector electromiográfico (EMG). El análisis de varianza mostró que estos dos grupos no eran estadísticamente diferentes en los niveles generales de actividad muscular durante la actividad diurna ni nocturna. Cuando los datos del EMG durante actividad diurna fueron separados en actividades funcionales y no-funcionales, se encontró una diferencia significativa entre los grupos durante funcionamiento de la mandíbula (masticar, hablar). Estos resultados sugieren que pacientes con cefalea utilizan el musculo temporal con menos eficiencia durante actividades funcionales que pacientes sin cefalea. Este EMG elevado es probablemente una consequencia al dolor (debido a mecanismos de protección) más que una causa en pacientes que sufren cefaleas de tipo tensional crónicas en la región temporal.

Zusammenfassung

Schläfenmuskel-emg-Aktivität Beim Wachsein und Während des Schlafens von Patienten mit Chronischen Schläfenregion-Spannungstypkopfweh

Diese Studie vegleicht Schläfenmuskelaktivität bei Patienten mit Spannungstypkopfweh (n = 36) mit einer gleichen Kontrollgruppe ohne Kopfweh (n = 36). Die Teilnehmer verzeichneten die kumulative Schläfenmuskelaktivität alle 30 Minuten 3 Tage und Nächt lang mit einem Elektromyographen (EMG). Die Varianzanalyse zeigt, daß weder die wachende noch die schlafende Gesamtaktivität zwischen den zwei Gruppen statistisch zu unterscheiden war. Wenn man die EMG Daten der nicht-schlafenden Gruppe in die folgenden Gebiete, Functionsaktivität und Nichtfunctionsaktivität zerteilt, zeigt sich ein statistischer Unterschied bei der Kieferfunktion (d.h., Kauen und Sprechen) der beiden Gruppen. Diese Daten deuten an, daß Kopfwehpatienten ihre Schläfenmuskeln während der Funktion weniger wirksam benützen, als Teilnehmer ohne Kopfweh. Diese erhöhte EMG ist wahrscheinlicher eine Folge der Schmerzen als eine Ursache bei Leidenden mit chronischem Spannungstypkopfweh in der Schläfenmuskelregion.